



# ALAGAPPA UNIVERSITY

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(A State University Established by the Government of Tamil Nadu)



KARAIKUDI – 630 003

## DIRECTORATE OF DISTANCE EDUCATION

M.Sc. (HOME SCIENCE – NUTRITION AND DIETETICS)

### III SEMESTER

36532

## DIETETICS IN LIFE STYLE DISEASES

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## INTRODUCTION

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The role of food in preventing and treating diseases, such as diabetes, is now well known. More dietitians and nutritionists will be needed to provide care for patients with various medical conditions and to advise people who want to improve their overall health. Dietetics is defined as ‘the application of the science of nutrition to the human being in health and disease.’ However, the term ‘dietitian,’ used to describe a practitioner of dietetics, was in use long before the science of nutrition had become an accepted discipline. Many of the ongoing diet and lifestyle interventions in low-and middle-income countries are relatively recent, and few have documented reductions in the rates of major chronic diseases. However, the successes of Finland, Singapore, and many other high-income countries in reducing rates of CAD, stroke, and smoking-related cancers strongly suggest that similar benefits will emerge in the developing countries.

This book on dietetics in life style diseases is divided into four blocks with fourteen units, and includes all these principles, their impacts diet therapy and different therapeutic diets for various diseases. The objectives of providing an insight into the nutritional problems and their implications, to understand the international contribution towards nutritional improvements in India, and developing the skills in organizing and evaluating nutrition projects in the community has been obliged. To serve the purpose of educating distance learning students, this book is presented in an understandable manner with simple language. Every unit is detailed with the structure to prepare the student for what to expect in the text. Check Your Progress Questions in between the units helps the students to recollect what had been discussed and Self-Assessment Questions at the end of each unit aids the student to estimate the level of understanding on their own.

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# **BLOCK-I: DIETETICS IN STRESS AND WEIGHT MANAGEMENT**

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*Dietetics in Stress and Weight  
Management*

NOTES

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## **UNIT-I**

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- 1.1 Objectives
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  - 1.2.2. Types
  - 1.2.3. Psychosomatic disorders due to stress and functional adjustment
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### **1.0 INTRODUCTION**

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Stress - causes of stress and how it can impact on you. Stress is a feeling that's created when you react to certain events. It's the body's way of preparing to meet a situation with focus and heightened alertness. Stress can become a problem when it starts to affect how you cope with day-to-day stuff. Stress is a common problem that affects almost all of us at some point in our lives. Learning to identify when we are under stress, what is stressing us, and different ways of coping with stress can greatly improve both our mental and physical well being.

This course provides us with some basic information on stress and some simple recommendations for dealing with stress. It is not intended to take the place of advice from a physician or counselor, but it can be the first step in deciding how to manage to stress and increase your well being.

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### **1.1 OBJECTIVES**

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After, learning this unit, you will be able to:

- Understand the nature, types and sources of stress as life challenges, examine the effects of stress on psychological functioning
- Learn ways to cope with stress
- Know about the life skills that help people to stay healthy, and
- Understand the factors that promote positive health and well-beingdefine stress.
- Describe its type of stress and discuss how psychosomatic disorders due to stress and functional adjustment.

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## **STRESS**

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To define the stress and its types with its psychosomatic disorder to go further with the chapter.

### **1.2.1 Definition**

Stress has a different meaning for different people under different conditions. The first and most generic definition of stress was that proposed by Hans Selye: "Stress is the nonspecific response of the body to any demand." Other definitions have evolved to cater for different situations—for example, cognitive.

Stress, Selye underscored, "is not something to be avoided. Indeed, it cannot be avoided, since just staying alive creates some demand for life-maintaining energy. Even when man is asleep, his heart, respiratory apparatus, digestive tract, nervous system and other organs must continue to function. Complete freedom from stress can be expected only after death." There has been much controversy and debate about Selye's concepts and particularly Selye's view that stress is best regarded as a non-specific response. Because of their heuristic value, these points will be further considered below.

Other definitions, reviewed in detail by Selye in his *Stress in Health and Disease* (1976), include the following:

1. In behavioral sciences, stress is regarded as the "perception of threat, with resulting anxiety discomfort, emotional tension, and difficulty in adjustment."
2. In the group situation, lack of structure or loss of anchor "makes it difficult or impossible for the group to cope with the requirements of the situation, and the problem of leadership and interpersonal behavior becomes one of evolving or supplying a structure or anchor and of supplying the expertness for coping with the demands of the situation."
3. Stress can also be defined in terms of pure neuroendocrinology. Eugene Yates, for example, defined stress as any stimulus that will provoke the release of ACTH and adrenal glucocorticoids. Presumably, the same might apply to the equally powerful sympathetic markers of stress, underscored earlier by Walter Cannon.
4. Selye also mentions Richard Lazarus, famous for his work in cognitive psychology and focus on the emotions. Lazarus underscores the difficulties of reaching a precise overarching definition of stress by setting out the following different meanings of the term: "In spite of consistent confusion about the precise meaning of the term, stress is widely recognized as a central problem in human life. Scientists of many disciplines have conceptualized stress but each field appears to have something different in mind concerning its meaning."

The orientation toward stress research is changing as awareness of the social and cultural contexts involved in stress and coping are examined. The bio-psychosocial model of stress incorporates a variety of social

factors into its model that influence stress reaction and perception. However, research into the cultural differences that may exist in stress reactions are also needed to examine how various social and cultural structures influence the individual's experience of stress. Culture and society may shape events that are perceived as stressful, what coping strategies are used in a particular society, and what institutional mechanisms we may turn to for assistance (FumikoNaughton, personal communication).

For the sociologist, it is social disequilibrium, that is, disturbances in the social structure within which people live. Engineers conceive of stress as some external force which produces strain in the materials exposed to it. Physiologists deal with the physical stressors that include a wide range of stimulus conditions that are noxious to the body. In the history of psychological stress research, there has been no clear separation between physical stressors which attack biological tissue systems and psychological stressors which produce their effects purely because of their psychological significance.”

According to experts, the definition of stress is the body's innate response to a physical, mental or emotional stressors that can either be real or imagined. The stressor can be a stressful event that can either be depressing or wonderful. Stress is powerful because it can affect one's overall health. Its effects can either be negative or positive depending on the types of stress one experience. In simple terms, the definition of stress is the body's natural defense or survival mechanism to protect itself. It is also called the “flight-or-fight response”. Its medical term is neuroendocrine response. When one is stressed most of the time, inflammation can set in inside the body. Chronic inflammation can feed immature or damaged cells in the body. Instead of dying, these cells can turn malignant.

The biological effects of stress on various systems such as brain, cardiovascular system, respiratory system, vital organ and immune systems are explained in details with all its relevant literature in UNIT-II

Now, we shall study the types and psychosomatic disorders due to stress and its functional adjustment.

### **1.2.2 TYPES OF STRESS**

The Neurobiology of stress is a complex operating mechanism. According to American Psychological Association (APA), there are 3 different types stress — acute stress, episodic acute stress, and chronic stress. The 3 types of stress each have their own characteristics, symptoms, duration, and treatment approaches. Stress management can be complicated and confusing because there are different types of stress — acute stress, episodic acute stress, and chronic stress — each with its own characteristics, symptoms, duration and treatment approaches. Let's look at each one.

#### **ACUTE STRESS**

Acute stress is the most common form of stress. It comes from demands and pressures of the recent past and anticipated demands and pressures of the near future. Acute stress is thrilling and exciting in small

doses, but too much is exhausting. A fast run down a challenging ski slope, for example, is exhilarating early in the day. That same ski run late in the day is taxing and wearing. Skiing beyond your limits can lead to falls and broken bones. By the same token, overdoing on short-term stress can lead to psychological distress, tension headaches, upset stomach and other symptoms.

Fortunately, acute stress symptoms are recognized by most people. It's a laundry list of what has gone awry in their lives: the auto accident that crumpled the car fender, the loss of an important contract, a deadline they're rushing to meet, and their child's occasional problems at school and so on.

Because it is short term, acute stress doesn't have enough time to do the extensive damage associated with long-term stress. The most common symptoms are:

- Emotional distress — some combination of anger or irritability, anxiety and depression, the three stress emotions.
- Muscular problems including tension headache, back pain, jaw pain, and the muscular tensions that lead to pulled muscles and tendon and ligament problems.
- Stomach, gut, and bowel problems such as heartburn, acid stomach, flatulence, diarrhea, constipation and irritable bowel syndrome.
- Transient overarousal leads to elevation in blood pressure, rapid heartbeat, sweaty palms, heart palpitations, dizziness, migraine headaches, cold hands or feet, shortness of breath and chest pain.

Acute stress can crop up in anyone's life, and it is highly treatable and manageable.

### **EPISODIC ACUTE STRESS**

There are those, however, who suffer acute stress frequently, whose lives are so disordered that they are studies in chaos and crisis. They're always in a rush, but always late. If something can go wrong, it does. They take on too much, have too many irons in the fire, and can't organize the slew of self-inflicted demands and pressures clamoring for their attention. They seem perpetually in the clutches of acute stress.

It is common for people with acute stress reactions to be over aroused, short-tempered, irritable, anxious, and tense. Often, they describe themselves as having "a lot of nervous energy." Always in a hurry, they tend to be abrupt, and sometimes their irritability comes across as hostility. Interpersonal relationships deteriorate rapidly when others respond with real hostility. The workplace becomes a very stressful place for them.

The cardiac prone, "Type A" personality described by cardiologists, Meter Friedman and Ray Rosenman, is similar to an extreme case of episodic acute stress. Type A's have an "excessive competitive drive, aggressiveness, impatience, and a harrying sense of time urgency." In addition there is a "free-floating, but well-rationalized form of hostility, and almost always a deep-seated insecurity." Such personality characteristics would seem to create frequent episodes of acute stress for the Type A individual. Friedman and Rosenman found Type A's to be

much more likely to develop coronary heart disease than Type B's, who show an opposite pattern of behavior.

Another form of episodic acute stress comes from ceaseless worry. "Worry warts" see disaster around every corner and pessimistically forecast catastrophe in every situation. The world is a dangerous, unrewarding, punitive place where something awful is always about to happen. These "awfulizers" also tend to be over aroused and tense, but are more anxious and depressed than angry and hostile.

The symptoms of episodic acute stress are the symptoms of extended over arousal: persistent tension headaches, migraines, hypertension, chest pain and heart disease. Treating episodic acute stress requires intervention on a number of levels, generally requiring professional help, which may take many months.

Often, lifestyle and personality issues are so ingrained and habitual with these individuals that they see nothing wrong with the way they conduct their lives. They blame their woes on other people and external events. Frequently, they see their lifestyle, their patterns of interacting with others, and their ways of perceiving the world as part and parcel of who and what they are.

Sufferers can be fiercely resistant to change. Only the promise of relief from pain and discomfort of their symptoms can keep them in treatment and on track in their recovery program.

### **CHRONIC STRESS**

While acute stress can be thrilling and exciting, chronic stress is not. This is the grinding stress that wears people away day after day, year after year. Chronic stress destroys bodies, minds and lives. It wreaks havoc through long-term attrition. It's the stress of poverty, of dysfunctional families, of being trapped in an unhappy marriage or in a despised job or career. It's the stress that the never-ending "troubles" have brought to the people of Northern Ireland, the tensions of the Middle East have brought to the Arab and Jew, and the endless ethnic rivalries that have been brought to the people of Eastern Europe and the former Soviet Union.

Chronic stress comes when a person never sees a way out of a miserable situation. It's the stress of unrelenting demands and pressures for seemingly interminable periods of time. With no hope, the individual gives up searching for solutions.

Some chronic stresses stem from traumatic, early childhood experiences that become internalized and remain forever painful and present. Some experiences profoundly affect personality. A view of the world, or a belief system, is created that causes unending stress for the individual (e.g., the world is a threatening place, people will find out you are a pretender, you must be perfect at all times). When personality or deep-seated convictions and beliefs must be reformulated, recovery requires active self-examination, often with professional help.

The worst aspect of chronic stress is that people get used to it. They forget it's there. People are immediately aware of acute stress because it is new; they ignore chronic stress because it is old, familiar, and sometimes, almost comfortable.



Chronic stress kills through suicide, violence, heart attack, stroke and, perhaps, even cancer. People wear down to a final, fatal breakdown. Because physical and mental resources are depleted through long-term attrition, the symptoms of chronic stress are difficult to treat and may require extended medical as well as behavioral treatment and stress management.

### **1.2.3. PSYCHOSOMATIC DISORDER DUE TO STRESS AND FUNCTIONAL ADJUSTMENT**

A psychosomatic disorder involves both the body and mind. These diseases have physical symptoms originating from mental or emotional causes. Most common causes are stress, anxiety, and depression. When these psychological entities are not perceived properly, it may result in somatic disease due to conversion hysteria.

In psychology, stress is a feeling of strain and pressure. Stress is a type of psychological pain. Small amounts of stress may be desired, beneficial, and even healthy. Positive stress helps improve athletic performance. It also plays a factor in motivation, adaptation, and reaction to the environment. Excessive amounts of stress, however, may lead to bodily harm. Stress can increase the risk of strokes, heart attacks, ulcers, and mental illnesses such as depression.

Stress can be external and related to the environment, but may also be caused by internal perceptions that cause an individual to experience anxiety or other negative emotions surrounding a situation, such as pressure, discomfort, etc., which they then deem stressful.

#### **TYPES**

A very much overlooked side of stress is its positive adaptations. Positive psychological stress can lead to motivation and challenge instead of anxiety. The effects of experiencing eustress, which is positive stress, versus distress, defined as negative stress, are significant. While colloquially lumped together, the various types of stress should be treated as separate concepts.

Selye (1974) proposed four variations of stress. On one axis he locates good stress (eustress) and bad stress (distress). On the other is over-stress (hyperstress) and understress (hypostress). Selye advocates balancing these: the ultimate goal would be to balance hyperstress and hypostress perfectly and have as much eustress as possible. Stress is extremely useful for a productive lifestyle because it makes working enjoyable instead of a chore, as seen with distress.

The term "eustress" comes from the Greek root *eu-* which means "good" (as in "euphoria"). Eustress results when a person perceives a stressor as positive. "Distress" stems from the Latin root *dis-* (as in "dissonance" or "disagreement"). Medically defined distress is a threat to the quality of life. It occurs when a demand vastly exceeds a person's capabilities.

#### **CAUSES NEUTRALITY OF STRESSORS**

Stress is a non-specific response. It is neutral, and what varies is the degree of response. It is all about the context of the individual and how they perceive the situation. Selye defined stress as "the nonspecific (that is, common) result of any demand upon the body, be the effect mental or

somatic.” This includes the medical definition of stress as a physical demand and the colloquial definition of stress as a psychological demand. A stressor is inherently neutral meaning that the same stressor can cause either distress or eustress. It is individual differences and responses that induce either distress or eustress.

### **Types of stressors**

A stressor is any event, experience, or environmental stimulus that causes stress in an individual. These events or experiences are perceived as threats or challenges to the individual and can be either physical or psychological. Researchers have found that stressors can make individuals more prone to both physical and psychological problems, including heart disease and anxiety.

Stressors are more likely to affect an individual's health when they are "chronic, highly disruptive, or perceived as uncontrollable". In psychology, researchers generally classify the different types of stressors into four categories:

1. Crises/catastrophes,
2. Major life events,
3. Daily hassles/microstressors, and
4. Ambient stressors.

#### ***Crises/catastrophes***

This type of stressor is unforeseen and unpredictable and, as such, is completely out of the control of the individual.<sup>[12]</sup> Examples of crises and catastrophes include: devastating natural disasters, such as major floods or earthquakes, wars, etc. Though rare in occurrence, this type of stressor typically causes a great deal of stress in a person's life. A study conducted by Stanford University found that after natural disasters, those affected experienced a significant increase in stress level. Combat stress is a widespread acute and chronic problem. With the rapid pace and the urgency of firing first, tragic episodes of accidentally killing friendly forces (“brother” killing “brother” or fratricide) may happen. Prevention requires stress reduction, emphasis on vehicle and other identification training, awareness of the tactical situation, and continual risk analysis by leaders at all echelons.

#### ***Major life events***

Common examples of major life events include: marriage, going to college, death of a loved one, birth of a child, moving houses, etc. These events, either positive or negative, can create a sense of uncertainty and fear, which will ultimately lead to stress. For instance, research has found the elevation of stress during the transition from high school to university, with college freshmen being about two times more likely to be stressed than final year students. Research has found major life events are somewhat rare to be major causes of stress, due to its rare occurrences.

The length of time since occurrence and whether or not it is a positive or negative event are factors in whether or not it causes stress and how much stress it causes. Researchers have found that events that have occurred within the past month generally are not linked to stress or illness, while chronic events that occurred more than several months ago are linked

to stress and illness and personality change. Additionally, positive life events are typically not linked to stress – and if so, generally only trivial stress – while negative life events can be linked to stress and the health problems that accompany it. However, positive experiences and positive life changes can predict decreases in neuroticism.

### ***Daily hassles/micro stressors***

This category includes daily annoyances and minor hassles.<sup>[12]</sup> Examples include: making decisions, meeting deadlines at work or school, traffic jams, encounters with irritating personalities, etc. Often, this type of stressor includes conflicts with other people. Daily stressors, however, are different for each individual, as not everyone perceives a certain event as stressful. For example, most people find public speaking to be stressful, nevertheless, a seasoned politician most likely will not.

Daily hassles are the most frequently occurring type of stressor in most adults. The high frequency of hassles causes this stressor to have the most physiological effect on an individual. Carolyn Aldwin, Ph.D., conducted a study at the Oregon State University that examined the perceived intensity of daily hassles on an individual's mortality. Aldwin's study concluded that there is a strong correlation between individuals who rate their hassles as very intense and a high level of mortality. One's perception of his/her daily stressors can have a modulating effect on the physiological impact of daily stressors.

There are three major psychological types of conflicts that can cause stress.

- The approach-approach conflict, occurs when a person is choosing between two equally attractive options, i.e. whether to go see a movie or to go see a concert.
- The avoidance-avoidance conflict, occurs where a person has to choose between two equally unattractive options, for example, to take out a second loan with unappealing terms to pay off the mortgage or to face foreclosure on one's house.
- The approach-avoidance conflict, occurs when a person is forced to choose whether or not to partake in something that has both attractive and unattractive traits – such as whether or not to attend an expensive college (meaning taking out loans now, but also meaning a quality education and employment after graduation).

Travel-related stress results from three main categories: lost time, surprises (an unforeseen event such as lost or delayed baggage) and routine breakers (inability to maintain daily habits).

### ***Ambient stressors***

As their name implies, these are global (as opposed to individual) low-grade stressors that are a part of the background environment. They are defined as stressors that are "chronic, negatively valued, non-urgent, physically perceptible, and intractable to the efforts of individuals to change them". Typical examples of ambient stressors are pollution, noise, crowding, and traffic. Unlike the other three types of stressor, ambient stressors can (but do not necessarily have to) negatively impact stress without conscious awareness. They are thus low on what Stokols called "perceptual salience".

### **Organizational stressors**

Studies conducted in military and combat fields show that some of the most potent stressors can be due to personal organizational problems in the unit or on the home front. Stress due to bad organizational practices is often connected to "Toxic Leadership", both in companies and in governmental organizations.

#### **Stressor impact**

Life events scales can be used to assess stressful things that people experience in their lives. One such scale is the Holmes and Rahe Stress Scale, also known as the Social Readjustment Rating Scale, or SRRS.<sup>[23]</sup> Developed by psychiatrists Thomas Holmes and Richard Rahe in 1967, the scale lists 43 stressful events.

To calculate one's score, add up the number of "life change units" if an event occurred in the past year. A score of more than 300 means that individual is at risk for illness, a score between 150 and 299 means risk of illness is moderate, and a score under 150 means that individual only has a slight risk of illness.

#### **Physical effects**

The body responds to stress in many ways. Readjusting chemical levels is just one of them. Here are some examples of adjustments and changes.

In terms of measuring the body's response to stress, psychologists tend to use Hans Selye's general adaptation syndrome. This model is also often referred to as the classic stress response, and it revolves around the concept of homeostasis. General adaptive syndrome occurs in three stages:

1. **The alarm reaction.** This stage occurs when the stressor is first presented. The body begins to gather resources to deal with the stressor. The hypothalamic-pituitary-adrenal axis and sympathetic nervous system are activated, resulting in the release of hormones from the adrenal gland such as cortisol, adrenaline (epinephrine), and norepinephrine into the bloodstream to adjust bodily processes. These hormonal adjustments increase energy levels, increase muscle tension, reduce sensitivity to pain, slow down the digestive system, and cause a rise in blood pressure. In addition, the Locus coeruleus, a collection of Norepinephrine-containing neurons in the pons of the brainstem whose axons project to various regions of the brain, is involved in releasing Norepinephrine directly onto neurons. High levels of Norepinephrine acting as a neurotransmitter on its receptors expressed on neurons in brain regions, such as the prefrontal cortex is thought to be involved in the effects of stress on executive functions, such as impaired working memory.
2. **The stage of resistance.** The body continues building up resistance throughout the stage of resistance, until either the body's resources are depleted, leading to the exhaustion phase, or the stressful stimulus is removed. As the body uses up more and more of its resources people become increasingly tired and susceptible to illness. This stage is where psychosomatic disorders first begin to appear.

3. **The stage of exhaustion.** The body is completely drained of the hormones and resources it was depending on to manage the stressor. The person now begins to exhibit behaviors such as anxiety, irritability, avoidance of responsibilities and relationships, self-destructive behavior, and poor judgment.

This physiological stress response involves high levels of sympathetic nervous system activation, often referred to as the "fight or flight" response. The response involves pupil dilation, release of endorphins, increased heart and respiration rates, cessation of digestive processes, secretion of adrenaline, arteriole dilation, and constriction of veins. This high level of arousal is often unnecessary to adequately cope with micro-stressors and daily hassles; yet, this is the response pattern seen in humans, which often leads to health issues commonly associated with high levels of stress.

### **Cancer**

Evidence for a link between stress and cancer is unclear as of 2019. This can be due to practical difficulties in designing and implementing adequate studies. Personal belief in stress as a risk factor for cancer was common in the UK, though awareness of risk factors overall was found to be low.

### **Sleep**

Sleep allows people to rest and re-energize for another day filled with interactions and tasks. If someone is stressed it is extremely important for them to get enough sleep so that they can think clearly. Unfortunately, chemical changes in the body caused by stress can make sleep a difficult thing. Glucocorticoids are released by the body in response to stress which can disrupt sleep.

### **Other effects**

There is likely a connection between stress and illness. Theories of the stress–illness link suggest that both acute and chronic stress can cause illness, and several studies found such a link. According to these theories, both kinds of stress can lead to changes in behavior and in physiology. Behavioral changes can be smoking and eating habits and physical activity. Physiological changes can be changes in sympathetic activation or hypothalamic pituitaryadrenocorticoidactivation, and immunological function. However, there is much variability in the link between stress and illness.

Stress can make the individual more susceptible to physical illnesses like the common cold. Stressful events, such as job changes, may result in insomnia, impaired sleeping, and health complaints. Research indicates the type of stressor (whether it is acute or chronic) and individual characteristics such as age and physical well-being before the onset of the stressor can combine to determine the effect of stress on an individual. An individual's personality characteristics (such as level of neuroticism), genetics, and childhood experiences with major stressors and traumas may also dictate their response to stressors.

Chronic stress and a lack of coping resources available or used by an individual can often lead to the development of psychological issues such as depression and anxiety. This is particularly true regarding chronic stressors. These are stressors that may not be as intense as an acute stressor

like a natural disaster or a major accident, but they persist over longer periods of time. These types of stressors tend to have a more negative impact on health because they are sustained and thus require the body's physiological response to occur daily. This depletes the body's energy more quickly and usually occurs over long periods of time, especially when these microstressors cannot be avoided (i.e. stress of living in a dangerous neighborhood).

Studies have also shown that perceived chronic stress and the hostility associated with Type A personalities are often associated with much higher risks of cardiovascular disease. This occurs because of the compromised immune system as well as the high levels of arousal in the sympathetic nervous system that occur as part of the body's physiological response to stressful events. However, it is possible for individuals to exhibit hardiness – a term referring to the ability to be both chronically stressed and healthy. Chronic stress can be associated with psychological disorders such as delusions. Pathological anxiety and chronic stress lead to structural degeneration and impaired functioning of the hippocampus.

It has long been believed that negative affective states, such as feelings of anxiety and depression, could influence the pathogenesis of physical disease, which in turn, have direct effects on biological process that could result in increased risk of disease in the end. However, studies done by the University of Wisconsin-Madison and other places have shown this to be partly untrue; although stress seems to increase the risk of reported poor health, the *perception* that stress is harmful increases the risk even further. For example, when humans are under chronic stress, permanent changes in their physiological, emotional, and behavioral responses are most likely to occur. Such changes could lead to disease. Chronic stress results from stressful events that persist over a relatively long period of time, such as caring for a spouse with dementia, or results from brief focal events that continue to be experienced as overwhelming even long after they are over, such as experiencing a sexual assault.

Experiments show that when healthy human individuals are exposed to acute laboratory stressors, they show an adaptive enhancement of some markers of natural immunity but a general suppression of functions of specific immunity. By comparison, when healthy human individuals are exposed to real-life chronic stress, this stress is associated with a biphasic immune response where partial suppression of cellular and humoral function coincides with low-grade, nonspecific inflammation.

Even though psychological stress is often connected with illness or disease, most healthy individuals can still remain disease-free after confronting chronic stressful events. Also, people who do not believe that stress will affect their health do not have an increased risk of illness, disease, or death. This suggests that there are individual differences in vulnerability to the potential pathogenic effects of stress; individual differences in vulnerability arise due to both genetic and psychological factors. In addition, the age at which the stress is experienced can dictate its effect on health. Research suggests chronic stress at a young age can have lifelong impacts on the biological, psychological, and behavioral responses to stress later in life.

## **SOCIAL IMPACT COMMUNICATION**

When someone is stressed, many challenges can arise; a recognized challenge being communication difficulties. Here are some examples of how stress can hinder communication.

The cultures of the world generally fall into two categories; individualistic and collectivistic.

- An individualistic culture, like that of the United States, where everyone is an independent entity defined by their accomplishments and goals.
- A collectivistic culture, like that of many Asian countries, prefers to see individuals as interdependent on each other. They value modesty and family.

These cultural differences can affect how people communicate when they are stressed. For example, a member of an individualistic culture would be hesitant to ask for pain medication for fear of being perceived as weak. A member of a collectivistic culture would not hesitate. They have been brought up in a culture where everyone helps each other and is one functional unit whereas the member of the individualistic culture is not as comfortable asking others for aid.<sup>[46]</sup>

### ***Language barriers***

Language barriers can cause stress by making people feel uncomfortable because differences in syntax, vocabulary, different ways of showing respect, and different use of body language can make things difficult, and along with a desire for successful social interactions, being uncomfortable with the communication around a person can discourage them from communicating at all.

### ***Changes in the home***

Divorce, death, and remarriage are all disruptive events in a household. Although everyone involved is affected by events such as these, it can be most drastically seen in children. Due to their age, children have relatively undeveloped coping skills. For this reason a stressful event may cause some changes in their behavior. Falling in with a new crowd, developing some new and sometimes undesirable habits are just some of the changes stress may trigger in their lives.

A particularly interesting response to stress is talking to an imaginary friend. A child may feel angry with a parent or their peers who they feel brought this change on them. They need someone to talk to but it definitely would not be the person with whom they are angry. That is when the imaginary friend comes in. They "talk" to this imaginary friend but in doing so they cut off communication with the real people around them.

### **Social support and health**

Researchers have long been interested in how an individual's level and types of social support impact the effect of stress on their health. Studies consistently show that social support can protect against physical and mental consequences of stress. This can occur through a variety of mechanisms. One model, known as the "direct effects" model, holds that social support has a direct, positive impact on health by increasing positive affect, promoting adaptive health behaviours, predictability and stability in life, and safeguarding against social, legal, and economic concerns that

could negatively impact health. Another model, the "buffering effect", says that social support exerts greatest influence on health in times of stress, either by helping individuals appraise situations in less threatening manners or coping with the actual stress. Researchers have found evidence to support both these pathways.

Social support is defined more specifically as psychological and material resources provided by a social network that are aimed at helping an individual cope with stress.<sup>[52]</sup> Researchers generally distinguish among several types of social support: instrumental support – which refers to material aid (e.g., financial support or assistance in transportation to a physician's appointment), informational support (e.g., knowledge, education or advice in problem-solving), and emotional support (e.g., empathy, reassurance, etc.). Social support can reduce the rate of stress during pregnancy.

### **Management**

Stress management refers to a wide spectrum of techniques and psychotherapies aimed at controlling a person's levels of stress, especially chronic stress, usually for the purpose of improving everyday functioning. It involves controlling and reducing the tension that occurs in stressful situations by making emotional and physical changes.

### **Prevention and resilience building**

Decreasing stressful behaviours is a part of prevention, some of the common strategies and techniques are: Self-monitoring, tailoring, material reinforcement, social reinforcement, social support, self-contracting, contracting with significant other, shaping, reminders, self-help groups, and professional help.

Although many techniques have traditionally been developed to deal with the consequences of stress considerable research has also been conducted on the prevention of stress, a subject closely related to psychological resilience-building. A number of self-help approaches to stress-prevention and resilience-building have been developed, drawing mainly on the theory and practice of cognitive-behavioral therapy.

### **Exercising to reduce stress**

Studies have shown that exercise reduces stress. Exercise effectively reduces fatigue, improves sleep, enhances overall cognitive function such as alertness and concentration, decreases overall levels of tension, and improves self-esteem. Because many of these are depleted when an individual experiences chronic stress, exercise provides an ideal coping mechanism. Despite popular belief, it is not necessary for exercise to be routine or intense in order to reduce stress. As little as five minutes of aerobic exercise can begin to stimulate anti-anxiety effects. Further, a 10-minute walk may have the same psychological benefits as a 45-minute workout, reinforcing the assertion that exercise in any amount or intensity will reduce stress.

### **Theoretical explanations**

A multitude of theories have been presented in attempts to explain why exercise effectively reduces stress. One theory, known as the time-out hypothesis, claims that exercise provides distraction from the stressor. The time out hypothesis claims that exercise effectively reduces stress because



it gives individuals a break from their stressors. This was tested in a recent study of college women who had identified studying as their primary stressor.<sup>[58]</sup> The women were then placed under four conditions at varying times: "rest," "studying," "exercising," and "studying while exercising." The stress levels of the participants were measured through self-assessments of stress and anxiety symptoms after each condition. The results demonstrated that the "exercise" condition had the most significant reduction in stress and anxiety symptoms. These results demonstrate the validity of the time-out hypothesis. It is also important to note that exercise provided greater stress reduction than rest.

### **Coping mechanisms**

#### ***Main article: Coping (psychology)***

The Lazarus and Folkman model suggests that external events create a form of pressure to achieve, engage in, or experience a stressful situation. Stress is not the external event itself, but rather an interpretation and response to the potential threat; this is when the coping process begins.

There are various ways individuals deal with perceived threats that may be stressful. However, people have a tendency to respond to threats with a predominant coping style, in which they dismiss feelings, or manipulate the stressful situation.

There are different classifications for coping, or defense mechanisms, however they all are variations on the same general idea: There are good/productive and negative/counterproductive ways to handle stress. Because stress is perceived, the following mechanisms do not necessarily deal with the actual situation that is causing an individual stress. However, they may be considered coping mechanisms if they allow the individual to cope better with the negative feelings/anxiety that they are experiencing due to the perceived stressful situation, as opposed to actually fixing the concrete obstacle causing the stress. The following mechanisms are adapted from the DSM-IV Adaptive Functioning Scale, APA, 1994.

#### ***Highly adaptive/active/problem-focused mechanisms***

These skills are what one could call as "facing the problem head on", or at least dealing with the negative emotions experienced by stress in a constructive manner. (Generally adaptive)

- **Affiliation** ("tend and befriend") – involves dealing with stress by turning to a social network for support, but an individual does not share with others in order to diffuse or avoid the responsibility.
- **Humor** – the individual steps outside of a situation in order to gain greater perspective, and also to highlight any comic aspect to be found in their stressful circumstances.

#### **Coping through laughter**

"The Association for Applied and Therapeutic Humor defines therapeutic humor as 'any intervention that promotes health and wellness by stimulating a playful discovery, expression or appreciation of the absurdity of or incongruity of life's situations. This intervention may enhance health or be used as a complementary treatment of illness to facilitate healing or coping whether physical, emotional, cognitive, or spiritual'".

Sigmund Freud, a well known neurologist, suggests the humor was an excellent defensive strategy in emotional situations. When one laughs

during a tough situation they feel absent from their worries, and this allows them to think differently. When one experiences a different mind set, they feel more in control of their response, and how they will go about dealing with the event that caused stress.

Lefcourt (2001) suggests that this perspective-taking humor is the most effective due to its ability to distance oneself from the situation of great stress. Studies show that the use of laughter and humor creates a sense of relief of stress that can last up to 45 minutes post-laughter.

Also, most hospitalized children have been seen to use laughter and play to relieve their fear, pain and stress. It has been discovered that there is a great importance in the use of laughter and humor in stress coping. Humans should use humor as a means to transcend their original understanding of an external event, take a different perspective, in which their anxiety may be minimized by.

- **Sublimation** – allows an "indirect resolution of conflict with neither adverse consequences nor consequences marked by loss of pleasure."<sup>[64]</sup> Essentially, this mechanism allows channeling of troubling emotions or impulses into an outlet that is socially acceptable.
- **Positive reappraisal** – redirects thoughts (cognitive energy) to good things that are either occurring or have not occurred. This can lead to personal growth, self-reflection, and awareness of the power/benefits of one's efforts. For example, studies on veterans of war or peacekeeping operations indicate that persons who construe a positive meaning from their combat or threat experiences tend to adjust better than those who do not.

The final path model fitted well (CF1 = 1, RMSEA = 0.00) and showed that direct quality of life paths with  $\beta = -0.2$ , and indirect social support with  $\beta = -0.088$  had the most effects on reduction of stress during pregnancy. Other adaptive coping mechanisms include anticipation, altruism, and self-observation.

#### ***Mental inhibition/disavowal mechanisms***

These mechanisms cause the individual to have a diminished (or in some cases non-existent) awareness about their anxiety, threatening ideas, fears, etc., that come from being conscious of the perceived threat.

- **Displacement** – This is when an individual redirects their emotional feelings about one situation to another, less threatening one.<sup>[67]</sup>
- **Repression** – Repression occurs when an individual attempts to remove all their thoughts, feelings, and anything related to the upsetting/stressful (perceived) threat out of their awareness in order to be disconnected from the entire situation. When done long enough in a successful way, this is more than just denial.
- **Reaction formation** – An attempt to remove any "unacceptable thoughts" from one's consciousness by replacing them with the exact opposite.

Other inhibition coping mechanisms include undoing, dissociation, denial, projection, and rationalization. Although

some people claim that inhibition coping mechanisms may eventually increase the stress level because the problem is not solved, detaching from the stressor can sometimes help people to temporarily release the stress and become more prepared to deal with problems later on.

### **Active mechanisms**

These methods deal with stress by an individual literally taking action, or withdrawing.

- **Acting out** – Often viewed as counter-normative, or problematic behavior. Instead of reflecting or problem-solving, an individual takes maladaptive action.<sup>[61]</sup>
- **Passive aggression** – When an individual indirectly deals with his or her anxiety and negative thoughts/feelings stemming from their stress by acting in a hostile or resentful manner towards others. Help-Rejecting Complaining can also be included in this category.

### **Health promotion**

There is an alternative method to coping with stress, in which one works to minimize their anxiety and stress in a preventative manner. If one works towards coping with stress daily, the feeling of stress and the ways in which one deals with it as the external event arises becomes less of a burden.

Suggested strategies to improve stress management include:

1. Regular exercise – set up a fitness program, 3–4 times a week
2. Support systems – to listen, offer advice, and support each other
3. Time management – develop an organizational system
4. Guided imagery and visualization – create a relaxing state of mind
5. Progressive muscle relaxation – loosen tense muscle groups
6. Assertiveness training – work on effective communication
7. Journal writing – express true emotion, self-reflection
8. Stress management in the workplace – organize a new system, switch tasks to reduce own stress.
9. HeartSpeak - a novel method for reducing stress and other stress-related conditions such as anxiousness, depression, and low self-esteem.

Depending on the situation, all of these coping mechanisms may be adaptive, or maladaptive.

#### **Check Your Progress**

1. Definition of stress
2. Types of stress
3. Psychosomatic disorder and its functional adjustments

## **1.6 SUMMARY**

This unit provides us an understanding about the Stress which is a part of life. Stress is neither a stimulus nor a response but an ongoing transactional process between the individual and the environment. There are three major types of stresses, physical and environmental, psychological and social. Sources of stress are life events, everyday hassles, and traumatic events. The response to stress is emotional, physiological, cognitive, and behavioural. This unit has clearly portraits

the stress and its relational behaviour in human subjects and its managerial solutions.

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## 1.7 KEY WORDS

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- **Stress:** Stress is a feeling of strain and pressure. Stress is a type of psychological pain. Small amounts of stress may be desired, beneficial, and even healthy. Positive stress helps improve athletic performance. It also plays a factor in motivation, adaptation, and reaction to the environment.
- **Psychosomatic disorder:** A psychosomatic disorder is a disease which involves both mind and body. Some physical diseases are thought to be particularly prone to be made worse by mental factors such as stress and anxiety. Your current mental state can affect how bad a physical disease is at any given time.

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## 1.8 SELF ASSESSMENT QUESTIONS AND ANSWERS

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### Short Answer Questions

1. Write a short note on stress.
2. Define stress. Explain its types of stress.

### Long Answer Questions

1. Explain in details about psychosomatic disorder due to stress and functional adjustment.
2. Write about the types stress and its management.

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## 1.9 FURTHER READINGS

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- Benjamin B. Wolman, 2018, Psychosomatic disorders, Springer, Boston, MA
- Srilakshmi.B, 2016, Nutrition Science, New Age International Publishers, New Delhi.

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## UNIT-II

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### Structure

- 2.0 Introduction
- 2.1 Objectives
- 2.2 Biological effects of stress on various system
  - 2.2.1. Brain
  - 2.2.2. Cardiovascular system
  - 2.2.3. Respiratory system
  - 2.2.4. Non vital organs
    - 2.2.4.1. Digestive system
    - 2.2.4.2. Esophagus
    - 2.2.4.3. Stomach
    - 2.2.4.4. Bowel
    - 2.2.4.5. Muscular system
  - 2.2.5. Endocrine
    - 2.2.5.1. The HPA axis
  - 2.2.6. Male reproductive system
    - 2.2.6.1. Female reproductive system
  - 2.2.7. Immune system
- 2.3 Answers to check your Progress Questions
- 2.4 Summary
- 2.5 Key Words
- 2.6 Self-Assessment Questions and Answers
- 2.7 Further Readings

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### 2.0 INTRODUCTION

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We have discussed stress and its types along with its psychosomatic disorders in our earlier UNIT-I. Which help us to know about the stress and its managements.

In this unit we shall learn about the biological effects of stress on various organs such as digestive systems, esophagus, stomach, muscular system and reproductive systems. Hence, from this UNIT we can understand the importance of stress and its effects.

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### 2.1 OBJECTIVES

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After studying this unit, you will be able to know the following

- Describe the different biological effects on organs.

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### 2.2 BIOLOGICAL EFFECTS OF STRESS ON VARIOUS SYSTEMS

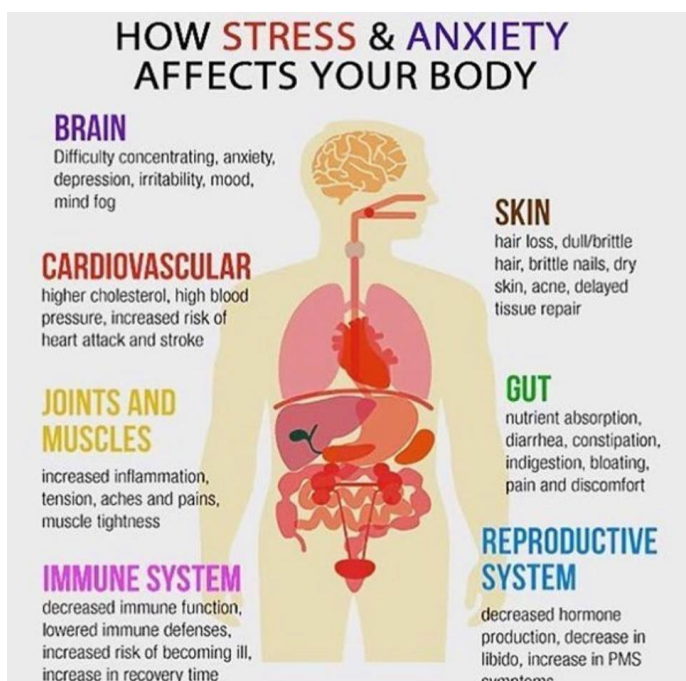
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Stress hormones affect your respiratory and cardiovascular systems. During the stress response, you breathe faster in an effort to quickly distribute oxygen-rich blood to your body. If you already have a breathing problem like asthma or emphysema, stress can make it even harder to breathe.

Stress and illness may have intersecting components. Several studies indicate such a link, while theories of the stress–illness link suggest that both acute and chronic stress can cause illness, and lead to changes in behavior and in physiology. Behavioral changes can include smoking, and changes in eating habits and physical activity. Physiological changes can include changes in sympathetic activation or HPA activity, and immunological function. However, there is much variability in the link between stress and illness.

The HPA axis regulates many bodily functions, both behavioral and physiological, through the release of glucocorticoid hormones. The HPA axis activity varies according to the circadian rhythm, with a spike in the morning. The axis involves the release of corticotropin releasing hormone and vasopressin from the hypothalamus which stimulates the pituitary to secrete ACTH. ACTH may then stimulate the adrenal glands to secrete cortisol. The HPA axis is subject to negative feedback regulation as well.

The release of CRH and VP are regulated by descending glutamnergic and GABAergic pathways from the amygdala, as well as noradrenergic projections. Increased cortisol usually acts to increase blood glucose, blood pressure, and surpasses lysosomal, and immunological activity. Under other circumstances the activity may differ. Increased cortisol also favors habit based learning, by favoring memory consolidation of emotional memories.



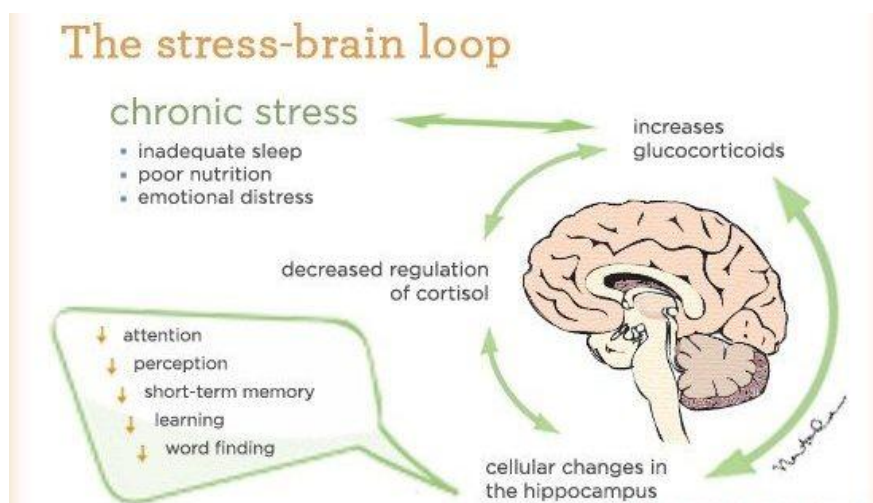
Selye demonstrated that stress decreases adaptability of an organism and proposed to describe the adaptability as a special resource, *adaptation energy*. One study considered adaptation energy as an internal coordinate on the "dominant path" in the model of adaptation. Stress can make the individual more susceptible to physical illnesses like the common cold. Stressful events, such as job changes, may result in

insomnia, impaired sleeping, and physical and psychological health complaints.

Research indicates the type of stressor (whether it is acute or chronic) and individual characteristics such as age and physical well-being before the onset of the stressor can combine to determine the effect of stress on an individual. An individual's personality characteristics (such as level of neuroticism), genetics, and childhood experiences with major stressors and traumas may also dictate their response to stressors.

### 2.2.1 BRAIN

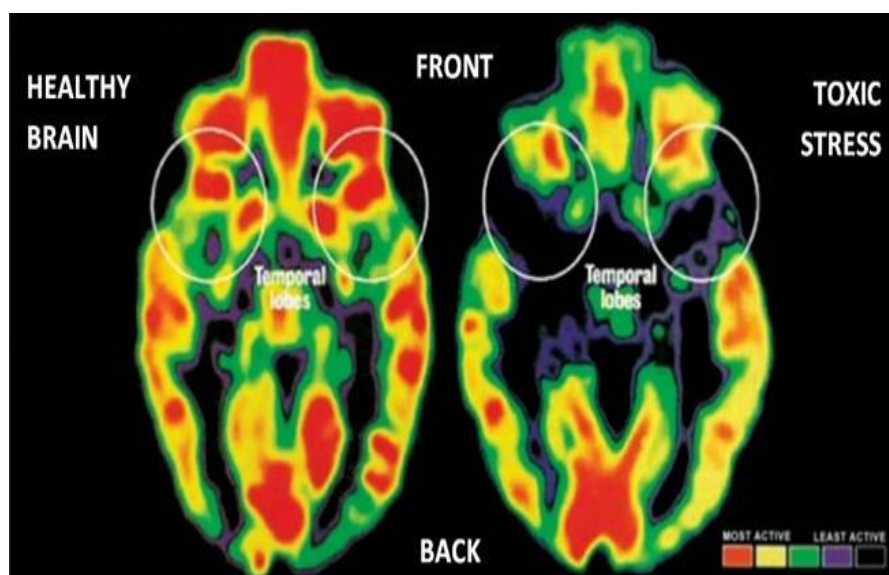
The nervous system has several divisions: the central division involving the brain and spinal cord and the peripheral division consisting of the autonomic and somatic nervous systems. The autonomic nervous system has a direct role in physical response to stress and is divided into the sympathetic nervous system (SNS), and the parasympathetic nervous system (PNS). When the body is stressed, the SNS contributes to what is known as the "fight or flight" response. The body shifts its energy resources toward fighting off a life threat, or fleeing from an enemy. The SNS signals the adrenal glands to release hormones called adrenalin (epinephrine) and cortisol. These hormones, together with direct actions of autonomic nerves, cause the heart to beat faster, respiration rate to increase, blood vessels in the arms and legs to dilate, digestive process to change and glucose levels (sugar energy) in the bloodstream to increase to deal with the emergency.



The SNS response is fairly sudden in order to prepare the body to respond to an emergency situation or acute stress, short term stressors. Once the crisis is over, the body usually returns to the pre-emergency, unstressed state. This recovery is facilitated by the PNS, which generally has opposing effects to the SNS. But PNS over-activity can also contribute to stress reactions, for example, by promoting bronchoconstriction (e.g., in asthma) or exaggerated vasodilation and compromised blood circulation. Both the SNS and the PNS have powerful interactions with the immune system, which can also modulate stress reactions. The central nervous system is particularly important in triggering stress-responses, as it

regulates the autonomic nervous system and plays a central role in interpreting contexts as potentially threatening.

Chronic stress, experiencing stressors over a prolonged period of time, can result in a long-term drain on the body. As the autonomic nervous system continues to trigger physical reactions, it causes a wear-and-tear on the body. It's not so much what chronic stress does to the nervous system, but what continuous activation of the nervous system does to other bodily systems that become problematic.



## 2.2.2. CARDIOVASCULAR SYSTEM

The heart and blood vessels comprise the two elements of the cardiovascular system that work together in providing nourishment and oxygen to the organs of the body. The activity of these two elements is also coordinated in the body's response to stress. Acute stress — stress that is momentary or short-term such as meeting deadlines, being stuck in traffic or suddenly slamming on the brakes to avoid an accident — causes an increase in heart rate and stronger contractions of the heart muscle, with the stress hormones — adrenaline, noradrenaline and cortisol — acting as messengers for these effects. In addition, the blood vessels that direct blood to the large muscles and the heart dilate, thereby increasing the amount of blood pumped to these parts of the body and elevating blood pressure. This is also known as the fight or flight response. Once the acute stress episode has passed, the body returns to its normal state.

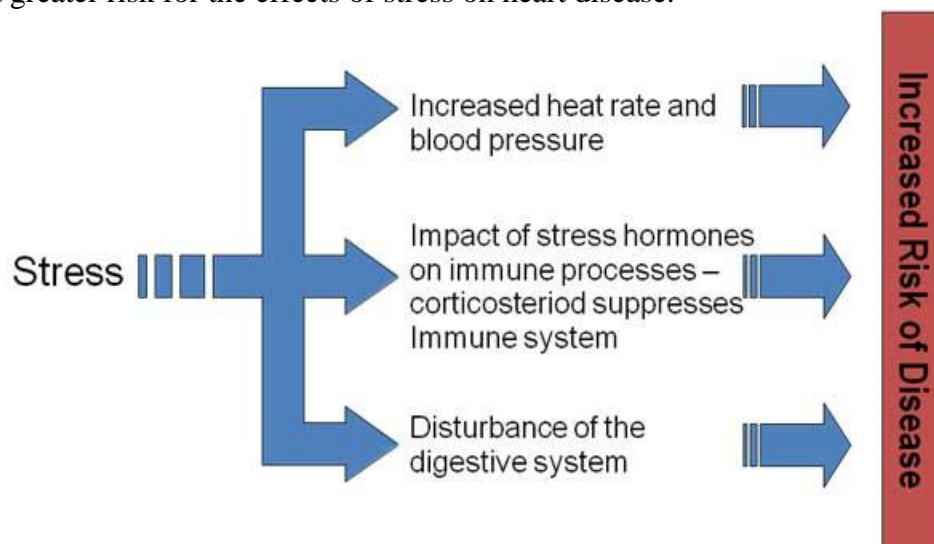
Chronic stress, or a constant stress experienced over a prolonged period of time, can contribute to long-term problems for heart and blood vessels. The consistent and ongoing increase in heart rate, and the elevated levels of stress hormones and of blood pressure, can take a toll on the body. This long-term ongoing stress can increase the risk for hypertension, heart attack or stroke.

Repeated acute stress and persistent chronic stress may also contribute to inflammation in the circulatory system, particularly in the



coronary arteries, and this is one pathway that is thought to tie stress to heart attack. It also appears that how a person responds to stress can affect cholesterol levels.

The risk for heart disease associated with stress appears to differ for women, depending on whether the woman is pre or postmenopausal. Levels of estrogen in premenopausal women appears to help blood vessels respond better during stress, thereby helping their bodies to better handle stress and protecting them against heart disease. Postmenopausal women lose this level of protection due to loss of estrogen, therefore putting them at greater risk for the effects of stress on heart disease.



### 2.2.3. RESPIRATORY SYSTEM

The respiratory system supplies oxygen to cells and removes carbon dioxide waste from the body. Air comes in through the nose and goes through the larynx in the throat, down through the trachea and into the lungs through the bronchi. The bronchioles then transfer oxygen to red blood cells for circulation.

Stress and strong emotions can present with respiratory symptoms, such as shortness of breath and rapid breathing, as the airway between the nose and the lungs constricts. For people without respiratory disease, this is generally not a problem as the body can manage the additional work to breathe comfortably, but psychological stressors can exacerbate breathing problems for people with pre-existing respiratory diseases such as asthma and chronic obstructive pulmonary disease (COPD; includes emphysema and chronic bronchitis).

Some studies show that an acute stress — such as the death of a loved one — can actually trigger asthma attacks. In addition, the rapid breathing — or hyperventilation — caused by stress can bring on a panic attack in someone prone to panic attacks.

Working with a psychologist to develop relaxation, breathing, and other cognitive behavioral strategies can help.

### 2.2.4. NON VITAL ORGANS

#### 2.2.4.1. DIGESTIVE SYSTEM

The gut has hundreds of millions of neurons which can function fairly independently and are in constant communication with the brain-

explaining the ability to feel “butterflies” in the stomach. Stress can affect this brain-gut communication, and may trigger pain, bloating and other gut discomfort to be felt more easily. The gut is also inhabited by millions of bacteria which can influence its health and the brain’s health which can impact the ability to think and affect emotions. Stress is associated with changes in gut bacteria which in turn can influence mood. Thus, the gut’s nerves and bacteria strongly influence the brain and vice versa.

Early life stress can change the development of the nervous system as well as how the body reacts to stress. These changes can increase the risk for later gut diseases or dysfunctioning.

#### **2.2.4.1.2. ESOPHAGUS**

When stressed, individuals may eat much more or much less than usual. More or different foods, or an increase in the use of alcohol or tobacco, can result in heartburn or acid reflux. Stress or exhaustion can also increase the severity of regularly occurring heartburn pain. A rare case of spasms in the esophagus can be set off by intense stress and can be easily mistaken for a heart attack. Stress also may make swallowing foods difficult or increase the amount of air that is swallowed, which increases burping, gassiness and bloating.

#### **2.2.4.1.3. STOMACH**

Stress may make pain, bloating, nausea and other stomach discomfort felt more easily. Vomiting may occur if the stress is severe enough. Furthermore, stress may cause an unnecessary increase or decrease in appetite. Unhealthy diets may in turn deteriorate one’s mood.

Contrary to popular belief, stress does not increase acid production in the stomach, nor causes stomach ulcers. The latter are actually caused by a bacterial infection. When stressed, the ulcers may be more bothersome.

#### **2.2.4.1.4. BOWEL**

Stress can also make pain, bloating or discomfort felt more easily in the bowels. It can affect how quickly food moves through the body which can cause either diarrhea or constipation. Furthermore, stress can induce muscle spasms in the bowel which can be painful.

Stress can affect digestion, and what nutrients the intestines absorb. Gas production related to nutrient absorption may increase. The intestines have a tight barrier to protect the body from (most) food related bacteria. Stress can make the intestinal barrier weaker and allow gut bacteria to enter the body. Although most of these bacteria are easily taken care of by the immune system and do not make us sick, the constant low need for inflammatory action can lead to chronic mild symptoms.

Stress especially affects people with chronic bowel disorders, such as inflammatory Bowel Disease or Irritable Bowel Syndrome. This may be due to the gut nerves being more sensitive, changes in gut microbiota, changes in how quickly food moves through the gut, and/or changes in gut immune responses.

#### **2.2.4.2. MUSCULAR SYSTEM**

When the body is stressed, muscles tense up. Muscle tension is almost a reflex reaction to stress — the body's way of guarding against injury and pain.

With sudden onset stress, the muscles tense up all at once, and then release their tension when the stress passes. Chronic stress causes the muscles in the body to be in a more or less constant state of guardedness. When muscles are taut and tense for long periods of time, this may trigger other reactions of the body and even promote stress-related disorders. For example, both tension-type headache and migraine headache are associated with chronic muscle tension in the area of the shoulders, neck and head. Musculoskeletal pain in the low back and upper extremities has also been linked to stress, especially job stress.

Millions of individuals suffer from chronic painful conditions secondary to musculoskeletal disorders. Often, but not always, there may be an injury that sets off the chronic painful state. What determines whether or not an injured person goes on to suffer from chronic pain is how they respond to the injury. Individuals who are fearful of pain and re-injury, and who seek only a physical cause and cure for the injury, generally have a worse recovery than individuals who maintain a certain level of moderate, physician-supervised activity. Muscle tension, and eventually, muscle atrophy due to disuse of the body, all promote chronic, stress-related musculoskeletal conditions.

Relaxation techniques and other stress-relieving activities and therapies have been shown to effectively reduce muscle tension, decrease the incidence of certain stress-related disorders, such as headache, and increase a sense of well-being. For those who develop chronic pain conditions, stress-relieving activities have been shown to improve mood and daily function.

#### **2.2.4.3. ENDOCRINE**

When someone perceives a situation to be challenging, threatening or uncontrollable, the brain initiates a cascade of events involving the hypothalamic-pituitary-adrenal (HPA) axis, which is the primary driver of the endocrine stress response. This ultimately results in an increase in the production of steroid hormones called glucocorticoids, which include cortisol, often referred to as the “stress hormone”.

##### **THE HPA AXIS**

During times of stress, the hypothalamus, a collection of nuclei that connects the brain and the endocrine system, signals the pituitary gland to produce a hormone, which in turn signals the adrenal glands, located above the kidneys, to increase the production of cortisol. Cortisol increases the level of energy fuel available by mobilizing glucose and fatty acids from the liver. Cortisol is normally produced in varying levels throughout the day, typically increasing in concentration upon awakening and slowly declining throughout the day, providing a daily cycle of energy. During a stressful event, an increase in cortisol can provide the energy required to deal with prolonged or extreme challenge.

## **STRESS AND HEALTH**

Glucocorticoids, including cortisol, are important for regulating the immune system and reducing inflammation. While this is valuable during stressful or threatening situations where injury might result in increased immune system activation, chronic stress can result in impaired communication between the immune system and the HPA axis. This impaired communication has been linked to the future development of numerous physical and mental health conditions, including chronic fatigue, metabolic disorders (e.g., diabetes, obesity), depression and immune disorders.

### **2.2.4.5. MALE REPRODUCTIVE SYSTEM**

The male reproductive system is influenced by the nervous system. The parasympathetic part of the nervous system causes relaxation whereas the sympathetic part causes arousal. In the male anatomy, the autonomic nervous system, also known as the fight or flight response, produces testosterone and activates the sympathetic nervous system which creates arousal. Stress causes the body to release the hormone cortisol, which is produced by the adrenal glands. Cortisol is important to blood pressure regulation and the normal functioning of several body systems including cardiovascular, circulatory and male reproduction. Excess amounts of cortisol can affect the normal biochemical functioning of the male reproductive system.

#### **Sexual desire**

Chronic stress, ongoing stress over an extended period of time, can affect testosterone production resulting in a decline in sex drive or libido, and can even cause erectile dysfunction or impotence.

#### **Reproduction**

Chronic stress can also negatively impact sperm production and maturation causing difficulties in couples who are trying to conceive. Researchers have found that men who experienced two or more stressful life events in the past year had a lower percentage of sperm motility (ability to swim) and a lower percentage of sperm of normal morphology (size and shape), compared with men who did not experience any stressful life events.

#### **Diseases of the reproductive system**

When stress affects the immune system, the body can become vulnerable to infection. In the male anatomy, infections to the testes, prostate gland and urethra, can affect normal male reproductive functioning.

### **2.2.4.6. FEMALE REPRODUCTIVE SYSTEM**

#### **Menstruation**

Stress may affect menstruation among adolescent girls and women in several ways. For example, high levels of stress may be associated with absent or irregular menstrual cycles, more painful periods and changes in the length of cycles.

### **Sexual desire**

Women juggle personal, family, professional, financial and a broad range of other demands across their life span. Stress, distraction, fatigue, etc., may reduce sexual desire — especially when women are simultaneously caring for young children or other ill family members, coping with chronic medical problems, feeling depressed, experiencing relationship difficulties or abuse, dealing with work problems, etc.

### **Pregnancy**

Stress can have significant impact on a woman's reproductive plans. Stress can negatively impact a woman's ability to conceive, the health of her pregnancy, and her postpartum adjustment. Depression is the leading complication of pregnancy and postpartum adjustment. Excess stress increases the likelihood of developing depression and anxiety during this time. Maternal stress can negatively impact fetal and ongoing childhood development and disrupt bonding with the baby in the weeks and months following delivery.

### **Premenstrual syndrome**

Stress may make premenstrual symptoms worse or more difficult to cope with and premenstrual symptoms may be stressful for many women. These symptoms include cramping, fluid retention and bloating, negative mood (feeling irritable and "blue") and mood swings.

### **Menopause**

As menopause approaches, hormone levels fluctuate rapidly. These changes are associated with anxiety, mood swings and feelings of distress. Thus menopause can be a stressor in and of itself. Some of the physical changes associated with menopause, especially hot flashes, can be difficult to cope with. Furthermore, emotional distress may cause the physical symptoms to be worse. For example, women who are more anxious may experience an increased number of hot flashes and/or more severe or intense hot flashes.

### **Diseases of the reproductive system**

When stress is high, there is increased chance of exacerbation of symptoms of reproductive disease states, such as: Herpes Simplex Virus or Polycystic Ovarian Syndrome. The diagnosis and treatment of reproductive cancers can cause significant stress, which warrants additional attention and support.

#### **2.2.4. IMMUNE SYSTEM**

The immune system is a collection of billions of cells that travel through the bloodstream. They move in and out of tissues and organs, defending the body against foreign bodies (antigens), such as bacteria, viruses and cancerous cells.

There are two types of lymphocytes:

- B cells- produce antibodies which are released into the fluid surrounding the body's cells to destroy the invading viruses and bacteria.
- T cells (see picture opposite) - if the invader gets inside a cell, these (T cells) lock on to the infected cell, multiply and destroy it.

The main types of immune cells are white blood cells. There are two types of white blood cells – lymphocytes and phagocytes.

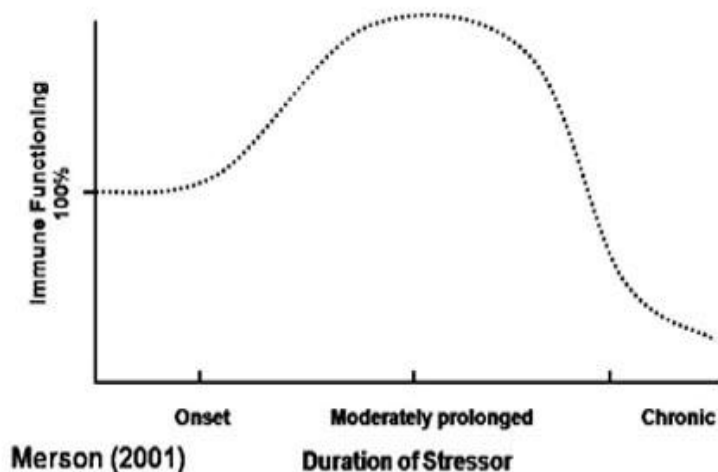
When we're stressed, the immune system's ability to fight off antigens is reduced. That is why we are more susceptible to infections.

The stress hormone corticosteroid can suppress the effectiveness of the immune system (e.g. lowers the number of lymphocytes).

Stress can also have an indirect effect on the immune system as a person may use unhealthy behavioral coping strategies to reduce their stress, such as drinking and smoking.

Stress is linked to: headaches; infectious illness (e.g. 'flu'); cardiovascular disease; diabetes, asthma and gastric ulcers.

Stress stimulates the immune system, which can be a plus for immediate situations. This stimulation can help you avoid infections and heal wounds. But over time, stress hormones will weaken your immune system and reduce your body's response to foreign invaders. People under chronic stress are more susceptible to viral illnesses like the flu and the common cold, as well as other infections. Stress can also increase the time it takes you to recover from an illness or injury.



Short term suppression of the immune system is not dangerous. However, chronic suppression leaves the body vulnerable to infection and disease.

A current example of this is AIDS - Acquired immune deficiency syndrome. Here the immune system is suppressed leaving the vulnerable to illness. Stress would just lead to frequent illness and infections.

Stress responses increase strain upon circulatory system due to increased heart rate etc. This may increase a person's risk of developing disorders of the heart and circulation e.g. Coronary Heart Disease (CHD). Individuals with type A personality have a greater risk of developing CHD.

Stress responses have an effect on digestive system. During stress digestion is inhibited. After stress digestive activity increases. This may affect the health of digestive system and cause gastric ulcers.

## STRESS MANAGEMENT

These recent discoveries about the effects of stress on health shouldn't leave you worrying. We now understand much more about effective strategies for reducing stress responses. Such beneficial strategies include:

1. Maintaining a healthy social support network.
2. Engaging in regular physical exercise.
3. Getting an adequate amount of sleep each night.

These approaches have important benefits for physical and mental health, and form critical building blocks for a healthy lifestyle. If you would like additional support or if you are experiencing extreme or chronic stress, a licensed psychologist can help you identify the challenges and stressors that affect your daily life and find ways to help you best cope for improving your overall physical and mental well-being.

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## 2.4 SUMMARY

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This unit provides us an understanding about the biological effects of stress on our body parts. Ongoing, chronic stress, however, can cause or exacerbate many serious health problems, including: Mental health problems, such as depression, anxiety, and personality disorders. Cardiovascular disease, including heart disease, high blood pressure, abnormal heart rhythms, heart attacks, and stroke

Stress is a familiar and common part of daily life. Stress happens each and every day and comes in a wide variety of forms. It might be the stress of trying to juggle family, work, and school commitments. It might involve issues like health, money, and relationships.

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## 2.5 KEY WORDS

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- **Brain:** The human brain is the central organ of the human nervous system, and with the spinal cord makes up the central nervous system.
- **Digestion:** Digestion involves the breakdown of food into smaller and smaller components, until they can be absorbed and assimilated into the body.

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## 2.6 SELF ASSESSMENT QUESTIONS AND ANSWERS

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### Short Answer Questions

1. Describe how stress affects the brain functions.
2. Write short notes on biological impacts of stress on male reproduction?
3. Explain how immune system affects by stress

### Long Answer Questions

1. Describe how stress affects our cardio vascular health.
2. Explain the clinical signs and symptoms of reproductive system affects by stress.

3. Explain in detail about the nervous system and no vital organs impacts on stress

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## **2.7 FURTHER READINGS**

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## UNIT-III

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### Structure

- 3.0 Introduction
- 3.1 Objectives
- 3.2. Stress enhancing foods
- 3.3. Anti stress food and nutrients
- 3.4. Dietary guidelines for the management of stress
- 3.5. Answers to Check Your Progress Questions
- 3.6. Summary
- 3.7. Key Words
- 3.8. Self-Assessment Questions
- 3.9. Further Readings

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### 3.0 INTRODUCTION

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Stress is a feeling of emotional or physical tension. It can come from any event or thought that makes you feel frustrated, angry, or nervous. Stress is your body's reaction to a challenge or demand. In short bursts, stress can be positive, such as when it helps you avoid danger or meet a deadline. But when stress lasts for a long time, it may harm your health.

Stress, in everyday terms, is a feeling that people have when they are overloaded and struggling to cope with demands. These demands can be related to finances, work, relationships, and other situations, but anything that poses a real or perceived challenge or threat to a person's well-being can cause stress.

Stress can be a motivator. It can be essential to survival. The "fight-or-flight" mechanism can tell us when and how to respond to danger. However, if this mechanism is triggered too easily, or when there are too many stressors at one time, it can undermine a person's mental and physical health and become harmful.

According to the annual stress survey conducted by the American Psychological Association (APA), average stress levels in the United States (U.S.) rose from 4.9 to 5.1 on a scale from 1 to 10 in 2015.

A brief note on stress have been given in Unit- I and Unit-II. We shall in detail discuss about the foods which are enhancing the stress and anti-stress foods along with guidelines.

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### 3.1. OBJECTIVES

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This unit will provide an insight to

- The foods which enhance the stress
- Anti-stress foods
- Dietary guidelines and its management

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### 3.2. STRESS ENHANCING FOODS

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Psychological stress is known to suppress immune function and increase susceptibility to infections and cancer. Paradoxically, stress is also known to exacerbate some allergic, autoimmune, and inflammatory diseases, which suggests that stress may enhance immune function under certain conditions.

When some body constantly stressed, our body remains in fight-or-flight mode, which triggers a host of physiological responses, including an increase in cortisol levels. And while that's helpful in situations like facing a wild grizzly bear head on, remaining in that stressed-out, fight-or-flight state over a long period of time can lead to health issues ranging from weight gain to increased inflammation (which is the root cause of most diseases) to elevated blood sugar. Chronic stress is also a cause of adrenal fatigue, a condition that happens when the body and adrenal glands struggle to keep up with the demands of external stressors, and can lead to symptoms like depression, inflammation, and poor focus.

As if that wasn't concerning enough, the reality is that it isn't just our environments or tough situations that cause stress. Indeed, certain foods can have the same negative impact on the body. So if you want to reduce stress levels and decrease your risk of disease, learning how to naturally relieve stress can help—as can steering clear of the following foods:

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#### 1. SUGAR

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If you want to reduce stress, sugar is one of the first ingredients to cut out of your diet. When you're stressed, the body releases more cortisol, a hormone responsible for helping us manage both stress and blood sugar levels. That's because when you eat sugary foods, blood sugar levels spike, and the body must release more cortisol to balance blood sugar. The problem is that increased cortisol can also cause sleep issues, decreased immune response, headaches, and unhealthy food cravings. Additionally, rapidly fluctuating blood sugar levels cause feelings that are similar to stress, including anxiousness and fear.

By eliminating foods with added sugars—like pastries, flavored yogurt, and soda—and eating more whole foods, you'll keep your blood sugar stable, which means fewer mood swings, reduced stress, and a happier body.

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#### 2. ARTIFICIAL SWEETENERS

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Sugar is bad enough on its own. But all too often, food products aren't even sweetened with the real thing—instead, they're packed with artificial sweeteners. These artificial sweeteners can lead to health problems like headaches, metabolic disease, and cardiovascular disease. But they can also result in an addiction to sugary foods by retraining your taste buds, causing you to seek out even sweeter (and largely unhealthy) foods.

If that wasn't bad enough, artificial sweeteners also have side effects that can lead to stress. Aspartame, for example, is found in more than 6,000 foods and drinks and in 500 prescription and over-the-counter

drugs, and it causes migraines, mood disorders, and manic episodes. And just like other types of sugar, these artificial sweeteners don't do your blood sugar any favors. Skip the sugar and fake sweeteners and try these natural sweeteners instead.

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### **3. PROCESSED CARBOHYDRATES**

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Processed, refined carbohydrates might taste good to your tongue, but they're not doing anything for your body. For starters, they have no nutritional value and are actually worthless calories. And they also lead to fluctuating blood sugar levels that can cause you to feel moody and irritable.

Many processed carbs, particularly standard packaged foods, are high in sodium. And aside from making you extra thirsty, that extra sodium causes your body to retain more fluid, which then forces your heart to work harder to keep the blood pumping. It can also increase blood pressure, making you feel bloated and generally unwell, which can certainly increase your stress levels.

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### **4. ALCOHOL**

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A glass of wine might help you feel more relaxed after an intense day, but have much more than that and you're likely negating any of alcohol's health benefits and, instead, adding more stress to your life. That's because drinking alcohol can increase the production of hormones that will leave you feeling anxious and more stressed than before you began imbibing. It can also increase blood pressure and heart rate and trigger those same stress-like symptoms within the body. Lots of alcoholic drinks are also packed with sugar, which means you'll be getting a double whammy of stress-causing ingredients—first from the alcohol and then from whatever way-too-sweet mixer is used in your cocktail.

And if you think you'll just sleep off the stress, that's unlikely. Alcohol disrupts your sleep patterns, so while you might fall asleep more easily than usual, you won't get the deep sleep that's necessary to feel refreshed. The result is a cranky morning after and—you guessed it!—more stress.

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### **5. EXCESS CAFFEINE**

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If you can't function without your morning cup of Joe, you don't need to give up coffee completely. But if you're regularly drinking several cups each day, you're likely going to find yourself feeling more stressed than you'd like. Too much caffeine can spell trouble for your adrenal glands by over stimulating the body. And because it stimulates the nervous system, caffeine can cause a rise in blood pressure and heart rate that will ultimately increase feelings of anxiety. In fact, if you suffer from anxiety, one of the first things you should do is cut out caffeine.

Finally, it's important to remember that caffeine isn't found in just coffee. It's also in soft drinks, certain types of tea, energy drinks, over-the-counter pain relievers, and even chocolate.

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### 3.3. ANTI-STRESS FOOD AND NUTRIENTS

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#### 1. Brazil nuts



Brazil nuts contain selenium, which may help to improve mood.

Brazil nuts are high in selenium. Selenium may improve mood by reducing inflammation, which is often at heightened levels when someone has a mood disorder, such as anxiety.

Selenium is also an antioxidant, which helps prevent cell damage. It is also anti-carcinogenic, which helps to prevent cancer from developing.

Other nuts, animal products, and vegetables, such as mushrooms and soybeans, are an excellent source of selenium.

It is important not to consume too much selenium as it can cause side effects. The recommended upper limit for selenium for an adult is 400 micrograms (mcg) per day. So be careful not to take supplements with high doses or eat more than a three to four Brazil nuts a day.

Brazil nuts and other nuts are also a good source of vitamin E. Vitamin E is an antioxidant. Antioxidants can be beneficial for treating anxiety, while some research has shown that low levels of vitamin E may lead to depression in some people.

#### 2. Fatty fish

Fatty fish, such as salmon, mackerel, sardines, trout, and herring, are high in omega-3. Omega-3 is a fatty acid that has a strong relationship with cognitive function as well as mental health.

However, recent research has shown that if a person eats too much of another fatty acid, called omega-6, and not enough omega-3, they may increase their risk of developing mood disorders, such as anxiety.

Omega-3-rich foods that contain alpha-linolenic acid (ALA) provides two essential fatty acids: eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA).

EPA and DHA regulate neurotransmitters, reduce inflammation, and promote healthy brain function.

A small study on 24 people with substance abuse problems found that EPA and DHA supplementation resulted in reduced levels of anxiety. However, more research is required.

Current recommendations suggest eating at least two servings of fatty fish a week. A study conducted on men found eating salmon three times a week reduced self-reported anxiety.

Salmon and sardines are also among the few foods that contain vitamin D.

### **Vitamin D**

Researchers are increasingly linking vitamin D deficiency to mood disorders, such as depression and anxiety. A report in the *Journal of Affective Disorders* believes that there is enough evidence to prove that vitamin D positively helps depression. Other studies on pregnant women and older adults have also highlighted how vitamin D might improve mood. Vitamin D may also improve seasonal disaffected disorder (SAD) during winter.

### **3. Eggs**

Egg yolks are another great source of vitamin D.

Eggs are also an excellent source of protein. It is a complete protein, meaning it contains all the essential amino acids the body needs for growth and development.

Eggs also contain tryptophan, which is an amino acid that helps create serotonin. Serotonin is a chemical neurotransmitter that helps to regulate mood, sleep, memory, and behavior. Serotonin is also thought to improve brain function and relieve anxiety.

### **4. Pumpkin seeds**

Pumpkin seeds are an excellent source of potassium, which helps regulate electrolyte balance and manage blood pressure. Eating potassium-rich foods such, as pumpkin seeds or bananas, may help reduce symptoms of stress and anxiety.

Pumpkin seeds are also a good source of the mineral zinc. One study carried out on 100 female high school students found that zinc deficiency may negatively affect mood.

Zinc is essential for brain and nerve development. The largest storage sites of zinc in the body are in the brain regions involved with emotions.

### **5. Dark chocolate**



Researchers have found that dark chocolate may help reduce stress. Experts have long suspected that dark chocolate might help reduce

stress and anxiety. A 2014 study found that 40g of dark chocolate helped reduce perceived stress in female students.

Other studies have generally found that dark chocolate or cocoa may improve mood. However, many of these studies are observational, so the results need to be interpreted with caution.

Although it is still unclear how dark chocolate reduces stress, it is a rich source of polyphenols, especially flavonoids. One study suggested that flavonoids might reduce neuroinflammation and cell death in the brain as well as improve blood flow.

Chocolate has a high tryptophan content, which the body uses to turn into mood-enhancing neurotransmitters, such as serotonin in the brain. Dark chocolate is also a good source of magnesium. Eating a diet with enough magnesium in it or taking supplements may reduce symptoms of depression.

When choosing dark chocolate, aim for 70 percent or more. Dark chocolate still contains added sugars and fats, so a small serving of 1 to 3 grams (g) is appropriate.

## 6. Turmeric

Turmeric is a spice commonly used in Indian and South-East Asian cooking. The active ingredient in turmeric is called curcumin. Curcumin may help lower anxiety by reducing inflammation and oxidative stress that often increase in people experiencing mood disorders, such as anxiety and depression. A 2015 study found that curcumin reduced anxiety in obese adults.

Another study found that an increase of curcumin in the diet also increased DHA and reduced anxiety. Turmeric is easy to add to meals. It has minimal flavor, so goes well in smoothies, curries, and casserole dishes.

## 7. Chamomile

Many people around the world use chamomile tea as an herbal remedy because of its anti-inflammatory, antibacterial, antioxidant, and relaxant properties.

Some people believe that the relaxant and anti-anxiety properties come from the flavonoids present in chamomile. A recent study found that chamomile did reduce anxiety symptoms. However, it did not prevent new episodes of anxiety.

Chamomile tea may be useful in managing anxiety. It is readily available and safe to use in high doses.

## 8. Yogurt

Yogurt contains healthful bacteria, *Lactobacillus* and *Bifidobacteria*.

There is emerging evidence that these bacteria and fermented products have positive effects on brain health.

According to a recent clinical review, yogurt and other dairy products may also produce an anti-inflammatory effect in the body. Some research suggests that chronic inflammation may be partly responsible for anxiety, stress, and depression.

A 2015 study found fermented foods reduced social anxiety in some young people, while multiple studies found consuming healthful bacteria increased happiness in some people.

Including yogurt and other fermented food in the diet can benefit the natural gut bacteria and may reduce anxiety and stress.

Fermented foods include cheese, sauerkraut, kimchi, and fermented soy products.

### **9. Green tea**

Green tea contains an amino acid called theanine, which is receiving increasing scrutiny due to its potential effects on mood disorders. Theanine has anti-anxiety and calming effects and may increase the production of serotonin and dopamine.

A 2017 review found that 200 mg of theanine improved self-reported relaxation and calmness while reducing tension in human trials.

Green tea is easy to add to the day-to-day diet. It is a suitable replacement for soft drinks, coffee, and alcoholic beverages.

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### **Other foods that may help**

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Swiss chard contains magnesium, which may help to ease anxiety.

Eat a varied and balanced diet with high quality, nutrient-dense carbohydrates, fats, and proteins.

Aim for whole foods, vegetables, fruit, legumes, whole grains, lean meats, and especially fish. Other foods that may help include:

- Turkey and other tryptophan-containing foods such as eggs, dark chocolate, cheese, pineapple, bananas, oats, and tofu.
- Nuts, especially almonds are an excellent source of vitamin E. Vitamin E deficiency has been linked to mood disorders.
- Chia seeds are also a good source of omega-3s.
- Protein sources, such as lean meat, fish, nuts, and dairy all provide amino acids, which the body converts into the mood-lifting neurotransmitters, such as serotonin.
- Spinach and Swiss chard are both high in magnesium.
- Cinnamon provides anti-inflammatory properties

Evidence increasingly shows that diets high in processed foods can increase anxiety.

### **3.4. DIETARY GUIDELINES FOR MANAGEMENT OF STRESS**

Management of Stress through Nutrients Management of stress may be a powerful tool for staying healthy. Researchers have investigated the relationships between stress and many different medical problems, such as cardiovascular diseases, diabetes [8], and cholesterol levels [9]. Because obesity is an underlying factor in these medical conditions, researchers [10-12] have often studied the role of stress in individuals' eating behaviours, such as the amount eaten and the types of foods consumed. Stress creates greater physiological demands. More energy, oxygen, circulation, and therefore more metabolic cofactors are needed (e.g. vitamins and minerals). The irony of stress is that people suffering stress need a more nutritionally dense diet but often opt for comfort foods (like sugary and fatty foods) lacking in the necessary nutrients, consequently inducing a situation of nutrient depletion that further compromises the metabolic systems. Stress not only influences the choice of food of a person but also the quantity of the food eaten. Role of specific nutrient in regulation of food intake, in the maintenance of homeostatic mechanisms and emotional processes is very dense. Serotonin (5-hydroxytryptamin or 5-HT) is synthesized from the dietary amino-acid tryptophan (TRP). Likewise, tyrosine is a precursor of noradrenaline (NA). Psychosocial and physical stress increases the rate of release of noradrenaline (NA) in both the periphery and the central nervous system hence more protein especially tyrosine is required. Likewise various other nutrients are required to reduce the levels of the stress chemicals (cortisol and adrenaline) that activate fight and flight response in the body. A detail description of various nutrients and role in coping with stress is mentioned as under:

#### **Complex carbohydrates-whole grains, vegetables and fruits will boost levels of serotonin, a chemical in the brain that makes us calm**

Stabilizing blood pressure as a way to reduce stress. Serotonin (5-hydroxytryptamin or 5-HT) concentrations rise when TRP is directly administered or when the diet is rich in carbohydrate (CHO) and poor in protein (a CR-PP diet). Depletion of the precursor of serotonin synthesis, tryptophan, has been found to increased depressive mood in healthy subjects and subjects with a prior history of depressions. Hence, increases in 5-HT may enhance the capacity to respond to stress and prevent further degeneration in terms of mood. Complex carbohydrates also contributes to fibre. Fiber helps to maintain good stomach and digestive functioning. It make the food stay longer in stomach hence the more slowly the body absorbs carbohydrate, the more steadily serotonin flows.

#### **Omega 3 fatty acids**

The brain needs omega 3 fatty acids for the formation of healthy nerve cells. It has also been reported that omega 3 fatty acids are associated with a lower risk of depression. Evidence from epidemiological, laboratory and clinical studies suggest that dietary lipids and other associated nutritional factors may influence vulnerability and outcome in depressive disorders. Flaxseed, hemp, canola and walnut oils are all generally rich sources of the parent omega-3, alpha linolenic acid (ALA). Dietary ALA can be metabolized in the liver to the longer-chain omega-3



eicosapentaenoic (EPA) and docosahexaenoic acid (DHA). His conversion is limited in human beings, it is estimated that only 5-15% of ALA is ultimately converted to DHA. Stress compromise this conversion. DHA (Docosahexaenoic acid) an essential component of the membrane of brain cells, enhances brain to utilize various chemicals and can turn on the genes that make serotonin. It is a good natural anti-depressant.

### **Proteins**

#### **Tryptophan:**

Tryptophan is an amino acid found in milk as well as in many proteins rich foods like whole grains. Serotonin is synthesized from tryptophan. Tryptophan works with vitamin B6, niacin and magnesium to synthesize serotonin. If too little tryptophan is available for the brain, then it may limit the amount of serotonin. To make tryptophan-laden meals more executive, make them high in complex carbohydrates but medium to low in protein. Carbohydrate makes tryptophan more available in the brain but protein has the opposite effect.

#### **Phenylalanine and tyrosine:**

Phenylalanine and tyrosine promote alertness, vitality and help in increasing the rate at which brain neurons produce antidepressants-dopamine and norepinephrine. Vitamin C is required to metabolize phenylalanine and tyrosine effectively. This show, rich source of vitamin C like citrus fruits may help in metabolism of phenylalanine and tyrosine. Tofu, dairy products, bananas, avocados, lima beans, pumpkin seeds, sesame seeds and almonds are the main sources of phenylalanine and tyrosine.

#### **Heanine:**

Heanine has been studied for its potential ability to reduce mental and physical stress, improve cognition, and boost mood and cognitive performance in a synergistic manner with casein [19]. It relaxes the brain, thereby reducing stress and anxiety with tranquilizing effects. L-theanine significantly increases activity in the alpha frequency band which indicates that it relaxes the mind without inducing drowsiness. However, this effect has only been established at higher doses than that typically found in a cup of black tea (approximately 20 mg). Tea is a good source of threonine.

#### **Vitamin C:**

Both emotional and physical stress may affect a person's Vitamin C status. It can increase requirement for vitamin C to maintain normal blood levels. When stress depletes vitamin C levels in the body, it reduces the body's resistance to infection and disease and increases the likelihood of further stress. When vitamin C intake is increased, the harmful effects of the stress hormones are reduced and the body's ability to cope with the stress response improves. Vitamin C helps to recover more quickly from emotional and physical stress, which may otherwise weaken adrenal glands and increase fatigue. A randomized placebo-controlled study was carried out by Peters et al. [2001] shows that marathon, runners receiving 1500 mg of vitamin C per day recovered normal cortisol levels more rapidly than those taking only 500 mg or the placebo. Because pumping out of cortisol stresses the adrenal glands, faster recovery means less fatigue. Vitamin C is also believed to be a stress buster and reduces the stress by supporting the adrenal glands and allows a person to bounce back more quickly. Brody et

al. [2002] in a randomized, double blind, placebo controlled trial on stress of public speaking concluded that those who received vitamin C supplements experienced less stage fright and showed a faster recovery of cortisol levels, indicating that the adrenal glands, which produce cortisol, the stress hormone, were functioning better.

### **Vitamin B:**

The majority of the B-vitamins function in the development and maintenance of the nervous system. The harmful effects of vitamin-B-deficiencies on the nervous system might increase the risk of developing stress-related symptoms such as irritability, lethargy, and depression. They also help maintain regular blood-sugar levels to help keep your energy and mood stable. Among B-vitamins, most important is vitamin B5 (pantothenic acid) which is often called anti-stress vitamin B5 helps support the adrenal glands and improves coping mechanisms. Some of the studies show that vitamin B12 may ease the mood changes. Almonds are packed with B and E vitamins, which help boost the immune system, and walnuts and pistachios help lower blood pressure. One of the B vitamins is folic acid which is believed to relieve stress, anxiety, panic and even depression. Folic acid deficiencies have been found to contribute to mental illness. Folic acid is present in kidney beans, whole meal bread, broccoli, brussel sprouts, dark green cabbage, chicory, peanuts, peas, egg yolks and green leafy vegetables. Asparagus is high in B vitamins and folic acid. Niacin, also referred to as nicotinamide, is not to be confused with nicotine from tobacco. A deficiency of niacin adversely affects tissue respiration and oxidation of glucose and results in the disease known as pellagra in humans. This is characterized by skin and mucous membrane disorders as well as depression and confusion. Pellagra can be cured by feeding niacin or by feeding the essential amino acid tryptophan from which niacin can be made in the body. Good sources of this vitamin are yeast, meat, fish, poultry, peanuts, legumes and whole grain cereals etc.

### **Magnesium:**

Magnesium is needed for a variety of tasks such as muscle relaxation, fatty acid formation, making new cells and heartbeat regulation. Stress and magnesium are said to be interrelated. Both physical and psychological stress may stimulate the stress hormones. This, in turn, increases magnesium loss from the cells (especially from the heart and other vital organs), stimulate urinary excretion and increase dietary requirements for the magnesium.

### **Selenium:**

Selenium is a mineral involved in the reactions which release energy from cells. Its deficiency may cause fatigue. Brazil nuts and also whole grains (if grown in selenium rich soil) are rich in selenium content. Adequate supply of vitamin E increases the effectiveness of selenium. Selenium have an impact on the function of the adrenal glands. Research shows that deficiencies of selenium can have a negative effect on adrenal function. In order to consume the following nutrients, a person needs to adopt a particular defined of eating plan or diet. Following a strict plan and including the under given food will strengthen the body against stress and other illnesses that are thrust upon the body.

### **Stress-busting foods**

Foods can elicit an emotional response when eaten. Foods can help tame stress in several ways. Comfort foods, like a bowl of warm oatmeal, boost levels of serotonin, a calming brain chemical. Other foods can cut levels of cortisol and adrenaline, stress hormones that take a toll on the body over time. A healthy diet can help counter the impact of stress by shoring up the immune system and lowering blood pressure. Few stress-busting foods are as follows:

**Oranges:** Oranges are a rich source of vitamin C. Stressed bodies are more prone to free radical formations. Vitamin C helps to keep free radicals in check and repairs the body. Basically, it helps protect the body from the cumulative effects of stress. Also, vitamin C lowers blood pressure and stress hormone cortisol. Orange juice contains folic acid, which helps to relieve stress. Drinking plenty of orange juice will help in the production of dopamine in the body and make the person feel relaxed.

**Spinach:** Spinach is considered to be a magic cocktail of all the greens. Being a rich source of magnesium (three cups of spinach supply about 40% of daily magnesium), it helps to lower stress levels by keeping a person in a calm state and by preventing blood pressure from spiking. Spinach is loaded with vitamin C (just a half-cup of raw spinach gives as close to 50% of recommended daily value (DV) for vitamin C), hence lowers blood pressure and stress hormone cortisol. Spinach is one of the richest food sources of folic acid (vitamin B9).

### **Chocolate:**

Consuming dark chocolate reduces stress in two ways—its chemical impact and its emotional impact. Chocolate not only plays a role in fighting off free radicals, but it can affect both mind and mood. Chocolate is a complex material, possessing numerous compounds that act upon the brain, producing a sense of delight that no other substance can replicate. Cocoa beans are also one of nature's most concentrated sources of theobromine, a molecular cousin of caffeine and theophylline, present in coffee and tea. Theobromine has been proved to be safe for human consumption. Cocoa liquor and cocoa butter are high in antioxidants, beneficial in lowering cholesterol levels, boosting blood flow, reducing hypertension, and also a good source of minerals. Phenylethylamine (PEA) present in cocoa increases the activity of neurotransmitters. It also contains a high level of epicatechin. Cocoa beans contain nutrients essential to human mental and physical health such as iron, magnesium, potassium, phosphorus, zinc, and polyphenols, mainly flavonoids. Chocolate's serotonin-elevating activity helps to modify mood in a positive way. Commonly known as a comfort food, research has now promoted the status of chocolate as a psycho-active food. It has been discovered that in addition to anandamide that is present in ice-cream, chocolate is also loaded with positive neurotransmitters such as oleoethanolamine, N-linoleoylethanolamine, etc. These chemicals produce active psychological effects in the consumer. Chocolates are also rich in tryptophan, which forms a rate-limiting step in the production of the mood-modulating neurotransmitter serotonin, known to diminish anxiety. It has also found use in the treatment of pre-menstrual syndrome (PMS) due to its rich content of

magnesium that lowers progesterone levels. Chocolate also contains amino acid gamma-aminobutyric acid (GABA) that is said to reduce anxiety.

### **Coffee**

Epidemiological and experimental studies have shown positive effects of regular coffee-drinkers on various aspects of health, such as psychoactive responses (alertness, mood change etc.). Caffeine, an alkaloid, is the most widely consumed stimulant of coffee. According to numerous medical studies, caffeine is beneficial to overall health of human beings. It stimulates the central nervous system and flow of blood in the brain and increases the secretion of serotonin. Caffeine enhances alertness, facilitates thought formation, and decreases fatigue. This alkaloid also improves mood, lifts the spirits, and enhances both cardiovascular function and respiration. Taken by adults at a dose of 300 mg/day or less, caffeine is safe and beneficial for human health.

### **Blueberries:**

Blueberries are full of antioxidants and vitamin C. These nutrients are said to be great stress busters. The antioxidants fight the free radicals which adversely affect the memory. Vitamin C along with antioxidants helps to combat stress hormone cortisol. It is also the fiber present in blueberries which help to relieve stress. Also, the high fiber content keeps sugar level low and, therefore, stress is relieved. Broccoli: One of the good mood foods is broccoli which has stress relieving vitamin B6. It also contains folic acid which is important in fighting depression. Fish: Fish like Mackerel, Salmon, tuna sardines contains omega 3 fatty acids which boost the levels of serotonin, a neurotransmitter for good mood. It also has stress fighters like B6 and B12. These are important for the optimum functioning of the brain and enhance memory and mood. Banana: Banana offers serious mood lifting power, with a combination of Vitamins B6, A and C; fiber, tryptophan, potassium, phosphorus, iron and protein. The combination of natural sugars and fibers creates long-lasting energy to help in prevention of blood sugar imbalance. Carbohydrates aid in the absorption of tryptophan in the brain, Vitamin B6 helps in conversion of tryptophan into mood-lifting serotonin and the potassium and iron work towards of fatigue by producing more energy. Iron in bananas exclusively is crucial to producing energy and fighting fatigue.

### **Walnuts:**

Walnuts have long been thought of as a 'brain food' because of their wrinkled, bi-lobed (brain like) appearance. They are an excellent source of omega 3 essential fatty acids and uridine. The combination of omega 3 fatty acids and uridine is thought to be a natural antidepressant. Walnuts also contain some other compounds like vitamin B6, tryptophan, protein, and folic acid which contribute to stress releasing. Higher blood levels of omega 3 fatty acids have been linked with better mood and lower rates of depression.

### **Eggs:**

A hard-boiled egg is easy to make and easy to transport as snack food product. Full of high-quality protein and omega 3 fatty acids (from the hens eating omega 3 fatty acids rich diet), eggs are also an excellent source of vitamin B12 and a good source of vitamins B2, B5, and vitamin

D. One boiled egg also contains more than 20 percent of the daily recommended amount of tryptophan hence considered a good stress busting food. Tea: Green tea contains L-theanine a protein which relaxes the brain, thereby reducing stress and anxiety with tranquilizing effects. Consumption of 50 mg of L-theanine (equivalent to two-three cups of tea) has shown to stimulate the alpha-brain waves. The beta-brain waves are associated with reduction of tension. It also contains an active component, epigallocatechingallate (EGCG) which has been associated with reduced physical and mental fatigue.

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#### **Flax seeds:**

Flaxseeds (*Linum usitatissimum*) has a warm, earthy and subtly nutty flavour combined with an abundance of omega 3 fatty acids make it a good choice by vegetarians and a good brain food. Flaxseeds are rich in alpha linolenic acid (ALA), an omega 3 fatty acid that is a precursor of eicosapentaenoic acid or EPA (omega 3 fatty acids found in fish oils). Therefore, along with ALA flaxseed provides several beneficial effects of EPA also. Flax seeds are the richest source of omega 3 fatty acids in the plant kingdom and are very promising functional food.

#### **Whole grains:**

Whole grains are the rich source of carbohydrates. Carbohydrates are used as a comfort food because it makes a chemical (serotonin) that comforts a person. Carbohydrates cause the body to make insulin, which allows tryptophan (precursor of serotonin) to get into the brain. Serotonin is considered to be the brain's natural "feel good" chemical and appetite suppressant. Complex carbohydrates take longer time to get digested and, therefore, keep a person calm for longer period of time. Complex carbohydrates also stabilizing blood sugar level. Turkey: Turkey contains precursor amino acid gets converted into dopamine in human body which elevates the mood and motivates the subjects. This acid is a good antidepressant and also helps us feel sharper and better.

#### **Probiotics:**

Probiotics may be defined as "a viable mono or mixed culture of bacteria which, when applied to animal or man, beneficially affects the host by improving the properties of the indigenous flora. There are numerous reasons for stress and its effect on the body is that it reduces the microflora in the gut and increases the growth of pathogenic bacteria. Probiotics exert their effect by inversely increasing the good bacteria, reducing the bad bacteria, improving barrier function, visceral senility and gut motility. Probiotic bacteria have the potential to alter brain neurochemistry and treat anxiety and depression-related disorders.

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### **3.4 SUMMARY**

This unit provides us an understanding about the biological effects of stress on our body parts. Ongoing, chronic stress, however, can cause or exacerbate many serious health problems, including: Mental health problems, such as depression, anxiety, and personality disorders. Cardiovascular disease, including heart disease, high blood pressure, abnormal heart rhythms, heart attacks, and stroke

Stress is a familiar and common part of daily life. Stress happens each and every day and comes in a wide variety of forms. It might be the stress of trying to juggle family, work, and school commitments. It might involve issues like health, money, and relationships.

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### 3.5 KEY WORDS

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- **Anti-stress:** Serving to prevent or alleviate **stress** and especially emotional or physical **stress**.
- **Stress foods:** Stress can take a toll on your body's natural defenses, but eating the right foods can offer relief.

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### 3.6 SELF ASSESSMENT QUESTIONS AND ANSWERS

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#### Short Answer Questions

1. Describe stress enhancing foods.
2. Write short notes on anti stress foods?
3. Explain about the management of stress.

#### Long Answer Questions

1. Describe how stress can be managed by using food and nutrients.
2. Explain about antistress foods in details

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### 3.7 FURTHER READINGS

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# UNIT-IV NUTRITION FOR WEIGHT MANAGEMENT

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## Structure

- 4.0 Introduction
- 4.1 Objectives
- 4.2 Nutrition for weight Management
  - 4.2.1. Components of body weight
  - 4.2.2. Adipose tissue and regulating of body weight
- 4.3 Obesity
  - 4.3.1 Assessment
  - 4.3.2 Types
  - 4.3.3. causes and complication
- 4.6 Answers to Check your Progress Questions
- 4.7 Summary
- 4.8 Key Words
- 4.9 Self-Assessment Questions
- 4.10 Further Readings

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## 4.0 INTRODUCTION

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The most important component of an effective weight-management program must be the *prevention* of unwanted weight gain from excess body fat. The military is in a unique position to address prevention from the first day of an individual's military career. Because the military population is selected from a pool of individuals who meet specific criteria for body mass index (BMI) and percent body fat, the primary goal should be to foster an environment that promotes maintenance of a healthy body weight and body composition throughout an individual's military career. There is significant evidence that losing excess body fat is difficult for most individuals and the risk of regaining lost weight is high. From the first day of initial entry training, an understanding of the fundamental causes of excess weight gain must be communicated to each individual, along with a strategy for maintaining a healthy body weight as a way of life.

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## 4.1 OBJECTIVES

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After studying this unit, you should be able to answer the following:

- Nutrition and weight management
- Components of body weight techniques for measuring body composition.
- Adipose tissue and regulations of body weight
- Obesity and assessment of obesity
- Indirect, direct and critical method
- Complications in obesity
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## 4.2 NUTRITION AND WEIGHT MANAGEMENT

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Weight management is the phrase used to describe both the techniques and underlying physiological processes that contribute to a person's ability to attain and maintain a certain weight. Most weight management techniques encompass long-term lifestyle strategies that promote healthy eating and daily physical activity. Moreover, weight management involves developing meaningful ways to track weight over time and to identify ideal body weights for different individuals.

Due to the rising obesity rates in many parts of the world, proper weight management strategies most often focus on achieving healthy weights through slow but steady weight loss, followed by maintenance of an ideal body weight over time.

Rising obesity rates are a major concern around the world, especially in North America. About 60% of Americans and Canadians are either overweight or obese. Understanding the basic science of weight management and some of the strategies for attaining and maintaining a healthy weight is very important to a person's overall health because obesity is a risk factor for many chronic diseases, like Type 2 diabetes, hypertension and cardiovascular disease.

The detailed about weight management is going to study in this UNIT -IV.

Let us go in deeply into the chapters discussed under this heading.

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## 4.3 COMPONENTS OF BODYWEIGHT

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The difference is that a standard scale performs one measurement—your total body weight. A body composition scale breaks down that total body weight into the various elements that make it up. Your body is composed of three main components: fat, lean body mass (muscle, bone, and organs), and water.

Body composition and growth are key components of health in both individuals and populations. The ongoing epidemic of obesity in children and adults has highlighted the importance of body fat for short term and long term health.<sup>1</sup> However, other components of body composition also influence health outcomes, and its measurement is increasingly considered valuable in clinical practice. The gold standard for body composition analysis is cadaver analysis, so no in vivo technique may be considered to meet the highest criteria of accuracy. As discussed below, only multicomponent models are now considered sufficiently accurate to act as reference or criterion methods for the molecular approach to measuring body composition (distinguishing fat and fat-free masses), against which other methods should be evaluated. Several techniques are available, varying in complexity and ease of use, and each making assumptions that may affect its suitability for different conditions. A single technique is unlikely to be optimal in all circumstances. A further important issue is that of the difficulty of validating techniques in humans. In vivo techniques do not measure body composition directly, but rather predict it from



measurements of body properties. Thus all techniques suffer from two types of error: methodological error when collecting raw data, and error in the assumptions by which raw data are converted to final values. The relative magnitude of these errors varies between techniques.

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## **COMPONENTS OF BODYWEIGHTTECHNIQUES FOR MEASURING BODY COMPOSITION**

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### **1. Weight**

Weight is an overall measure of your body mass. This measurement includes all of the elements of your body – bones, blood, organs, muscles, and fat. A number of different factors contribute to your weight, including hereditary components, hormonal abnormalities, exercise, diet, and lifestyle. Even so, measuring body weight is a pretty straightforward calculation, usually made using a scale. Being underweight or overweight can significantly impact your physical and psychological wellbeing, so it is an important component in considering your overall health and wellness.

### **2. Body Fat**

A component of the body that most “dieters” want to get rid of, body fat is actually an important component of the body for overall health. More technically known as adipose tissue, body fat is a spongy tissue that is stored in the bones, organs, central nervous system, and muscles. The amount needed in the body is different for men and women. Generally speaking, men should have at least 2 to 5 percent body fat while healthy women need anywhere from 10 to 13 percent for essential bodily functions. The excess fat in the body is known as nonessential fat and is where excess energy is stored. There are different ways to estimate or measure body fat, but one of the most effective calculations is to measure body fat by percentage.

### **3. BMI**

Your BMI, or Body Mass Index, is a measurement of your weight versus your height. Your BMI is an important factor in considering overall health, as it can be an indicator of high overall body fat. This calculation can also be used to screen for specific health conditions that may be related to disproportionate weight. BMI is calculated by dividing your weight by your height.

### **4. Body Water**

Body water is an important physiological measure that can significantly impact overall health. This factor is a measure of the water content in the various tissues, blood, bones, and other components of your body. This water contributes significantly to the human body both in terms of weight and volume. Maintaining the right amount of water in your body is part of fluid balance and homeostasis. The average adult male is approximately 70 percent water; however, most adults fall somewhere below 65 percent. A body fat scale is one of the only ways to estimate your total body water percentage at home.

### **5. Lean Mass**

Your lean body mass is a calculation of the amount of weight your body carries that isn't fat. Bodybuilders and fitness enthusiasts often focus on dropping weight while maintaining lean body mass;

however, it can also present important information about your overall health. To measure your lean body mass, you must first identify your overall body fat percentage.

### **6. Visceral Fat Rating**

Visceral fat is an extremely important calculation that present information regarding your overall health and your potential to develop a number of alarming health conditions. Visceral fat can be described as the body fat that surrounds the waist. It is stored deep under the skin and is generally wrapped around major organs, such as your liver, pancreas, and kidneys. This is an important component of your body, as it ensures there is appropriate distance between each organ. But too much visceral fat creates too much space and can lead to an increase in blood pressure and an increased risk of heart attack. Visceral fat can be calculated by measuring the largest parts around your waist and hips.

### **7. Muscle Mass**

Muscle mass is a key measurement of the muscles in your body in pounds or kilograms. This measure plays an important role in your overall fitness, as muscles burn energy and fat all the time. As your muscles mass increases. Your body is able to burn calories or energy faster, which has the effect of increasing weight loss. Muscle mass includes the measurement of the smooth and skeletal muscles as well as water in the body.

### **8. Bone Mass**

Bone mass is a measurement of the overall bone mineral density of your body. This measure provides an important snapshot of your overall bone health. Low bone density can be a key indicator of osteoporosis.

### **9. Daily Caloric Intake**

Caloric intake is a combination of all of the food take in each day. Calories are used to measure the energy content of food and beverages. In order to lose weight, need to eat fewer calories than body needs for energy each day and vice versa. This scale measures and tracks these nine aspects of body composition with medical-grade accuracy. Instead of just getting a measure of weight and a limited view of health, have an accurate snapshot of overall health.

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## **4.4 Adipose Tissue And Regulation Of Body Weight**

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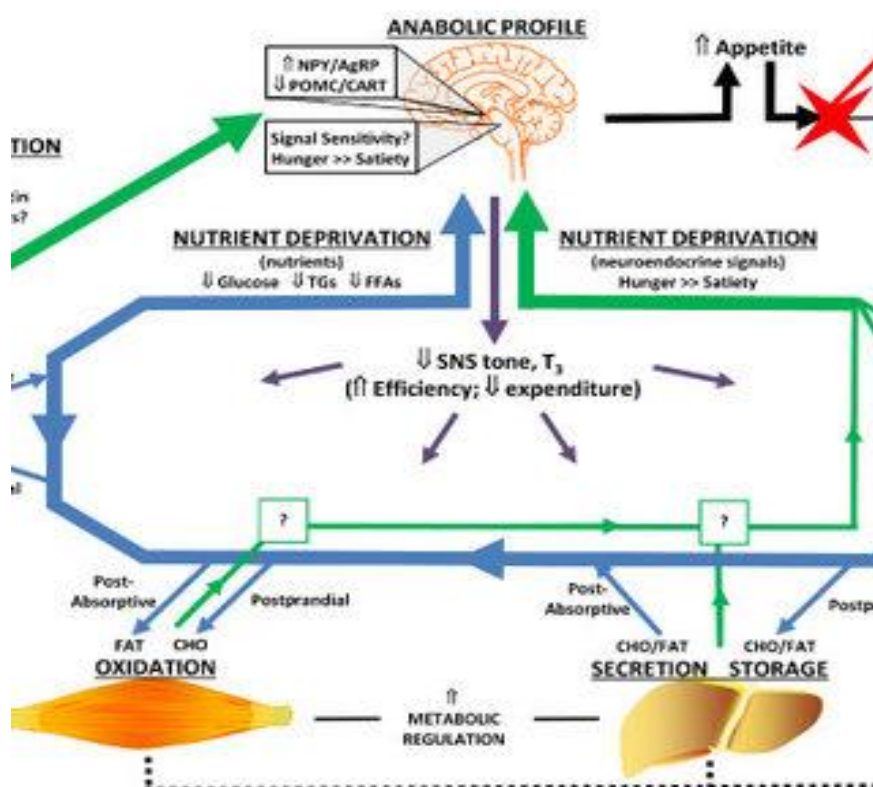
### **Introduction**

Over 60% of adults and close to 20% of children in the United States are overweight or obese. Weight loss strategies are only transiently effective for most people, as the vast majority of individuals who attempt to lose weight are not able to achieve and maintain a 10% reduction over a year. Over a third of lost weight tends to return within the first year and the majority is gained back within 3 to 5 years. A number of reasons have been proposed for the high recidivism rates, but there is substantial evidence for a biological drive to regain weight after weight loss. The objective of this review is to summarize the contribution of white adipose tissue to this

biological drive and discuss how changes in its cellularity and metabolic characteristics may facilitate weight regain.

### **The biological drive to regain weight**

The biological control of body weight involves a complex feedback loop between the brain and periphery. The brain receives signals from the periphery regarding long-term energy stores (i.e. adipose tissue triglyceride) and short-term nutrient availability (i.e. immediate availability of circulating nutrients) and based upon these integrated signals, adjusts energy balance to meet both the long-term and short-term objectives of energy homeostasis. This feedback system adapts when energy intake is cognitively (in humans) or forcefully (in animal models) restricted. In a previous review, we summarized the adaptations to energy-restricted weight loss that are thought to promote weight regain (Fig. 1). This adaptive response involves coordinated changes in the brain, gut, muscle, liver, adipose tissue and neuroendocrine system, which culminate in a concerted effect on energy balance. Peripheral signals create an 'anabolic' neural profile in the hypothalamus and hindbrain, increasing appetite and sending neuroendocrine efferent signals to enhance metabolic efficiency in peripheral tissues. Metabolic requirements decline as a function of (i) lost mass, (ii) reduced consumption of food and (iii) increased metabolic efficiency of peripheral tissues. Peripheral tissues clear circulating nutrients more effectively and utilize fuels more efficiently to produce the energy they need. Signals from the periphery convey to the brain that energy stores are depleted and nutrient availability is low and these signals integrate in key circuits of the hypothalamus and hindbrain that serve as the primary control centres for energy balance regulation. The response to these integrated signals is that appetite increases and the expenditure of energy declines. We have referred to this quantitative difference between the caloric value reflecting appetite and expenditure requirements as the energy gap. To maintain the reduced weight, food intake must be cognitively (in humans) or forcefully (in animals) restricted to the level that expended energy is suppressed. During weight maintenance after weight loss, this energy gap reflects the magnitude of the daily burden that thwarts cognitive efforts to maintain the reduced weight. When efforts to restrict intake fail, overfeeding occurs, and the excess nutrients are rapidly cleared and stored, and the relapse to obesity begins. This pressure to continue to overfeed generally persists until the lost weight returns. In some cases, the biological pressures may lead to weight gain that surpasses the original weight.



**Figure 1** Homeostatic adaptations to weight loss that persist in weight maintenance

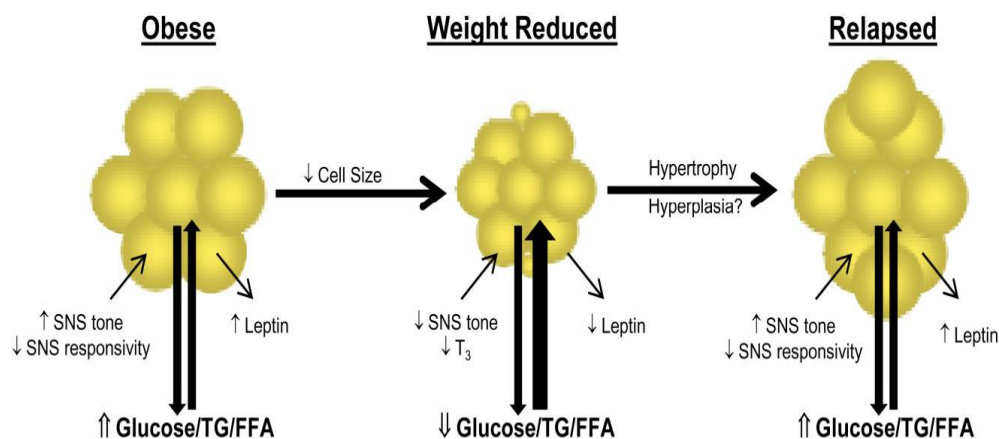
A fundamental understanding of this energy gap, dictated solely by biological pressures, has emerged from preclinical studies of weight regain in diet-induced obesity (DIO) models. The energy gap at the maintenance-relapse transition is influenced in predictable ways by diet composition, by the length of time in weight maintenance after weight loss and by physical activity levels. Weight regain driven solely by this biological pressure reflects a first-order growth curvesuch that the energy gap diminishes as the relapse to obesity progresses. As such, the magnitude of the energy gap is greatest at the nadir weight after weight loss. Furthermore, this energy gap does not dissipate with time in weight maintenance. Rather, studies indicate that the magnitude of the energy gap gradually increases the longer an animal maintains their reduced weight with an energy-restricted diet. The implications from these observations are that the biological pressures may strengthen with time during weight maintenance and with the amount of weight lost. White adipose tissue is a critical node in the homeostatic system that controls body weight and it plays a particularly important role in the biological drive to regain lost weight. Over the past several decades, adipose tissue has been recognized as a dynamic, multifunctional organ with a number of different types of cells. It houses the majority of stored energy as triglyceride, which is thought to be the primary targeted parameter for regulation in long-term energy homeostasis. The adipocyte serves its primary purpose of long-term storage of energy and as weight is gained, lost and regained, adipocytes and their support cells must undergo a substantial amount of remodelling to accommodate the gain or loss of stored energy. As an integrated node in the feedback

system, adipose tissues must send and receive important signals to and from the brain and other peripheral tissues to appropriately adjust the level of stored energy. Changes in adipocyte cellularity

**Adipocyte size:** highly modified Weight loss is accompanied by a dramatic reduction in the size of adipocytes (Fig. 2), which is reversed when weight is regained. An individual adipose depot contains adipocytes that vary with respect to their size, and a size frequency distribution provides a clear picture of this variability within a depot. Because adipose depots exhibit differing cellularity profiles, a frequency distribution is often more informative than an average diameter. Studies in both humans and rodents suggest that adipocyte size is the most changeable aspect of cellularity characteristics in studies of weight loss and regain. During weight loss, energy stores are mobilized from adipocytes and adipocytes become smaller. During weight gain and weight regain, energy is accumulated and adipocytes become larger. The broad range for adipocyte size provides enormous flexibility for the amount of energy that can be stored at any one time. However, as adipocytes change size with the mobilization or accumulation of energy, the extracellular matrix must be remodelled to accommodate the change or a considerable mechanical strain will be imposed upon the adipocytes. Mariman has hypothesized that weight loss cause's cellular stress in adipocytes, resulting in an altered metabolic profile that would relieve the stress via increased storage of lipid. From this perspective, one portion of the biological drive to regain weight ould be based in the mechanical and molecular changes that are working to relieve the cellular stress and mechanical strain of the adipocyte.

**Adipocyte number:** modified unidirectionally Weight loss does not lead to any discernible change in the number of adipocytes in adipose tissue (Fig. 2). The number of adipocytes in a normal, healthy individual remains relatively constant throughout adulthood, but there are conditions in which the number of adipocytes in particular adipose depots may increase. Our studies in a rodent paradigm of weight loss and regain suggest that the metabolic conditions during the relapse to obesity may provide the conditions that promote hyperplasia. Early in the relapse process, we observed the emergence of a population of very small (<20µm) adipocytes, which was accompanied by an increase in total number of adipocytes in the depot. This increase in cell number persisted throughout the relapse process as all of the adipocytes became larger. We have speculated that this increased cell number partially explains animals in this model surpassing their pre-weight loss weight following relapse. While substantiating the temporal changes in cell size frequency distribution and total cell number in humans presents a logistical challenge, a hypercellularity phenomenon with similar characteristics has been reported in post-obese humans. Even so, this relapse-induced hyperplasia of adipose tissue, if it does occur, is likely limited to individuals who have a genetic predisposition for obesity. We have yet to observe an increase in cell number in diet-resistant rats or in DIO mice, which tend to relapse to their previous weight. Regardless, increasing the number of adipocytes in a depot in effect increases the overall capacity of that depot for triglyceride storage, and what flexibility exists for changing cell number appears to be unidirectional. There is very little evidence that the number of adipocytes is ever reduced under normal metabolic

conditions associated with changes in weight. The implication is that increasing the number of adipocytes in a depot represents a permanent increase in the overall capacity of that depot to store triglyceride



**Figure 2 Adipocyte cellularity changes with weight loss and weight regain**

Adipocyte turnover: a tightly controlled balance Because the number of adipocytes was observed to be relatively stable in normal, healthy adults, it was long thought that the adipocytes produced by puberty represented the population of cells that persisted throughout life. Tracer studies have discounted this notion by revealing that new adipocytes are being produced and mature adipocytes are being cleared with some regularity. A wide demographic study of Swedish adults observed that the turnover rate for adipocytes is approximately 8–10% per year. The generation of new adipocytes involves two distinct steps: (i) the proliferation of preadipocytes and (ii) the differentiation of preadipocytes into functioning adipocytes, capable of storing and releasing energy. The clearance of mature adipocytes is less understood, but is known to involve the recruitment of macrophages. The crown-like structures that are observed in adipose tissues represent adipocytes targeted for clearance, surrounded by the recruited macrophages. While the regulatory mechanisms for the generation and clearance of adipocytes are very different, they must be tightly linked to some global regulatory system that keeps them balanced, otherwise adipocyte number would be much less stable. The development of obesity is accompanied by a higher absolute amount of turnover, which is reflected in their greater fat mass and higher number of total adipocytes in their depots. The generation of new cells and clearance of mature cells remains, in general, balanced at a higher level in the obese. When adjusted for the difference in fat mass, the actual rate of cell turnover per unit fat mass is similar. At present, we do not know how adipocyte turnover is affected with weight loss or during the process of weight regain. However, if hyperplasia does occur, there must be some transient imbalance between new cell generation and mature cell clearance to account for the difference in cell number. Our ongoing studies will likely clarify how and when this balance is altered to elicit the hyperplasia we observed in our rodent

paradigm of weight regain. Metabolic capacity of the adipocyte Changes in global gene expression Adipose tissues experience a global down-regulation of gene expression in obese subjects in response to energy restricted weight loss, which includes all of the key metabolic pathways. However, this effect is partly reversed at the transition to weight maintenance. With weight maintenance and during weight regain, an expression profile that would enhance energy conservation and the repletion of energy stores emerges. Markers of oxidative stress and inflammatory cytokines, which are also known to suppress appetite and increase expenditure, decline. The impaired induction of lipogenesis by insulin, glucose and feeding associated with obesity resolves after energy-restricted weight loss. Finally, the enhanced metabolic response to ingested energy enhances nutrient clearance during weight maintenance and during sustained periods of overfeeding. These adaptive responses in the adipocyte prime the tissue to replete energy stores when nutrients once again become readily available.

Metabolic changes linked to adipocyte size Insulin sensitivity is inversely related to size of the adipocyte (41). Compared with large adipocytes, small adipocytes exhibit higher rates of insulin-stimulated glucose uptake, higher levels of glucose oxidation and a lower sensitivity to antilipolytic action of insulin. In addition, smaller adipocytes exhibit a lower basal and catecholamine-induced lipolysis, have a lower rate of turnover of stored lipid and express genes favouring energy storage (28,45,46). The higher lipolytic capacity and triglyceride turnover in larger adipocytes is associated higher levels of Adipocyte triglyceride lipase (ATGL), Hormone sensitive lipase (HSL) and Lipoprotein lipase (LPL). De novo lipogenesis is also down-regulated as adipocytes increase in size.

#### **Functional changes of the adipocyte?**

Beyond the cellularity characteristics, there is growing evidence to suggest that adipocytes have the capacity to alter their metabolic profiles and engage in wholesale changes in function, given the right metabolic context (14). White adipocytes have been observed in vivo to undergo transdifferentiation into brown adipocytes, which serve to dissipate, rather than store energy (56–58). Likewise, white adipocytes in the mammary gland have even been reported to transdifferentiate into glandular milk-producing epithelial cells during lactation, an effect that reverses after involution (57). These observations provide a novel perspective of the versatility of adipocytes that was once unappreciated. At present, few studies have considered such dramatic functional transformations in the context of weight loss, weight maintenance and weight regain studies. Given the metabolic extremes that can occur with weight loss and weight regain, it would be prudent for future studies to consider the extent to which adipocytes might be altered with energy restriction and gross overfeeding. The versatility of metabolic profiles of adipocytes in changing environments may partly depend on the origins of the adipocytes. New adipocytes may primarily arise from resident preadipocytes and progenitors of the mesenchymal lineage, but recent findings demonstrate that bone marrow-derived progenitors (BMP) of the hematopoietic lineage can also migrate out of the skeleton and differentiate into adipocytes (59–62). Although this phenomenon needs to be demonstrated in humans, they may

have an important role during weight regain if hyperplasia occurs. For instance, the observations of preferential homing and differentiation in visceral depots and lower leptin expression than white adipocytes suggest that BMP adipocytes could be a detriment to energy balance and metabolic health (60). The behaviour of these adipocytes during and after weight loss has not been determined, but would be essential for hypothesizing their relative role in energy balance and weight regain. Neuroendocrine signals affecting adipose tissue Energy-restricted weight loss from obesity is accompanied by a reduced sympathetic (SNS) tone (and reduced thyroid hormone levels. In contrast to the effects on SNS, the effect on thyroid hormones is observed less consistently and/or is more transiently tied to the early stages of weight loss. Collectively, these neuroendocrine changes can act upon adipose tissues to affect the size and number of resident adipocytes (Fig. 2). The SNS has established effects on the metabolic state and cellularity of adipose tissues and a decline in SNS tone in this tissue could explain the shift in metabolic state favouring the uptake and deposition ingested energy, as well as the hyperplasia. Other studies indicate that both preadipocytes and adipocytes are responsive to Thyroid Stimulating Hormone (TSH) and thyroid hormones in a similar fashion. Both the SNS and thyroid hormones have inhibitory effects on preadipocyte proliferation and stimulatory effects on preadipocyte differentiation. As such, a decline in SNS tone and thyroid axis activity during weight maintenance may provide permissive conditions for preadipocyte proliferation, while the reversal of these neuroendocrine inputs during weight regain could underlie the hyperplasia. While these neuroendocrine inputs provide a plausible explanation for both metabolic and cellularity adaptations with weight loss and regain, their actual contribution to the adaptive response in adipose tissues requires further study.

Adipose signals for long-term energy stores Leptin and insulin are often referred to as 'adiposity signals' because their levels generally reflect fat mass. Fasting levels of both hormones decrease with the decline in adiposity that occurs with weight loss (Fig. 2). The decline in leptin is more intuitive because it is secreted directly from adipocytes. The impact on insulin is indirect, reflecting the improvement in insulin sensitivity that occurs with weight loss. Interestingly, a number of studies have observed that leptin and insulin are actually reduced to a greater extent than would be expected for the amount of fat mass. We speculate that this may occur because leptin, and perhaps insulin, levels reflect both the amount of stored lipid and the size of the constituent adipocytes. Smaller adipocytes secrete less leptin and result in lower circulating levels for a given fat mass. Smaller adipocytes are also more insulin sensitive, which presumably means they require lower circulating levels of insulin to impart the same metabolic control. The reduction in cell size and the loss of total fat mass, therefore, may contribute independently to the decline in leptin and insulin. If new, very small adipocytes are generated early in the relapse process, the impact of cell size could be compounded. Regardless, the integrated adiposity signal conveyed to the brain is that the total energy reserves are low and that the adipocytes are far below their maximal capacity to store



energy. The changes in these hormones directly contribute to enhanced hypothalamic expression of arcuate nucleus (ARC) neuropeptide Y (88–92) and agouti-related peptide (91,92), as well as decreased expression of proopiomelanocortin (Fig. 1). These changes are the hypothalamic hallmark of an ‘anabolic’ state, leading to a positive energy imbalance and weight gain. Although the concept that the adiposity signals reflect both total stores and the fraction of maximal capacity filled is consistent with observations in weight loss studies, it needs to be tested more rigorously. What complicates the role of leptin and insulin as ‘adipose signals’ is that their relationship to adiposity is maintained only during energy balance and the correlations only apply to fasted levels of the hormones. When an energy imbalance occurs, leptin and insulin reflect the metabolic state (anabolic or catabolic) of adipose tissue, as it deposits or mobilizes energy. Overfeeding increases circulating levels of leptin and insulin and, with persistent overfeeding during weight regain, both leptin and insulin resolve long before the weight is fully regained. For this reason, leptin and insulin, by themselves, do not appear to sustain the signal of energy depletion as weight is being regained. Nutrient availability as a reflection of the capacity to store excess energy To complement the signal of energy depletion from these hormones, we have proposed that signals reflecting nutrient availability play a more critical role during the dynamic phases of weight regain. Signals could be either the nutrients or their surrogate neuroendocrine signals. The improvement in systemic metabolic regulation is often accompanied by lower fasting levels of glucose, free fatty acids (FFAs) and triglycerides (TGs), and more consistently yields reduced postprandial excursions of glucose and TGs with potentiated postprandial reductions in FFAs. This wholesale, consistent change in circulating nutrients undoubtedly imparts some homeostatic influence on the signals of nutrient status (Fig. 1). Levels of glucose are detected by nutrient-sensing systems in both the periphery and brain, with consequences to energy balance and fuel utilization in the periphery. Triglycerides may even be sensed via their putative effects on leptin and insulin transport across the blood–brain barrier. FFAs are sensed, such as glucose, in the central and peripheral nutrient-sensing systems and can reduce subsequent food intake when infused into the gut, into the circulation or directly into the brain. The cellular and metabolic adaptations in adipose tissues certainly contribute to the attenuated postprandial excursions of circulating nutrients following weight loss. The consequence to systemic metabolism is that postprandial glucose excursions would be attenuated and the postprandial suppression of circulating FFAs would be potentiated.

### **The ‘nutrient clearance’ hypotheses for the dynamic phase of weight regain**

This hypothesis suggests that the energy gap between appetite and expended energy persists during weight regain as a function of the capacity of adipose tissue to clear and store excess energy (Fig. 2). Early in relapse, the adipose tissue’s capacity to clear excess energy is pitted against the rate at which nutrients are ingested and absorbed. As weight regain progresses, the adipocytes gradually increase in size and their capacity to clear excess energy diminishes. Excursions of glucose and TGs become larger and the suppression of FFA under dynamic (postprandial) states of metabolism

would gradually become attenuated. Once the adipocytes near a critical threshold of size and the maximal capacity for stored energy is approached, the rate of weight regain would diminish. As the pre-weight loss weight is once again achieved, or surpassed if adipocyte hyperplasia has occurred, the fasting and postprandial levels of circulating nutrients would once again reflect the high levels observed with the insulin resistant state. This simplistic hypothesis integrates the long-term adipose signals, reflecting the level of 'stored energy', with short-term signals of nutrient availability, which essentially reflect the 'capacity to store energy'. Both signals are fundamentally rooted in the cellular and metabolic profiles of adipose tissues. Conceptually, the long-term signals provided by leptin and insulin would establish the global 'anabolic' tone in the hypothalamus, hindbrain and peripheral tissues. In this anabolic context, circulating nutrients and their surrogate neuroendocrine signals would become more important under postprandial conditions and during extended bouts of overfeeding while the weight is being regained. The convergence of these long-term and short-term signals in the energy homeostatic circuits of the brain would then dictate the magnitude and persistence of the energy gap. The fundamental ideas behind this hypothesis are not entirely novel and they certainly present a simplified picture of the feedback system. Decades of research and numerous publications have provided a basic understanding of the key nodes of the homeostatic system controlling body weight and of adipocyte biology. Practically, the picture becomes much more complex as the integrated feedback signal from adipose tissues includes feedback from multiple adipose depots that have different metabolic and cellularity characteristics (15). Dieting and weight regain tend to alter visceral adipose depots more than subcutaneous depots (107–109), but less is understood about the interplay between depots, how they collectively establish a capacity-related 'threshold' for adipocyte size and about their relative contribution to the signals of energy depletion and nutrient availability during weight maintenance and weight regain. Furthermore, there is a large variability between individuals with respect to the metabolic and cellularity characteristics of their adipose depots (22). This variation may translate into different 'thresholds' for adipocyte size and, consequently, different maximal capacities for a given adipocyte number. Even so, the value of this hypothesis is that it provides a basic explanation for the persistence of the energy gap driving weight regain in both static (during weight maintenance) and dynamic (during weight regain) phases of the relapse to obesity. In addition, it frames the integration of long-term signals for stored energy and short-term signals of nutrient availability in a manner that links both to the cellular and metabolic characteristic of adipose tissues.

**Check Your Progress**

- 1. Explain about weight management.**
- 2. Describe about bodyweight components.**

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## **4.5 OBESITY**

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Obesity is a complex disease involving an excessive amount of body fat. Obesity isn't just a cosmetic concern. It is a medical problem that increases your risk of other diseases and health problems, such as heart disease, diabetes, high blood pressure and certain cancers. The global increase in the prevalence of obesity has led to an increased need for measurement tools for research, management, and treatment of the obese person. The physical size limitations imposed by obesity, variations in body composition from that of normal weight, and a complex psychopathology all pose tremendous challenges to the assessment of an obese person. The field of obesity research would benefit from having more uniform methods of assessment which would enable researchers for clinical and community-based studies, evaluation teams to assess intervention programs, and health professionals for counseling individuals. Standardized assessment methods support better comparison of health between different studies and across diverse populations. This is particularly important since the reported results are attributed value that drives policy, organization, and treatment.

### **ASSESSMENT**

#### **Introduction**

Most people recognize obesity when they see it (or think they do). Obesity is an increasing health problem with the CDC reporting that about a third of the adults in the United States are overweight by at least 20% above their ideal weight. Additionally, about 17% of children and adolescents are obese. Most people—including health practitioners—assess obesity by having the person stand on a scale. While this provides information about weight, it provides no information about composition or distribution of body fat and provides only the most general assessment of health risk. Charts are available that provide “normal” ranges based on height and weight, but these charts often don't take into account differences in ethnicity, age, body build, and muscle development. For health purposes, the percentage of body fat and its distribution are important factors, but determining these factors is not as easy as one might think. Different assessment methods yield different results, some more accurate than others. There are primarily four types of assessment to determine obesity: anthropometry (direct body measurements), density, conductivity, and radiography.

#### **INDIRECT METHODS**

Anthropometric measurements are the most basic method of assessing body composition. Anthropometric measurements describe body mass, size, shape, and level of fatness. Because body size changes with weight gain, anthropometry gives the researcher or clinician an adequate assessment of the overall adiposity of an individual. However, the associative power among anthropometric measures and indices is altered as weight is gained or lost.

#### **Body mass index (BMI)**

The body mass index (BMI) is a standard measurement used to determine whether a person's weight is within normal range. The BMI is

based on average findings and uses weight and height to determine approximately the degree of obesity. Charts are available to show BMI, but calculating is simple. Calculate BMI by dividing weight in pounds (lbs) by height in inches (in) squared and multiplying by a conversion factor of 703.

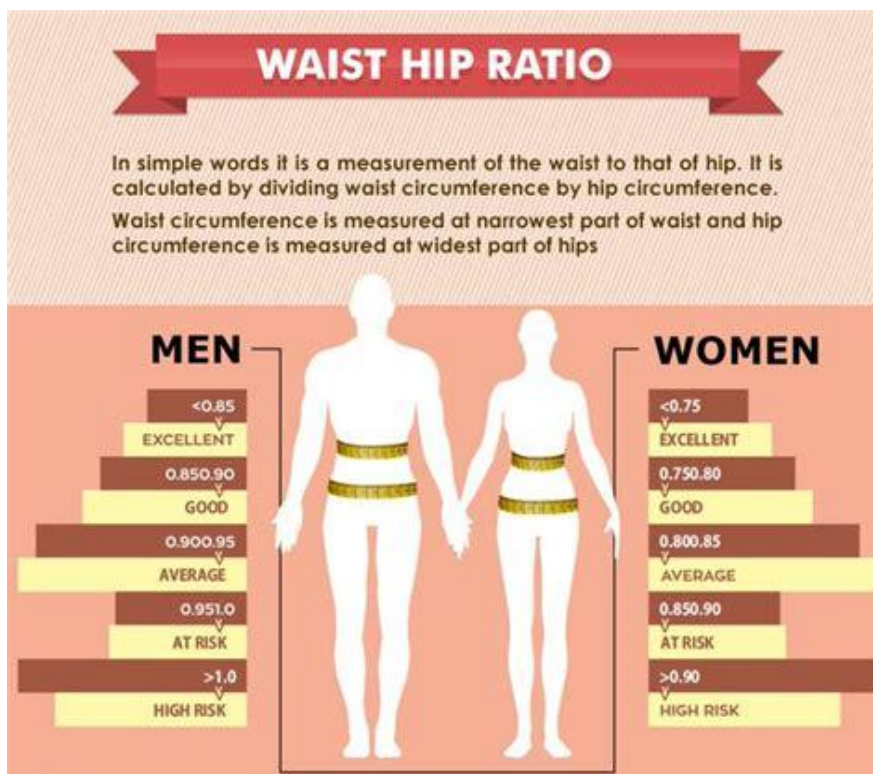
- Formula:  $\text{Weight (lb)} / [\text{height (in)}]^2 \times 703 = \text{BMI}$
- Example: Weight = 185 lbs, Height = 5'4" (64") Calculation: 185 divided by (64) <sup>2</sup> x 703 = 31.75.
- Metric formula: Weight is in kilograms and height is in meters. There is no conversion factor.
  - $\text{Weight} / (\text{Height})^2 = \text{BMI}$

Standards have been established regarding weight status, so one can compare the BMI finding (in this case 31.75) to the weight status chart and see that, according to the chart, this person is obese.

BMI	Weight Status
<b>Below 18.5</b>	Underweight
<b>18.5 – 24.9</b>	Normal
<b>25.0 – 29.9</b>	Overweight
<b>30.0 – 34.9</b>	Obese
<b>35.0 – 39.9</b>	Severe obesity
<b>≥40</b>	Morbid obesity

The BMI is a simple and fairly reliable measure to determine rates of obesity for large populations, but it is less accurate for the individual because it does not measure fat directly and is solely based on height and weight. Thus, a person who has not exercised or eaten an adequate diet--common with a person who has chronic health problems or is older--may lose muscle mass and replace it with fat but still retain a normal BMI while in actuality the person is overweight. Additionally, a body builder or physically active person who bulks up, causing weight gain, but has a low percentage of body fat may have a BMI in the overweight category. This is problematical because much of the research correlating weight with disease is based on BMI measurements. For example, a recent published report found that increased BMI correlated with increased rates of congestive heart disease, but increased survival rates also correlated with increased BMI, a seeming contradiction. When researchers restudied some of the participants and measured their degree of obesity using other methods, they found that many of the people classified as having normal BMI were, in fact, overweight—altering the findings.

Waist circumference



Another simple method of determining obesity is to measure waist circumference. Waist circumference has been found to be a good (although not completely accurate) predictor of obesity-related health risks. People with large waists are at increased risk even if BMI is normal. Typically, men put on fat in the abdomen, developing what is often described as a “beer belly.”



Women, prior to menopause, tend to put on fat in the buttocks and hips; but after menopause (as estrogen levels fall), many women begin to deposit fat in the same manner as men, in the abdomen. Increased abdominal fat is associated with diabetes type 2, hyperlipidemia, elevated triglycerides, hypertension, coronary artery disease, hormonal cancers (such as breast cancer), and sleep apnea. The waist is measured right above the hipbones, holding the tape snug but not compressing the skin.

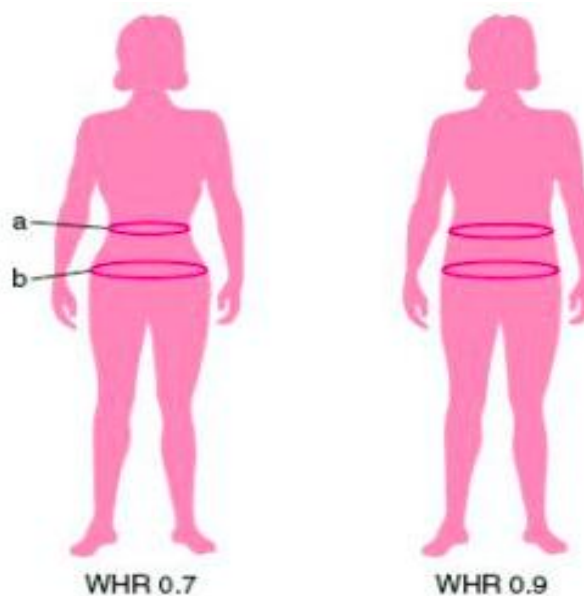
Gender	Low risk	High risk
Male	≤40 inches	>40 inches
Female	≤35 inches	>35 inches

BMI	Status	WC ≤35 (female) or ≤40 (male)	WC >35 (female) or >40 (male)
<18.5	Underweight		
18.5 – 24.9	Normal		
25.0 – 29.9	Overweight	Increased risk	High risk
30.0 – 34.9	Obese	High risk	Very high risk
35.0 – 39.9	Severe obesity	Very high risk	Very high risk
≥40	Morbid obesity	Extremely high risk	Extremely high risk

Obviously, waist circumference is based on averages, so if people are outside the average in height (too short, too tall) or body build (too stocky, too slim), waist circumference alone may not be an accurate measure. Additionally, waist circumference (WC) may be combined with BMI when assessing obesity. As the BMI increases, risk increases even with a waist circumference in normal range; but the risk is greater with increased waist circumference.

#### **Waist-to-hip ratio (WHR)**

Body shape is often described as “pear” with excess fat deposited in the buttocks and hips or “apple” with excess fat deposited in the abdomen. Apple-shaped individuals with increased intra-abdominal fat are at higher risk of health problems. In addition to simply measuring weight circumference, the waist-to-hip ratio (WHR) considers the ratio of the waist circumference to the hip circumference to determine if the person is an “apple” or “pear.” The waist is measured at its smallest point (above the umbilicus) and the hips at the widest point:



- **Formula: Waist (inches) divided by hips (inches) = ratio.**
- **Example: 35 (waist) / 41 (hip) = 0.85**

Gender	Ideal	Increased risk	High risk
Male	0.9 to 0.95	0.96 to 1.0	>1.0
Female	0.7 to 0.8	0.81 to 0.85	>0.85

Researchers have found that one cause of abdominal fat deposits is increased cortisol, a response to stress. The amount of abdominal fat correlates with the degree of stress, so it is hard to determine which is the causative factor in health problems—the fat or the stress or (more likely) some combination of both. However, regardless, the WHR is an important determinant of risk. Canadian researchers in the INTERHART study (2005) evaluated the relationship between BMI, waist and hip circumference, and WHR in relation to risk of myocardial infarction. Evaluating 27,000 cases in 52 counties, this study showed that after adjusting for other risk factors, the WHR showed a significant association with MI worldwide and was a much better predictor than the BMI. The researchers recommended that BMI no longer be used as an evaluation of obesity but should be replaced with WHR.

### Skinfold thickness

Measurement of skinfold thickness is a direct measure of fat, unlike the previous methods. The premise is that subcutaneous fat is a reflection of the proportion of total body fat. Calipers are used to grasp and measure fatty tissue in various parts of the body (protocols vary), such as the chin, biceps, triceps, subscapular, chest, abdominal hip, thigh, knee and calf, and then an equation used to calculate the percentage of body fat. The selected sites (usually 3 to 7) are believed to represent the average thickness of subcutaneous fat.

Specific procedures must be followed for each measurement; for example, the abdominal measure is typically taken at the horizontal fold, 5 cm lateral to and at the same level as the umbilicus. The procedure for skinfold thickness measurement includes grasping the skin and underlying

tissue, shaking it to separate fat from muscle, and then pinching it between the arms of the caliper. Generally, two measurements are taken at each site and the two averaged to arrive at a measurement.



If done correctly, measurement of skinfold thickness can be up to 98% accurate in determining body fat. However, one problem with skinfold thickness measurement is that different clinicians measuring the same person may get different results, depending upon their knowledge and experience. Another problem is that various equations are used to assess results, so there is little consistency. Skinfold measurement may also be less accurate with those who are morbidly obese because the calipers don't open widely enough for accurate measurement. Skinfold thickness may be most predictive if combined with other measurements, such as circumference (of various parts of the body) and bone breadth.

## **DIRECT METHODS**

### **Total Body Water**

Total body water is easy to measure because it does not require undressing or any real physical participation. Water is the most abundant molecule in the body, and TBW volume is measured by isotope dilution. Water maintains a relatively stable relationship to FFM; therefore, measured water/isotope-dilution volumes allow prediction of FFM and fat (i.e., body weight minus FFM) in normal weight individuals. As with the other methods mentioned earlier, the TBW method is limited in the obese. The major assumption is that FFM is estimated from TBW based on an assumed average proportion of TBW in FFM of 73%, but this proportion ranges from 67 to 80%. In addition, about 15 to 30% of TBW is present in adipose tissue as extracellular fluid, and this proportion increases with the degree of adiposity.<sup>46</sup> These proportions tend to be higher in women than in men, higher in the obese, and therefore produce underestimates of FFM and overestimates of fatness.<sup>41</sup> Importantly, variation in the distribution of TBW as a result of disease associated with obesity, such as diabetes and renal failure, affects estimates of FFM and TBF further.

Total body water is a potentially useful method applicable to the obese but there are details that need to be considered. The several analytical chemical



methods used to quantify the concentration of TBW (and extracellular fluid) have errors of almost a liter. Equilibration times for isotope dilution in relation to levels of body fatness are unknown because, theoretically, it might (and should) take longer for the dilution dose to equilibrate in an obese person as compared with a normal weight individual. Also, a measure of extracellular space is necessary to correct the amount of FFM in an obese person. Such data could also be very useful in the treatment of end-stage renal disease.

### **Total Body Counting and Neutron Activation**

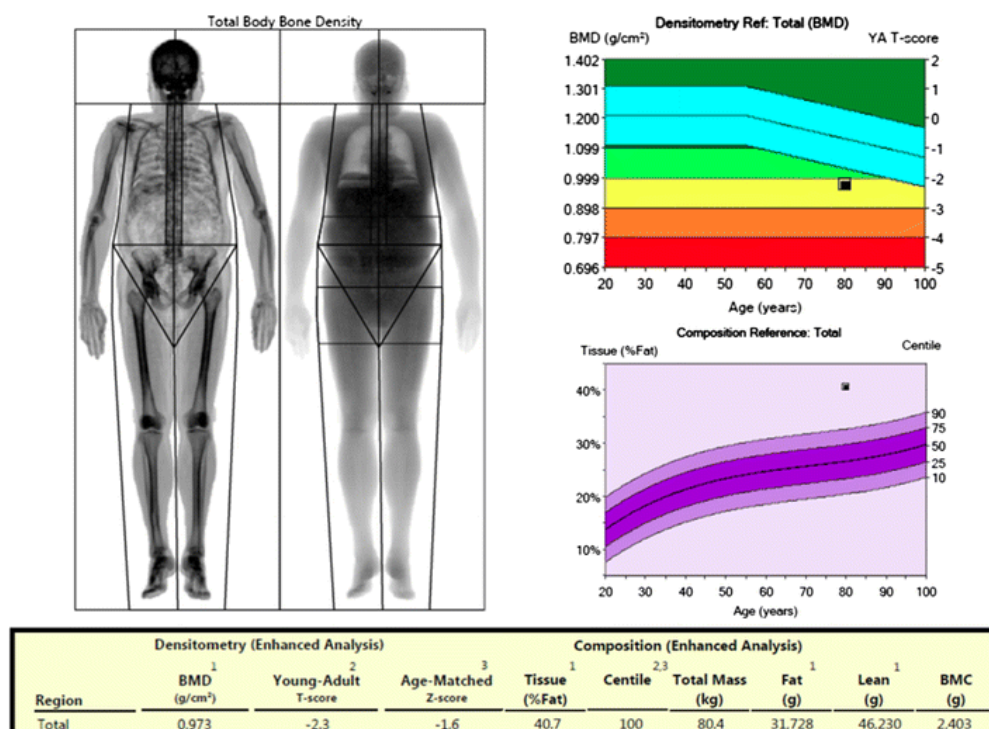
In addition to total body water, two other direct methods of body composition assessment are available to the researcher/clinician: total body counting and neutron activation. Total body counting (also called whole body counting) measures the amount of naturally radioactive potassium 40 (40K) in the body. Because potassium is found almost entirely within cell bodies, measuring potassium can provide an estimate of body cell mass. Fat-free mass can then be estimated once total body potassium is known, assuming a constant concentration of potassium in FFM. There are only a few of the detectors required for this technique currently in use in the United States, which precludes its use in most research. For further details regarding total body counting, readers are encouraged to consult Ellis.

Neutron activation techniques have been reported to be highly accurate for tissue-specific body composition, with a typical body scan occupying up to 1 hour. After subject exposure to a neutron field, gamma output can be measured as the cell nucleus relaxes and goes back to its pre-exposed state. Gamma output can be measured immediately upon activation (“prompt gamma neutron activation”) or at a somewhat delayed period (“delayed gamma neutron activation”). Using this technique, many elements in the body can be measured, including carbon, nitrogen, sodium, and calcium. Body nitrogen quantified by this method has been used to predict the amount of protein in the body to further analyze components of FFM. A significant concern with this technique is that it involves high levels of neutron radiation exposure and therefore has not been used in large-scale population research.

### **CRITERION METHODS**

#### **Dual energy X-ray absorptiometry (DXA)**

DXA, more commonly used to determine bone mineral density for diagnosis of osteoporosis, can also be used to determine fat content of the body and provides one of the most accurate measurements. Two different types of beams scan the body. One is absorbed more readily by fat than the other, so the computer is able to differentiate the fat from other tissues and provide the percentage of body fat.



**RMR (Resting Metabolic Rate)**  
1,494 cal/day  
Harris-Benedict

**RSMI (Relative Skeletal Muscle Index)**  
7.06 kg/m<sup>2</sup>  
Baumgartner

This method has the advantage of measuring fat over the entire body rather than in just certain areas. However, while DXA is generally considered the gold standard, the results may not always be accurate because fat mass is calculated indirectly by subtracting it from lean soft tissue or body cell mass. The percentage of body fat is calculated and risk assessment based on percentage ranges established for males and females.

Gender	Normal	Overweight	Obese
Adult Male	4-25%	25-30%	>30%
Adult Female	12-29%	29-35%	>35%

A problem with DXA is that it's not always readily available, especially in more rural areas, and most insurance companies do not reimburse the cost of the test, so the person has to pay out of pocket, and DXA can be expensive.

### **Near-infrared interactance**

Near-infrared interactance (NIR) uses a fiberoptic probe and a digital analyzer to determine the body's fat composition. The probe is placed against body sites, most often the biceps. The person's height, weight, size of frame, and level of activity are entered into the analyzer. Light penetrates the tissue and reflects off of the bone back to the equipment, which records optical densities. Based on the information received from the probe and the information entered, the machine calculates the percentage of body fat. NIR is increasingly popular because it can be done quickly and inexpensively in a doctor's office or sports facility, but it's not a reliable assessment method, especially for those who are very thin or very obese. Determining frame-size and level of activity is subjective, and people are not always honest about level of activity. Single-site assessment tends to be less accurate than multiple-site. Additionally, other factors, such as skin color, pressure applied to probe, and hydration, may interfere with readings. Research has not been done to establish accuracy of NIR, so it should not be used to determine the need for medical intervention.



### **Hydrostatic (underwater) weighing**

Underwater (hydrostatic) weighing is based on the fact that lean body tissue is more dense than fat. While underwater weighing has been used to establish references for percentage of body fat and is generally considered the most accurate measure of body fat, the equipment is usually only available in research facilities. Testing requires a large tank of water (about 1000 gallons) with the water maintained at a constant temperature. Additionally, the procedure can be unpleasant or virtually impossible for some people. The person is submerged repeatedly (8 to 10 times) in a large tub of water. After submersion, the person holds her breath for about 10 to 15 seconds while the technician records the weight. Calculations are based

on the difference between weight in the air and weight under water. The body's density is calculated by dividing the body mass by the volume of water it displaces (subtracting air left in the lungs). From that result, another calculation determines body fat.



### **Air displacement plethysmography (BodPod)**

The air displacement plethysmography method of analyzing body composition utilizes a special enclosed chamber, such as the BodPod, in which the person sits. Because clothing and hair can interfere with the results, the person must wear form-fitting swimwear or compression shorts (and bra for females), such as those made with spandex. The person must remove all jewelry and glasses and cover the hair with a swim cap. Prior to entering the capsule, the person is weighed on a scale to determine the mass measurement (weight), and the volume of air inside the BodPod is measured. The person sits inside while sensors determine the volume of air the person displaces. For example, if the volume inside the empty chamber was 400 L, and it reduces to 340 L with the person inside, then the volume of the person's body is 60 L. Measurements are taken 2 or 3 times for about 50 seconds each time during which the person can breathe normally. Between measurements, the door can be opened briefly. The person's thoracic gas volume (total volume of air in the lungs) is then measured by the person's breathing into a mouthpiece and tubing (unless standard predictive settings or previous testing results were entered into the

computer). The body volume, weight, and thoracic gas volume are used to calculate the percentage of lean and fat mass. The test takes about 5-10 minutes.



**Typical readout:**

Parameter	Results
Percent fat	10.6%
Percent fat free mass (other tissues)	89.4%
Fat mass	18.560lb
Fat free mass	156.072ib
Body mass (total weight)	174.632lb
Body volume	73.712L
Body density	1.075kg/L
Thoracic gas volume	4239L

The BodPod is increasingly used in sports medicine. People who are morbidly obese may not fit into the chamber, and those with claustrophobia may not be able to tolerate being closed inside, even for short durations. Additionally, some studies comparing the BodPod to underwater testing show that the BodPod tends to overestimate body fat by 2 to 3% while other studies show that it underestimates body fat. One study showed the BodPod overestimated body fat in women by 7% and underestimated it by 16% in men. In general, the BodPod tends to be more

accurate with those of average weight or overweight and less accurate with those who are lean.

### **Bioelectrical impedance analysis (BIA)**

The more water tissue contains, the faster electrical current is conducted through the tissue. Thus, electrical current passes through lean body tissue and fluids (blood, urine, muscle) faster than air, bone or fat tissue. This is the basis for bioelectrical impedance analysis (BIA). The BIA equipment measures resistance and estimates body fat by combining measures with height, weight, gender, fitness level, and age. BIA converts the results to percentage of body fat mass and estimate of total body water. Different procedures are available. Single frequency analysis takes only a few seconds but is less accurate than multiple frequency analysis, which takes a few minutes. During BIA, the person should stand with legs apart and arms held away from the body, making sure to avoid contact with any conducting surfaces. Results can be skewed by changes in hydration and intestinal contents and time of day. Additionally, BIA often overestimates body fat in lean individuals and underestimates fat in obese individuals. For increased accuracy, people should follow guidelines, which usually include avoiding food and drink for 4 hours prior to testing and exercise for 12 hours.



However, the digital scales only measure fat in the lower extremities (the current travels up one leg and down the other), and the handheld devices only measure fat in the upper body (current travels from one arm through the other), so neither of these devices account for abdominal fat. BIA equipment is widely available and often quite inexpensive, so it is frequently used in gyms and homes for personal use. However, the equipment varies widely in accuracy, and there is a considerable margin of error with BIA.

## **Types of Obesity**

There are multiple classifications of Obesity.

### **I. Depending on the area of fat deposition, there are three forms of obesity**

1. Peripheral: Accumulation of excess fat in the hips, buttocks and thighs.
2. Central: Accumulation of excess fat in the abdominal area.
3. Combination of both peripheral and central obesity.

Abdominal area is considered the most dangerous area for the accumulation of fat because it is closely located to the vital organs and their blood supply.

### **II. Depending on the association with other diseases there are two types**

1. Type-1 obesity: It is not caused by any disease. It is due to excessive intake of calories and lack of physical activity.
2. Type-2 obesity: It is caused by a disease (like Cushing syndrome, hypothyroidism, polycystic ovarian disease, and insulinoma, are some internal secretion diseases). It accounts for less than 1% of obesity cases and is observed that there is abnormal weight gain with type-2 obesity even on little intake of food.

### **III. Depending on the size and number of fat cells, obesity can be divided into**

1. Adult-type: In this type of obesity, only the size of fat cells is increased, it may happen mostly in middle age
2. Child-type: In this type of obesity, the number of fat cells is increased. It is extremely difficult to reduce the number of fat cells which are already made.

### **Causes of obesity**

Weight gain occurs when you eat more calories than your body uses up. If the food you eat provides more calories than your body needs, the excess is converted to fat. Initially, fat cells increase in size. When they can no longer expand, they increase in number. If you lose weight, the size of the fat cells decreases, but the number of cells does not.

- Obesity, however, has many causes. The reasons for the imbalance between calorie intake and consumption vary by individual. Your age, gender, genes, psychological makeup, socioeconomic, and environmental factors all may contribute.
  - ✓ Genes: Your genes may play a role in efficiency of metabolism and storage and distribution of body fat.
  - ✓ Family lifestyle: Obesity tends to run in families. This is caused both by genes and by shared diet and lifestyle habits. If one of your parents is obese, you have a higher risk of being obese.
  - ✓ Emotions: Some people overeat because of depression, hopelessness, anger, boredom, and many other reasons that have nothing to do with hunger. This doesn't mean that overweight and obese people have more emotional problems than other people. It just means that their feelings influence their eating habits, causing them to overeat.

- ✓ Environmental factors: The most important environmental factor is lifestyle. Your eating habits and activity level are partly learned from the people around you. Overeating and sedentary habits (inactivity) are the most important risk factors for obesity.
- ✓ Socioeconomic factors: social factor is the common cause of obesity
- ✓ Sex: Men have more muscle than women, on average. Because muscle burns more calories than other types of tissue, men use more calories than women, even at rest. Thus, women are more likely than men to gain weight with the same calorie intake.
- ✓ Age: People tend to lose muscle and gain fat as they age. Their metabolism also slows somewhat. Both of these lower their calorie requirements.
- ✓ Pregnancy: Women tend to weigh an average of 4-6 pounds more after a pregnancy than they did before the pregnancy. This can compound with each pregnancy.
- Certain medical conditions and medications can cause or promote obesity, although these are much less common causes of obesity than overeating and inactivity. Some examples of these are as follows:
  - ✓ Cushing syndrome
  - ✓ Depression
  - ✓ Certain medications (examples are steroids, antidepressants, birth control pills)
  - ✓ Prader-Willi syndrome
  - ✓ Polycystic ovarian syndrome
- Obesity can be associated with other eating disorders, such as binge eating or bulimia.
- The distribution of your body fat also plays a role in determining your risk of obesity-related health problems. There are at least two different kinds of body fat. Studies conducted in Scandinavia have shown that excess body fat distributed around the waist (apple-shaped figure, intra-abdominal fat) carries more risk than fat distributed on the hips and thighs (pear-shaped figure, fat under the skin).

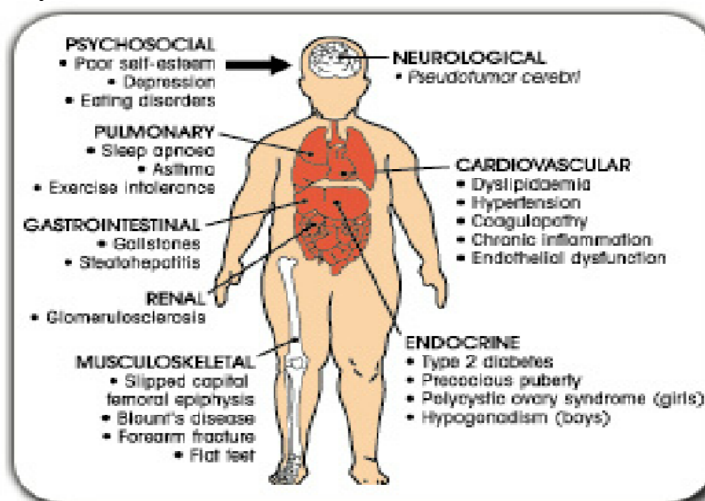
### **Complication of obesity**

Morbidities related to obesity Impaired glucose tolerance and Diabetes mellitus. There is currently no controversy that obesity is associated with impaired glucose tolerance or type 2 diabetes mellitus. The underlying mechanism is thought to be due to insulin resistance. However, there is currently limited data accurately quantifying insulin resistance using the standard hyperinsulinemic euglycemic clamp, largely because the invasive nature of the procedure makes it unsuitable for general epidemiological studies. The association of obesity with diabetes has been shown in several studies. In one of the biggest cohort studies, in which 84,941 female nurses were followed up for 16 years, there were 3,300 new cases of diabetes mellitus. Importantly, the study revealed that overweight or obesity was the main predictor of type 2 diabetes mellitus.<sup>13</sup> In men, there were similar findings from the Health Professional follow-up study.



An age adjusted relative risk of 60.9 for developing diabetes was found in those with a BMI  $\geq 35$  Kg/m<sup>2</sup> in comparison to those with BMI  $\geq 35$  Kg/m<sup>2</sup> in comparison to those with BMI  $< 23$  Kg/m<sup>2</sup>.

In Malawi, the prevalence of diabetes in adults aged 25- 64 years is estimated at 5.6%. However, there is limited data on obesity attributable diabetes in Malawian adults. Hypertension Data available shows a strong association between obesity and hypertension. In one large cohort study of 82,473 participants, BMI was positively associated with hypertension at age 18 and midlife. There was also marked increase in risk of hypertension with weight gain. In the Framingham study, the relative risk of hypertension in overweight men and women were 1.46 and 1.75, respectively, after adjusting for age.<sup>16</sup> In the same study, reduction of weight in obese women at age 18 reduced the risk of hypertension. In older populations, hypertension and obesity continue to relate in a predictable manner as has been shown in the Honolulu Heart Program and Japanese data survey. Recently, waist circumference (WC) has been shown to be important in assessing obesity and the risk of hypertension. When WC and BMI were compared as continuous variables in the same regression model, WC was found to be a better predictor for obesity related risk, including hypertension, than BMI.<sup>19</sup> However, when WC was used as a categorical variable (normal or high), BMI was a better predictor. WC may be a valuable means of quantifying the risk of hypertension in the obviously obese individuals as it is cheap, easier and faster to apply than BMI which, in addition to a stadiometer, requires a weighing scale and calculation of the index. Following a recent survey in Malawi, hypertension was estimated at 32.9% in adults aged 25-64 years. The survey also found that 29% of the population in this age range were either overweight or obese possibly indicating that obesity may have played a role in hypertension. However, the association of obesity and hypertension was not interrogated in this survey.



### Heart Disease

There is unequivocal evidence that there is an increased risk of coronary artery disease (CAD) in obesity. In the Asian Pacific Cohort Collaboration study in which more than 300, 000 participants were followed, there was a 9 percent increase in events of ischaemic heart disease for a unit change in BMI. Increased risk of CAD has also been

found in the Framingham and Nurses Health Studies.<sup>16,20</sup> When the risk of heart failure (HF) was evaluated in the Framingham study, the risk of HF was found to be 2-fold in the obese group than in the non-obese group.<sup>21</sup> However, it appears that having a higher BMI improves survival in patients with congestive heart failure (CHF). In a retrospective analysis of 7,767 patients with CHF who were categorised into 4 BMI ranges including obesity (BMI>30kg/m<sup>2</sup>), there was reduced crude all case mortality with consecutively higher BMI groups in an almost linear fashion. After further analysis, overweight and obese patients had a hazard ratio of 0.88 compared to healthy weight patients (taken as the reference group) whereas underweight patients with stable CHF had a 1.21 risk of death when they were compared to the same reference group.<sup>22</sup> The reason for this is not clear. The authors argue that other cardiovascular morbidities associated with obesity and overweight may have lead to the diagnosis of HF in its earlier stages in the obese group than in the group with lower BMI, therefore, reducing the risk of death from CHF.<sup>22</sup> However, the cardiopulmonary testing results of overweight and healthy weight patients, with CHF have been found to be similar.<sup>23, 24</sup> Therefore, the foregoing argument is unlikely to account for this difference.

Clearly, with the known adverse effects of obesity and in the absence of knowledge on the mechanism of this 'paradox', recommending overweight or obesity for purposes of reduction of CHF associated mortality is not an option. Elucidating the mechanism of this paradox is currently an area of research interest.

### **Dyslipidaemia**

Dyslipidaemia, manifested by reduced high density lipoprotein (HDL) and increased triglycerides, is associated with obesity.<sup>25</sup> The underlying mechanism is largely due to insulin resistance. Very low density lipoprotein (VLDL) clearance in plasma is dependent on the rate of hepatic synthesis and catabolism by lipoprotein lipase, an enzyme which is also involved in formation of HDL.<sup>25,26</sup> In obesity, insulin resistance is associated with increased hepatic synthesis of VLDL and impaired lipoprotein lipase.<sup>26,27</sup> There is evidence that dyslipidaemia can still occur in the absence of insulin resistance in obesity. In 1998, a study by Gary et al. showed a significant association between obesity, particularly central obesity, and dyslipidaemia after adjusting for insulin resistance.

### **Cerebrovascular Disease**

Currently available evidence shows that the risk of haemorrhagic and ischaemic stroke, in relation to obesity, is increased in men. In women this relation is true with ischaemic stroke but not haemorrhage stroke. In the Korean prospective study involving 234,863 men who were followed up for 9 years, a significant positive association was found between BMI and the risk of ischemic stroke whereas, with haemorrhagic stroke, a J-shaped association was found showing that the risk increased more than that of ischaemic stroke at the upper and lower extremes of BMI.<sup>28</sup> Controlling for confounding factors attenuated the association but still yielded significant association. In a prospective study of 39,053 participants (all women) followed up for an average of ten years, 432 strokes occurred. Three hundred and seven were ischaemic, 81 hemorrhagic and 4 undefined.

In obese subjects (BMI > 30kg/m<sup>2</sup>), the hazard ratios (95% CI) for total stroke, ischaemic stroke and hemorrhagic stroke were 1.5 (1.16 to 1.94), 1.72 (1.30 to 2.28) and 0.82 (0.43 to 1.58), respectively. This was in comparison with the group of women with BMI less than 25kg/m<sup>2</sup>.<sup>29</sup> The reason for the discrepancy in risks of hemorrhagic stroke between men and women is not clear. Following findings from other studies that have shown higher incidence of hemorrhagic stroke in Asian populations in the setting of low cholesterol and lean body weight, the authors argue that these factors (low cholesterol and lean body weight) may explain the observed findings in the latter studies. However, this still remains a hypothesis which needs investigating. Further, with this hypothesis one would expect men in the Korean and Physicians' studies to have shown increased risk of hemorrhagic stroke as well with the group with BMI towards lean body weight who presumably would have had low cholesterol levels. Lately, central obesity (where fat is preferentially distributed around the trunk) has been shown to be important in predicting stroke mortality. In the Israel heart disease study, stroke mortality was predicted by trunk obesity alone independent of BMI, hypertension, diabetes and socioeconomic status.

### **Metabolic syndrome**

According to the National Cholesterol Education Program's Adult Treatment Panel III (NCEP: ATP III), the metabolic syndrome is defined when an individual has any 3 of the following 5 features: (i) waist circumference above 40 inches for men and >35 inches for women, (ii) Triglycerides above 150mg/dl, (iii) HDL cholesterol above 40mg/dl for men and 50mg/dl for women, (iv) Blood pressure above 130/85 mmHg, (v) Fasting glucose above 100mg/dl. Central obesity and insulin resistance, which leads to altered lipid and glucose metabolism, appear to be the basis for the features seen in metabolic syndrome. The syndrome was originally intended for prediction of the risk of cardiovascular disease, however, this has recently been questioned as the sum of the combined risk factors appears not to offer more than the sum of individual factors.

### **Pulmonary abnormalities**

Several studies have linked obesity and obstructive sleep apnea (OSA). In the Wisconsin Sleep Cohort study, obesity had a strong association with OSA. In another study, increased neck circumference, which was also shown to correlate very well with obesity, had been shown to correlate with obstructive sleep apnea. There have been two mechanisms that have been thought to contribute to OSA. Firstly, is the direct effect of increased fat tissue along the airway which impinges on the lumen.<sup>38</sup> Secondly, increased fat tissue has been implicated in increasing the collapsibility of the airway. Asthma is another condition that may occur as a complication of obesity. There is evidence that obesity increases the risk of asthma. In one prospective multicentre study, the prevalence of asthma was observed to increase in obese patients. Seventy five per cent that presented with an asthmatic emergency were either obese or overweight. Further prospective studies have shown that obesity predicts asthma. The mechanism linking obesity and asthma includes increased airway hyper-responsiveness, decreased functional and tidal volumes, chronic systemic inflammation driven by increased inflammatory cytokines and chemokines,

adipocytes derived factors leptin, adiponectin and plasminogen activator inhibitor.

### **Gastrointestinal abnormalities**

Most epidemiological studies have found an association between obesity and increased risk of Gastroesophageal reflux disease (GORD). In one large cross-sectional population study, which was part of a randomized trial, involving 10, 537 subjects, the adjusted odds ratios for heart burn and acid regurgitation occurring once in a week in obese patients were 2.91 (95% CI 2.07 – 4.08) and 2.23 (95% 1.44-1.99) respectively, compared with those with normal BMI.<sup>46</sup> Recent evidence from a meta-analysis involving data from studies between 1966 and 2004 has shown obesity to be significantly associated with GORD, esophageal cancer and erosive esophagitis and that these disorders appear to increase with increasing weight.<sup>43</sup> Another gastrointestinal condition that has been studied in relation to obesity is cholelithiasis. Data from the Nurses' study showed that females with BMI of more than 45Kg/ m<sup>2</sup> had a seven-fold increase in risk of gallstone disease compared to those with BMI of less than 24Kg/m<sup>2</sup>.<sup>44</sup> Men have had similar results.

### **Reproductive disease**

Polycystic ovary syndrome (PCOS), characterized by anovulation, hyperandrogenism and a polycystic ovary, is associated with obesity as well as insulin resistance. It has been noted that increased visceral fat assessed by waist circumference of more than 88cm is associated with hyperandrogenemia in patients with PCOS and that reduction of insulin resistance by weight loss or drugs that increase peripheral sensitivity of insulin leads to improve hormonal aberrations and ovulation.<sup>46</sup> In men, abdominal obesity has been associated with impotence and infertility. In one single blinded randomised controlled trial of 110 obese men with erectile problems but no other risk factors namely diabetes, hyperlipidemia or hypertension, there was improvement of sexual function associated with decreased BMI. There are other reproductive complications of obesity that occur in pregnancy and labour. These include gestational diabetes, macrosomia, dystocia and increased rates of caesarean sections.<sup>48</sup> Psychosocial problems Obesity in the affluent society has been associated with several untoward outcomes in terms of psychosocial or socioeconomic wellbeing. Obese females for example were found to be less likely to complete school, had a 20% less chance of getting married, earned less and had more household poverty in comparison to females that were not overweight.<sup>49</sup> However, the direction of causality can be either way since status causes obesity and obesity causes status.<sup>50</sup> Several psychiatric disorders have been linked to obesity. In one study involving psychiatric evaluation of 294 patients before bariatric surgery, the prevalence rates were as follows: somatization (29.3%), phobia (18%), hypochondriasis (18%) and obsessive-compulsive disorders 13.6%. Follow up of these patients after surgery showed that these psychopathologies had been reduced significantly.

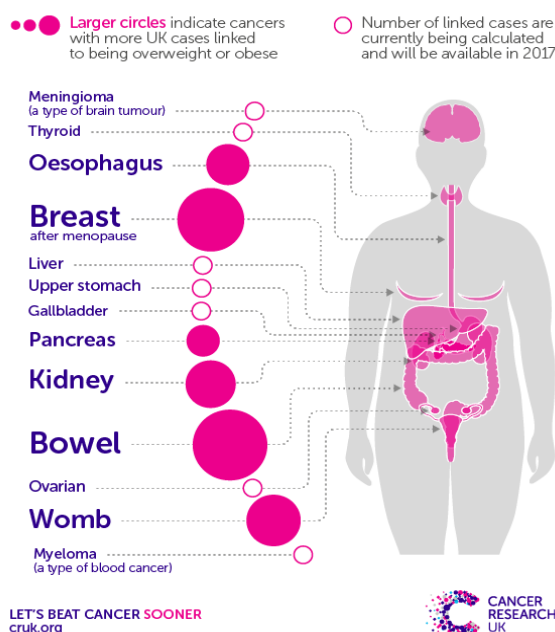
### **Osteoarthritis**

Osteoarthritis (OA) appears to follow obesity. In the Framingham cohort study, data from 1420 participants indicated that obesity was an important independent risk factor for OA after adjusting for age, physical activity and the levels of uric acid. Other studies looking at the effect of weight reduction on obesity have shown a significant reduction in the odds of developing OA overtime, further providing evidence for this link. OA involving weight bearing joints is common later in life and the prevalence is above 50% in both men and women by age 65 years.<sup>54</sup> The mechanism of OA has been presumed to be due to direct chronic strain on the joints related to the overweight. However, there are now notions that nonmechanical mechanisms may contribute to OA in obesity as the same changes of OA seen in weight bearing joints have also been seen in non-weight bearing joints. There is growing evidence that dysregulation of adipokines (hormones from adipose tissue) such as adiponectin, visfatin and resistin may explain the link between obesity and OA – suggesting that osteoarthritis may be a systemic disease in obesity.

### Cancer

There is considerable evidence of an association between obesity and some cancers. These include cancer of gallbladder, esophagus (adenocarcinoma), thyroid, kidney, uterus, colon and breast. This link has further been strengthened by the observation that there is reduced incidence of cancer and mortality with weight loss. However the underlying mechanism linking these cancers to obesity is not clear. For uterus and breast cancers, it is thought to be due to higher oestrogen levels synthesized from fat tissue in obese women.

### BEING OVERWEIGHT CAN CAUSE 13 TYPES OF CANCER



### Dietary management

Dietary intervention is the cornerstone of weight loss therapy. Most of the dietary regimens proposed for weight loss focus on energy content and macronutrient composition. It is the energy content that determines the efficiency of the dietary regimens. Obesity treatment guidelines issued by

the NIH recommend that persons who are overweight or who have class I obesity and who have two or more risk factors should reduce their energy intake by 500 kcal/day. Persons with class II and class III obesity should strive for 500–1000 kcal/day reduction. With a reduction of 500 kcal/day energy intake, a weight reduction of 0.5 kg/week can be achieved. To provide a diet those results in the desired energy deficit, it is necessary to determine the patient's daily energy requirement, which can be estimated by using the Harris–Benedict equation<sup>10</sup> or the WHO equation<sup>11</sup> or American Gastroenterological Association dietary guidelines.

### **Type of diets**

In general, there are four types of dietary regimens used in the treatment of the overweight or obese persons: (Table 1)

1. Low-calorie diet (LCD)
2. Low-fat diet
3. Low-carbohydrate diet
4. Very low-calorie diet (VLCD)

The first three diets are 800–1500 kcal/day while VLCD is < 800 kcal/day. LCD. LCDs are high in carbohydrate (55–60%), low in fat (less than 30% of energy intake), and high in fiber and have a lowglycemic index. Alcohol and energy-dense snacks should be avoided. LCD has been shown in 34 randomized trials to reduce body weight by 8% during 3–12-month period. Overweight or obese patients tend to underestimate their energy intake. To help them overcome this, portion-controlled or prepackaged meals that make up the required energy intake are available. Replacement meals are available as drinks, nutrition bars, or prepackaged meals. A 4-year study demonstrated weight loss improvement in blood sugar and blood pressure for persons taking meal replacement diets.

### **Low-fat diets**

These diets reduce the daily intake of fat to 20–25% of total energy intake. For a person on a 1500-calorie diet, this translates to 30–37 g of fat, which can be counted using food label from packages. Alternatively, a dietician can provide the person with a specific menu plan that has reduced fat. According to a meta-analysis of 16 trials, low-fat diet used over 2–12 months resulted in mean weight loss of 3.2 kg and improved cardiovascular risk factors (Table 1).

### **Low-carbohydrate diet**

The carbohydrate content of the diet is an important determinant of short-term (less than 2 weeks) weight loss. Low-carbohydrate (60–150 g of carbohydrate/day) and very low-carbohydrate diet (0 to < 60 g) have been popular for many years. Glycogen utilization occurs when carbohydrate intake is restricted. When the carbohydrate intake is less than 50 g/day, ketosis will develop from glycogenolysis, resulting in fluid loss. Many of the current low-carbohydrate diets (e.g. Atkins diet) limit carbohydrate intake to 20 g/day but allow unrestricted amounts of fat and protein. A meta-analysis of five trials found that weight loss at 6 months favoring low-carbohydrate over low-fat diet is not sustained at 12 months. Triglycerides and high-density lipoprotein (HDL) cholesterol changed more favorably in people assigned to low-fat diet. There are data from the National Health Study and Health Professional, Follow Up study that low-

carbohydrate diet with the highest decile for animal protein and fat were associated with higher all-cause and cardiovascular mortality.

### **VLCD**

VLCDs are diets with energy content of 200–800 kcal/ day. Diets below 200 kcal/day are starvation diets. VLCDs are not recommended for general use, as there are significant adverse events such as electrolyte unbalance, low blood pressure, and increased risk of gallstones. Its use needs to be supervised by trained medical personnel.

Each of the four types of diet for weight loss has its proponents. In a meta-analysis of 80 weight loss studies, mean weight loss of 5 to 8.5 kg (5–9%) was observed during the first 6 months from interventions involving a reduced-energy diet and/or weight loss medications with weight plateaus at approximately 6 months, with maintenance of 3 to 6 kg (3–6%) of weight loss at 48 months. A randomized controlled trial comparing four weight loss diets with different compositions of fat, carbohydrate and protein found no difference in outcomes, with a 2- to 4-kg weight loss with all diets after a year.<sup>19</sup> After 2 years, all calorie-restricted diets result in equal weight loss irrespective of the macronutrient composition. In contrast, all studies found that dietary adherence is an important determinant of weight loss. Thus, choosing a diet with a macronutrient composition based on a subject's taste preference can achieve better compliance.

### **Exercise and obesity**

Physical activity alone is not an effective method for achieving initial weight loss, although most overweight or obese people tend to choose exercise as the first interventional option. Without calorie restriction, weight loss through exercise alone is quite small, about 0.1 kg/week. A meta-analysis showed that exercise alone did not result in significant weight loss attempts, although no further weight gain was observed after 12 months. Although exercise is not effective for initial weight loss, physical activity is important for maintaining weight loss achieved through dietary intervention. Meta-analyses of 493 studies have shown that people who diet and exercise maintained their weight loss better than those who relied on diet alone. Before starting an exercise program, patients should be advised of joint and musculoskeletal injuries as well as cardiovascular risks. The risk of exercise stress testing before an exercise program is controversial. The American College of Cardiology and American Heart Association recommend treadmill for asymptomatic subjects with diabetes mellitus, men older than 45 years of age, and women older than 55 years of age before embarking on an exercise program. Other organizations recommend no stress testing for symptomatic subjects undergoing moderate-intensity exercise with guidance in exercise intensity. In our hospital, we use a physical exercise readiness questionnaire for screening purposes. The American College of Sports Medicine recommended in 2009 that moderate-intensity exercising between 150 and 250 min weekly is effective in preventing weight gain. To provide and maintain a clinically significant weight loss, at least 200–300 min/ week of moderate-intensity aerobic exercise is required. Resistance training does not enhance weight loss but may increase fat-free mass. Even in the absence of significant weight loss, regular aerobic and resistance exercise

improves cardiovascular fitness and obesity-related comorbidities such as NAFLD. A supervised exercise program involving personal trainers induces and maintains weight loss more effectively than unsupervised physical activity.<sup>22</sup> Exercise reduces food intake by increasing the satiating efficiency of a fixed meal.<sup>24</sup>

### **NAFLD**

NAFLD patients are usually overweight or obese and have underlying insulin and or leptin resistance leading to dysfunctional energy metabolism. Weight loss of 10% in overweight NAFLD patients improves liver biochemistry as well as hepatic steatosis and necroinflammation. Lifestyle modification consisting of exercise and diet can help the patients to achieve these goals. A 4–4.5% weight loss can result in 50% reduction in serum alanine aminotransferase, while with exercise alone and no weight loss, significant improvement in aminotransferase levels can occur, but its effect on liver histology is unknown.<sup>23</sup> The American Association for the Study of Liver Diseases, the American College of Gastroenterology, and the American Gastroenterology Association recommend weight loss as the preferred method in management of NAFLD.

### **Bariatric surgery**

Bariatric surgery is defined as gastrointestinal surgery to help severely obese patients lose weight. The US National Institutes of Health's 2013 guidelines recommended surgery for adults with BMI  $\geq 40$  kg/m<sup>2</sup> without comorbidities or 35 kg/m<sup>2</sup> with comorbidities who fail to lose weight by nonsurgical methods, and suggested that patients with BMI of 30–34.9 kg/m<sup>2</sup> with diabetes or metabolic syndrome may also be offered a bariatric procedure, although current evidence is limited by the lack of longterm data demonstrating net benefit. A recent Asian Consensus Meeting on Metabolic Surgery also recommended that the BMI cutoffs be lowered to 35 and 32.5, respectively, and that surgery be considered for Asian adults with BMI  $\geq 30$  kg/m<sup>2</sup> and central obesity (WC > 80 cm in females or > 90 cm in males) and at least two features of metabolic syndrome (raised triglycerides, low HDL cholesterol, hypertension, high-fasting plasma glucose). Gastric banding is a reversible restrictive procedure, while laparoscopic sleeve gastrectomy, Roux-en-Y gastric bypass, and biliopancreatic diversion combine restrictive and malabsorptive effects that produce 15–35% loss of baseline weight and improve other comorbidities.

#### **Check Your Progress**

**What is obesity? List the assessment for obesity.**

**Explain the causes of obesity.**

## **4.6 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS**

**Weight management** is the phrase used to describe both the techniques and underlying physiological processes that contribute to a person's ability to attain and maintain a certain weight. Most weight management



techniques encompass long-term lifestyle strategies that promote healthy eating and daily physical activity.

**Components of bodyweight:** A body composition scale breaks down that total **body weight** into the various **elements** that make it up. Your **body** is composed of three main **components**: fat, lean **body** mass (muscle, bone, and organs), and water.

**Techniques used for body weight - Body mass index (BMI)**

The body mass index (BMI) is a standard measurement used to determine whether a person’s weight is within normal range. The BMI is based on average findings and uses weight and height to determine approximately the degree of obesity.

BMI	Weight Status
Below 18.5	Underweight
18.5 – 24.9	Normal
25.0 – 29.9	Overweight
30.0 – 34.9	Obese
35.0 – 39.9	Severe obesity
≥40	Morbid obesity

**Waist circumference**

Gender	Low risk	High risk
Male	≤40 inches	>40 inches
Female	≤35 inches	>35 inches

BMI	Status	WC ≤35 (female) or ≤40 (male)	WC >35 (female) or >40 (male)
<18.5	Underweight		
18.5 – 24.9	Normal		
25.0 – 29.9	Overweight	Increased risk	High risk
30.0 – 34.9	Obese	High risk	Very high risk
35.0 – 39.9	Severe obesity	Very high risk	Very high risk
≥40	Morbid obesity	Extremely high risk	Extremely high risk

**Waist-to-hip ratio (WHR)**

Body shape is often described as “pear” with excess fat deposited in the buttocks and hips or “apple” with excess fat deposited in the abdomen. Apple-shaped individuals with increased intra-abdominal fat are at higher risk of health problems. In addition to simply measuring weight circumference, the waist-to-hip ratio (WHR) considers the ratio of the waist circumference to the hip circumference to determine if the person is an “apple” or “pear.”

**Skinfold thickness**

Measurement of skinfold thickness is a direct measure of fat, unlike the previous methods. The premise is that subcutaneous fat is a reflection of the proportion of total body fat. Calipers are used to grasp and measure fatty tissue in various parts of the body (protocols vary), such as the chin, biceps, triceps, subscapular, chest, abdominal hip, thigh, knee and calf, and then an equation used to calculate the percentage of body fat. The selected sites (usually 3 to 7) are believed to represent the average thickness of subcutaneous fat.

DXA, more commonly used to determine bone mineral density for diagnosis of osteoporosis, can also be used to determine fat content of the body and provides one of the most accurate measurements.

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## 4.7 SUMMARY

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This unit has taught us about weight management and obesity. Obesity, which broadly refers to excess body fat, has become an important public health problem. Its prevalence continues to increase worldwide. Obesity is predicted to rise over the coming years. Interventions to reduce the burden of obesity partly depend on recognising and understanding the complications of obesity. Clinicians are reminded to look for these complications in obese patients and institute interventions emphasizing the benefits of weight loss in obese patients.

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## 4.8 KEY WORDS

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- **Weight:** a body's relative mass or the quantity of matter contained by it, giving rise to a downward force; the heaviness of a person or thing.
- **Obesity:** Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health.
- **BMI:**Body Mass Index.

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## 4.9 SELF ASSESSMENT QUESTIONS AND ANSWERS

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### Short Answer Questions

1. Explain the weight management.
2. What are the different types of obesity?
3. Mention about the assessment followed in bodyweight components.
4. Brief the importance of obesity.

### Long Answer Questions

1. Elaborate on the different types of body weight components assessments.
2. Explain the types and causes of obesity.
3. Narrate the measures to overcome obesity.
4. Explain the dietary management of obesity.

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## **4.10 FURTHER READINGS**

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- 1 Ackroyd R, Mouiel J, Chevallier JM, Daoud F. Cost-effectiveness and budget impact of obesity surgery in patients with type-2 diabetes in three European countries. *Obesity Surgery*. 2006;16(11):1488–1503.
- 2 Agras WS, Berkowitz RI, Arnow BA, Telch CF, Marnell M, Henderson J, et al. Maintenance following a very-low-calorie diet. *Journal of Consulting and Clinical Psychology*. 1996;64(3):610–613.
- 3 Anselmino M, Bammer T, Cebrian JMF, Daoud F, Romagnoli G, Torres A. Cost-effectiveness and budget impact of obesity surgery in patients with type 2 diabetes in three European countries (II). *Obesity Surgery*. 2009;19(11):1542–1549.

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# **BLOCK – II: DIETETICS IN WEIGHT REDUCTION AND DIABETES**

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## **UNIT- V WEIGHT REDUCTION TECHNIQUES**

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### **Structure**

- 5.0 Introduction
- 5.1 Objectives
- 5.2 Weight Reduction Techniques
  - 5.2.1 Dietary Management
  - 5.2.2 Surgical Management
  - 5.2.3. Lifestyle modification
- 5.3 Underweight
  - 5.3.1 Causes
  - 5.3.2 Complications
  - 5.3.3 Dietary Management
- 5.4 Answer to check your progress questions
- 5.5 Summary
- 5.6 Key Words
- 5.7 Self-Assessment Questions
- 5.8 Further Readings

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### **4.0 INTRODUCTION**

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We have discussed enough about stress, weight management and obesity in our previous units. Now in this unit we shall discuss about the weight reduction and diabetes that are in common prevalence. A healthful eating pattern, regular physical activity, and often pharmacotherapy are key components of diabetes management. For many individuals with diabetes, the most challenging part of the treatment plan is determining what to eat. It is the position of the American Diabetes Association (ADA) that there is not a “one-size-fits-all” eating pattern for individuals with diabetes. The ADA also recognizes the integral role of nutrition therapy in overall diabetes management and has historically recommended that each person with diabetes be actively engaged in self-management, education, and treatment planning with his or her health care provider, which includes the collaborative development of an individualized eating plan (1,2). Therefore, it is important that all members of the health care team be knowledgeable about diabetes nutrition therapy and support its implementation.

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### **5.1 OBJECTIVES**

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After going through this unit, you will be able to:

- Discuss about weight reduction techniques, underweight, diabetes mellitus and diabetes incipidus and gestation diabetes
- Classifications, symptoms and complications
- Lifestyle modifications
- Impacts of dietary management

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## 5.2 WEIGHT REDUCTION TECHNIQUES

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Weight management is the phrase used to describe both the techniques and underlying physiological processes that contribute to a person's ability to attain and maintain a certain weight. Most weight management techniques encompass long-term lifestyle strategies that promote healthy eating and daily physical activity. Moreover, weight management involves developing meaningful ways to track weight over time and to identify ideal body weights for different individuals.

The most important component of an effective weight-management program must be the *prevention* of unwanted weight gain from excess body fat. There is significant evidence that losing excess body fat is difficult for most individuals and the risk of regaining lost weight is high. The principle of weight gain is simple: energy intake exceeds energy expenditure. Overweight and obesity are clearly the result of a complex set of interactions among genetic, behavioral, and environmental factors. While hundreds, if not thousands, of weight-loss strategies, diets, potions, and devices have been offered to the overweight public, the multi-factorial etiology of overweight challenges practitioners, researchers, and the overweight themselves to identify permanent, effective strategies for weight loss and maintenance. The percentage of individuals who lose weight and successfully maintain the loss has been estimated to be as small as 1 to 3 percent.

There are many factors that contribute to a person's weight, including: diet, physical activity, genetics, environmental factors, medications, and illnesses. Each of these factors affect weight in different ways and to varying degrees, but health professionals most often stress the importance of diet and physical activity above all other factors because they can be affected by conscious behavior modification.

Increased physical activity is an essential component of a comprehensive weight-reduction strategy for overweight adults who are otherwise healthy. One of the best predictors of success in the long-term management of overweight and obesity is the ability to develop and sustain an exercise program.

As valuable as exercise is, the existing research literature on overweight individuals indicates that exercise programs alone do not produce significant weight loss. The failure of exercise alone to produce significant weight loss may be because the neurochemical mechanisms that regulate eating behavior cause individuals to compensate for the calories expended in exercise by increasing food (calorie) intake. While exercise programs can result in an average weight loss of 2 to 3 kg in the short-term, outcome improves significantly when physical activity is combined with dietary intervention.

### 5.2.1. Dietary Management

Obesity is a growing concern because being overweight is widely regarded as a major risk factor for metabolic syndrome, cardiovascular disease, and premature death. Although the mechanisms for this weight gain have not been entirely elucidated, dietary factors may be important in the development of obesity. Diet consists of combinations of foods, and these individual components may have interactive or synergistic effects that make studying dietary factors in isolation difficult. Dietary patterns that represent a combination of foods may be more strongly associated with disease risk than an individual food and nutrient. Previous studies have reported that dietary patterns that are high in fruits, vegetables, and fibre might be associated with a reduced risk of obesity.

For the maintenance of a healthy weight, with a balanced diet and sufficient physical activity, in the general population, the guidance recalls dietary guidance and recommendations on active lifestyles, including building activity into daily life. Principles of Dietetics management are: Low calorie, normal protein, vitamin and mineral (except sodium), restricted carbohydrate, restricted fat and liberal fluid, high fiber diet are given.

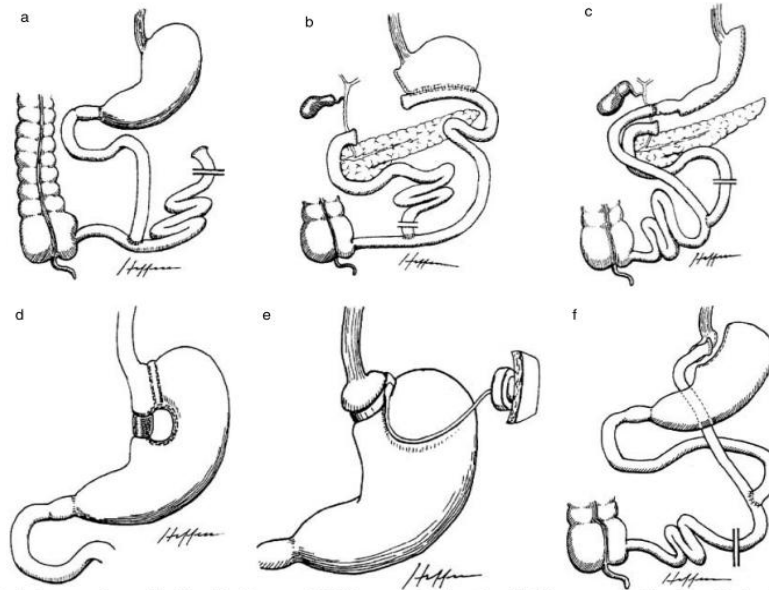
### 5.2.2. Surgical Management

Overweight and obesity are associated with insulin resistance, type 2 diabetes, dyslipidemia, hypertension, cholelithiasis, certain forms of cancer, steatosis hepatis, gastroesophageal reflux, obstructive sleep apnea, degenerative joint disease, gout, lower back pain, and polycystic ovary syndrome. Being overweight or obese at the age of 40 reduces life expectancy by at least 3 or 6 years.

The endemic extent of overweight individuals and obesity with their associated comorbidities has led to the development of therapies aimed at weight loss. The rates of bariatric surgery procedures are increasing sharply and to date it is the only option resulting in substantial and durable long-term weight loss.

Surgical treatments of obesity (bariatric surgery) techniques are divided into two groups: malabsorptive and restrictive procedures.

Malabsorptive procedures induce decreased absorption of nutrients by shortening the functional length of the small intestine. The created short-bowel syndrome leads to a negative energy balance and weight loss. The jejunoileal bypass (Fig. 1a) was one of the first bariatric operations. It is associated with substantial long-term complications including liver failure, malnutrition, electrolyte imbalances, vitamin deficiencies, renal (oxalate) stones, and death. This procedure is therefore no longer performed. Currently used malabsorptive techniques are the biliopancreatic diversion and the biliopancreatic diversion with duodenal switch (Fig. 1b and c).



**Figure 1** Bariatric procedures. (a) Jejunioleal bypass; (b) biliopancreatic diversion; (c) biliopancreatic diversion with duodenal switch; (d) vertical banded gastroplasty; (e) laparoscopic adjustable gastric band; and (f) Roux-en-Y gastric bypass. With permission from Ref. (129).

In both procedures, a partial gastrectomy is performed, creating a 100–150 ml gastric pouch. The biliopancreatic diversion (Fig. 1b) consists of a horizontal distal gastrectomy with a gastro-jejunosomy or gastroileostomy; this long (food) limb is anastomosed to the biliopancreatic (bile, a pancreatic juice) limb. In a biliopancreatic diversion with duodenal switch (Fig. 1c), a pylorus-sparing sleeve gastrectomy with duodeno-ileostomy is performed. It is generally accepted that biliopancreatic diversion with duodenal switch results in less cases of dumping and marginal ulcers than a classical biliopancreatic diversion. In both procedures, the length of the common limb – i.e., the time during which digestion and nutrient absorption can occur – determines the degree of malabsorption. Restrictive operations reduce the storage capacity of the stomach and as a result early satiety arises, leading to a decreased caloric intake. In general, restrictive procedures are simpler to perform and are accompanied by less procedural complications than malabsorptive procedures.

The vertical banded gastroplasty and the laparoscopic adjustable gastric band represent the current most frequently performed restrictive procedures (Fig. 1d and e). During a vertical banded gastroplasty (Fig. 1d), the fundus of the stomach is stapled parallel to the lesser curve using a surgical stapling device. The distal exit of the created pouch is narrowed with a band. A foodreceiving reservoir of w50 ml remains and the banding provides an outlet diameter of 10–12 mm. The laparoscopic adjustable gastric band technique (Fig. 1e) involves placing a silicon inflatable gastric band horizontally around the proximal part of the stomach. By inflating the gastric band via a s.c. port, a pouch is created. Moreover, the diameter of the band can be adjusted to the individual needs of the patient. The advantage of the latter technique is that in the case of excessive vomiting or reflux following the operation (i.e., when the exit of the pouch is too narrow) the tension of the gastric band can be reduced.

### Effectiveness of bariatric surgery

## Weight

The effectiveness of bariatric surgery has recently been studied in two meta-analyses. On average surgical treatment of obesity results in 20–40 kg of weight loss and a 10–15 kg/m<sup>2</sup> reduction in BMI. In the SOS study, the average 10-year weight loss was well over 19 kg. This result was achieved despite the fact that vertical banded gastroplasty was the dominant procedure and only 5% of those who had a follow-up of 10 years had a Roux-en-Y gastric bypass (which is superior to vertical banded gastroplasty regarding weight loss). After a period of 15 years, patients who had undergone laparoscopic gastric banding had lost 13±14% compared with the baseline weight. Corresponding weight losses 15 years after vertical banded gastroplasty and Roux-en-Y gastric bypass were 18±11% and 27±12% respectively.

Few studies have compared weight loss between surgical procedures. In two randomized clinical trials collectively enrolling 231 patients, Roux-en-Y gastric bypass was compared with vertical banded gastroplasty. Pooled results showed that at 12 and 36 months patients assigned to Roux-en-Y gastric bypass lost substantially more weight than those assigned to vertical banded gastroplasty (42.43 kg versus 34.45 kg and 39.73 kg versus 30.65 kg at 12 and 36 months respectively). On the basis of these and other studies, it can be concluded that in regard to weight loss, Roux-en-Y gastric bypass is superior to vertical banded gastroplasty. Recently, laparoscopic adjustable gastric banding was compared with laparoscopic vertical banded gastroplasty in a randomized trial involving 100 patients. In this study, excess weight loss was 58.9% 3 years after laparoscopic vertical banded gastroplasty and 39% 3 years after laparoscopic adjustable gastric banding. Biliopancreatic diversion resulted in an excess weight loss of 74±12% at 2 years and 72±10% at 8 years after surgery.

In biliopancreatic diversion with duodenal switch, a mean weight loss of 46±20 kg was reported in 252 patients followed for a mean of 8.3 years. Placement of an intragastric balloon for 6 months resulted in an excess weight loss of 33.9±18.7%. In well-selected patients, it seems possible to extend the treatment period to 1 year. However, only about half of the patients maintain a weight loss of over 50% in the year after removal of the balloon. Taken together it can be concluded that long-term weight loss after Roux-en-Y gastric bypass is less than after biliopancreatic diversion, but more than when compared with restrictive surgical procedures including intragastric balloon. Mean weight loss is maximal after 1–2 years and slowly increases until year 8–10 after which body weight stabilizes.

## Diabetes mellitus

Recovery from type 2 diabetes was established in 76.8% of the patients who underwent bariatric surgery. In the surgically treated group of the SOS study, type 2 diabetes had disappeared in 72% after 2 years. Unfortunately, 'only' 36% of those who had diabetes at entry remained free of the disorder at 10 years. In the conventionally treated group, these percentages were 21 and 13% respectively. Similar recovery rates are described in other studies as well. Moreover, these percentages seem related to the operative procedure that is used, with vertical banded



gastroplasty, laparoscopic adjustable gastric banding, and Roux-en-Y gastric bypass resulting in resolution of diabetes in about 40, 60, and 80% of patients respectively. Several studies using homeostatic model assessment have reported improvements in insulin sensitivity and b-cell function.

These data are substantiated by studies that have used euglycemic–hyperinsulinemic clamp and i.v. glucose tolerance testing. Interestingly, many patients become euglycemic well before the weight loss occurs. Those procedures that expedite nutrient supply to the lower gastrointestinal tract appear to be especially promising in this respect. Indeed, in surgically treated obese (albeit nondiabetic) subjects studied with the insulin clamp technique, gastric bypass improves insulin sensitivity in proportion to weight loss whereas insulin resistance is completely restored long before normalization of body weight after biliopancreatic diversion.

### **Lipids**

Hypercholesterolemia and hypertriglyceridemia improve after surgical treatment of obesity irrespective of the technique used. Total and low density lipoprotein (LDL) cholesterol concentrations decreased with an average of 0.86 mmol/l (95% confidence interval (CI) 0.60–1.13 mmol/l) and 0.76 mmol/l (95% CI 0.46–1.06 mmol/l) respectively. While triglyceride concentrations decrease with an average of 0.90 mmol/l (95% CI 0.73–1.08 mmol/l), high density lipoprotein (HDL) cholesterol concentration showed no difference in a combined analysis of all surgical procedures. However, patients who underwent vertical banded gastroplasty (nZ253) or gastric banding (nZ623) showed an increase in concentration of HDL cholesterol of 0.13 mmol/l (95% CI 0.02–0.24 mmol/l) and 0.12 mmol/l (95% CI 0.04–0.20 mmol/l) respectively.

### **Hypertension, obstructive sleep apnea, and polycystic ovary syndrome**

A benefit of surgery in reducing the prevalence of hypertension and obstructive sleep apnea has also been shown. Of the surgically treated patients, 62% of those with hypertension and 86% of those with obstructive sleep apnea recovered. In patients with polycystic ovary syndrome treated with bariatric surgery (either laparoscopic gastric bypass or biliopancreatic diversion), hirsutism, hyperandrogenemia, insulin resistance, and ovulation and/or restoration of menstrual cycle significantly improved in all.

### **Quality of life**

The SOS study found a dramatic improvement in the quality of life at 2 years among patients who had had surgical treatment for obesity, particularly concerning psychological performance. Unsurprisingly, there is a strong positive correlation between the degree of improvement in quality of life and the degree of weight loss. In another study, 95% of 275 patients who underwent laparoscopic Roux-en-Y gastric bypass surgery reported improvement in quality of life. Similar results have been reported for laparoscopic adjustable gastric banding.

### **Mortality**

Retrospective cohort studies have suggested that bariatric surgery leads to a decrease in mortality; two published studies have shown that for obese patients with diabetes mellitus, bariatric surgery reduces mortality considerably. Moreover, in nondiabetic patient's mortality reduction after bariatric surgery has also been demonstrated: in a cohort of 1035 patients with an average BMI of 50 kg/m<sup>2</sup> undergoing bariatric surgery, mortality declined by 89% when compared with a control group which did not receive surgical treatment for obesity (0.68% vs 6.17%; relative risk 0.11, 95% CI 0.04–0.27). Long-term mortality data for gastric bypass surgery were very recently reported. In a large retrospective cohort study (9949 gastric bypass procedures versus 9628 severely obese controls), long-term mortality from any cause (mean follow-up of 7.1 years) decreased by 40% compared with that in the control group. Prospective mortality data are provided by the 'SOS' study; during an average of 10.9 years follow-up subjects who underwent bariatric surgery had an overall mortality hazard ratio of 0.76 when compared with control subjects. In conclusion, bariatric surgery is an effective treatment option for long-term reduction of body weight and amelioration of obesity-related comorbid conditions. Importantly, evidence shows that for obese patients – with or without diabetes mellitus – surgical treatment of obesity leads to a reduction in mortality.

### **5.2.3. LIFESTYLE MODIFICATION**

The use of behavior and lifestyle modification in weight management is based on a body of evidence that people become or remain overweight as the result of modifiable habits or behaviors, and that by changing those behaviors, weight can be lost and the loss can be maintained. The primary goals of behavioral strategies for weight control are to increase physical activity and to reduce caloric intake by altering eating habits.

#### **Overview of lifestyle treatments for weight loss**

Lifestyle treatments for weight loss focus on reducing energy intake and increasing physical activity through diet, exercise and behavioural measures. You should try to alter your bad eating and activity habits. (Please refer to eating habits of women and eating habits of men for more information). In general, a combination of treatments is the most effective way to achieve weight loss.

A program is now available online that helps you to determine which of your lifestyle behaviours are unhealthy. This is called the Diet, Activity and Behaviour Questionnaire (DAB-Q). It consists of a series of simple questions about eating and activity behaviours and helps to grade how easy it would be for us to make appropriate changes. At the end of the questionnaire you will be given a graded score of the most important factors contributing to your excess weight, which you can hopefully address in the future.

Lifestyle treatments for obesity and weight loss can be challenging and time consuming. To re-emphasise, much of the success of treatment will rely on our own dedication and effort.

### **Dietary approaches for weight loss**

Dietary control has probably been the main treatment used for weight loss in the past. Diets are based on the principles of metabolism and work by reducing the intake of calories (energy) to create a negative energy balance (i.e. more energy is used than is consumed). There are countless commercial diets available and you should try to choose one that is suitable for you. The crash or fad diets published in magazines should generally be avoided, as they can be dangerous to your health and tend not to produce good long term results. You should try to choose diets that are medically proven or diets developed by dedicated weight loss services such as Weight Watchers or Jenny Craig. Your doctor can help you choose a diet which is safe, effective and suited to your needs. Below are some of the different mechanisms used in diets for weight loss:

- Reducing fat, carbohydrate (especially those with a high glycaemic index), protein or alcohol intake. Reducing alcohol intake is a very good way to lose weight, as each gram of alcohol contains a large amount of energy on top of your normal daily intake.
- Smaller portion sizes. You can try using a smaller plate size at each meal.
- Food restrictions at various times of the day. It can be helpful to have a big breakfast and reduce the intake of energy rich foods later in the day. This can help your metabolism and ensure that most of the energy that you consume is burnt throughout the day.
- Combining different foods to reduce total energy intake. Some diets have set food regimes for weight loss.
- Diets centered on a single low-energy food.
- 



Most diets produce some weight loss and are successful in the short-term. However, less than 10% of patients will maintain the weight loss in the long term. Once again it must be emphasised that permanent changes to eating habits are required. You must be careful whilst dieting to ensure that you still receive all the essential proteins, vitamins and trace elements. The best way to achieve this is by eating a well balanced diet with a wide variety of nutritious foods. A successful weight loss program may include cutting fats and sugars from your diet while ensuring you eat lots of healthy foods such as wholegrain, fruits and vegetables. Some diet programs may also require you to take vitamin or mineral supplements.

Be careful when shopping for low fat foods or brands labeled as 'diet' in the supermarket. Many low fat foods can be high in sugar and still

contribute a lot to your daily energy or caloric intake. You should learn how to read food labels carefully and aim to consume foods that are low in saturated fat, sugars and, if appropriate, glycaemic index.

Diets that teach you how to select and prepare healthy foods may be more successful in the long term than restrictive diets with strict daily eating regimes or pre-prepared meals.

**Reduced energy diets** encourage you to choose healthy meal options, aiming to reduce your energy intake by a small amount every day. They teach you healthy eating behaviours which you can maintain in the long term.

**Low energy diets** are more restrictive and limit your energy intake to a greater degree. Set meal programs need to be followed. This type of diet can lead to a weight loss between 7-13 kilograms and may be used if you have significant health problems related to obesity.

**Very low energy diets** cut daily energy intake significantly and tend to be reserved for people who have failed other treatments or who have significant co-morbidities. These types of diets are generally followed for 8-16 weeks and often consist of liquid meal replacements (discussed below) from pharmacies. Unfortunately much of the weight lost is regained after the diet is stopped, but behavioural or drug therapies following treatment can help maintain some of the weight loss.

### **Meal Replacement Programs**

Recently weight loss programs have been developed that replace normal meals with prepared meal plans or meal supplements (such as vitamin-rich shakes, soups and bars). These supplements act as complete meals, as they contain all the required vitamins and minerals. Meal Replacement Programs operate on the principles of a low calorie diet and induce a mild state of ketosis. The diets limit your intake of carbohydrates so that the body starts to break down fat stores for energy. The meal replacement programs are specially formulated so that they contain adequate energy and do not cause malnutrition.

### **Physical activity for weight loss**



In addition to reducing your energy intake, increased physical activity is essential for the maintenance of weight loss and should form part of any weight loss program. However, to achieve significant weight loss

from exercise alone, a very high level of activity is required, which can be challenging.

You should gradually build up your exercise as your personal fitness allows. You can start with simple measures such as walking to nearby places rather than driving, or climbing the stairs rather than using an elevator or escalator. Thirty minutes of walking 3-5 times per week is a good starting point.

If you are very overweight, some exercises (especially those that require weight bearing) can be physically difficult. In this case you could try activities such as swimming, walking in water or cycling. Once your fitness levels improve you could change to other exercises in the long term. You should try to choose activities that you enjoy, as you will be more likely to continue them in the long term. Participating in team sports or exercising with a friend or family member can help you to remain motivated. It may be useful to see a physiotherapist or exercise physiologist to help you develop an appropriate exercise program. Try to focus on overall lifestyle measures as they tend to produce greater long term adherence.

Exercise has benefits beyond modest reductions in weight. Increasing your physical activity can improve your cardio-respiratory fitness, metabolic health, quality of life and general wellbeing. However, strenuous exercise can be risky in some patients such as those with cardiovascular problems. You should consult your doctor to discuss what level of physical activity will be safe for you.

### **Behavioural therapy for weight loss**

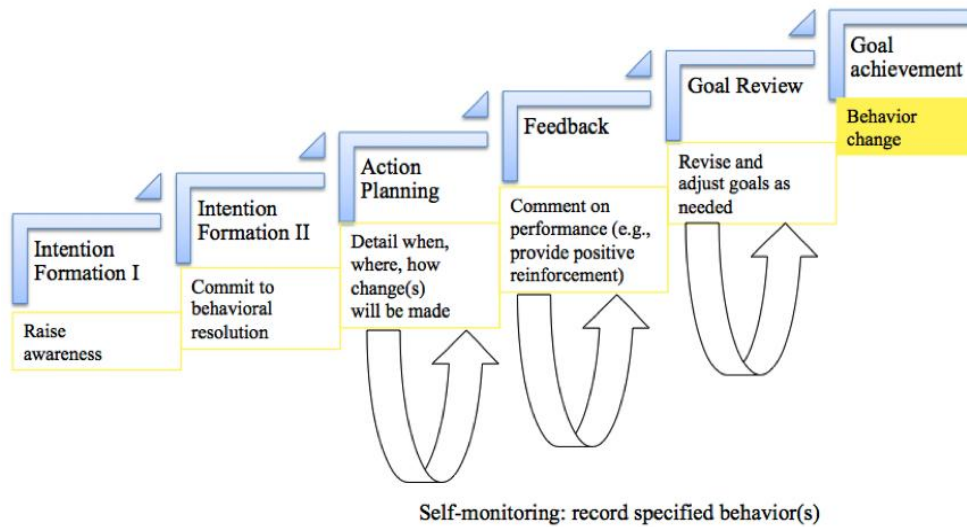
Behavioural techniques may be useful in conjunction with diet and exercise programs to improve long-term weight loss. Behavioural treatment is usually performed by a psychologist who analyses your eating, physical activity and thinking habits. Cognitive behavioural therapy (CBT) is a classic example which encourages you to change these behaviours and take responsibility for your lifestyle changes. Other strategies may include stress management, relapse prevention, counselling, and techniques such as hypnosis and psychotherapy. However, some of these behavioural techniques can be very time consuming and expensive.

Some basic steps may include:

- **Self monitoring:** Identifying and recording any adverse patterns of behaviour.
- **Stimulus control:** This involves removing factors that encourage you to eat badly. For example, you should shop carefully and remove trouble foods (such as chocolates and chips) from the house so you cannot be tempted.
- **Problem solving:** Identifying and addressing problems associated with eating and physical activity. You should consider which factors have led to your excess weight and address them appropriately.

**Reward systems:** You could reward yourself for positive behaviours such as exercise by treating yourself to new clothes.

- **Social support:** Strong social support from your friends and family can improve weight loss. It may be beneficial for you to enrol in a commercial program which offers a social support network.



### Other

Beyond the specific measures highlighted above, it is also important that you maintain an overall balanced and healthy lifestyle. You should avoid stress, depression, boredom and frustration as these can be triggers to unhealthy eating behaviours. Keeping yourself occupied with enjoyable activities such as sports or socializing with friends and family can help you achieve a healthy lifestyle. If you eat excessively due to stress you could consider enrolling in relaxation programs.

#### Check Your Progress

1. Write a short note on weight reduction.
2. Write about weight reduction bariatric surgery?

## 5.3 UNDERWEIGHT

An underweight person is a type of person whose body weight is considered too low to be healthy. Underweight people have a Body Mass Index (BMI) of under 18.5 or a weight 15% to 20% below that normal for their age and height group.

Underweight is body weight that is too low for a normal healthy adult or child. It is also known by various other names such as wasting, emaciation, thinness, stunting, etc., and is caused by multiple factors especially lack of adequate nutrients in the body.

Half the Indian rural population is underweight with up to three-quarters being so classified in deprived communities.

A very large proportion of rural Indian households have inadequate food supplies. Chronic energy deficiency is due to chronic food deficiency.

In today's world, thinness is often praised. But being underweight, when your weight is lower than what is considered healthy for your height, can be a sign of a serious health problem.

If you're underweight, you may be more likely to also be malnourished if your low BMI is caused by an unbalanced diet or an underlying disease that affects nutrient absorption. Malnutrition can also lead to anemia or a deficiency in essential vitamins. Anemia can also be caused by malabsorption of nutrients.

Being underweight can represent as many health concerns to an individual as being overweight can. If a person is underweight, their body may not be getting the nutrients it needs to build healthy bones, skin, and hair. While some people may have a genetic background or a medical illness that prevents them from putting on weight, there are interventions doctors can recommend to help a person gain weight.

In this book, we look at ways to tell if you are underweight, causes, complications, and dietary managements.

### 5.3.1. Causes

If you're underweight, you may not be eating enough healthy foods with key nutrients to fuel your body. That can cause malnutrition. Over time, malnutrition can affect your health in a number of different ways that may be noticeable to you or those around you.

There are a variety of reasons why a person may be underweight. Sometimes, multiple underlying causes may be related. Causes of being underweight include:

- **Family history.** Some people have a naturally low BMI due to physical characteristics that run in their family.
- **Frequent physical activity.** Underweight may occur in people who are active, tense, nervous and who never take rest. When it reaches a severe state, psychological efficiency as a whole is reduced and their power for concentration, decision making and withstanding calamities are very poor. Athletes or people who engage in high levels of physical activity, such as runners, may burn significant amounts of calories that result in low body weight.
- **Physical illness or chronic disease.** Some disease types can cause regular nausea, vomiting, and diarrhea, making it difficult to gain weight. Other conditions may decrease a person's appetite, so they do not feel like eating. Examples include cancer, diabetes, thyroid disorders, and digestive conditions, such as Crohn's disease or ulcerative colitis. Infections are common among them. In these cases tonics are not useful as they only help to improve the appetite but they do not increase weight. It is wise and necessary to spend money on nourishing high calorie foods.
- **Mental illness.** Poor mental health can affect a person's ability to eat, including depression, anxiety, obsessive-compulsive disorder (OCD), and eating disorders, such as anorexia and bulimia. Each of these conditions can affect a person's body image and appetite.
- It occur in pathological conditions such as fevers, gastrointestinal disturbances, where the digestion and absorption capacities are decreased and in hyperthyroidism.

- Starvation occurs either due to famine conditions or an inadequate diet in proteins or an attempt at reducing weight. During starvation fatty tissue is lost and the skeletal, heart muscles and small intestine are atrophied losing its absorptive function. This result in low blood pressure, marked emaciation, loss of hair and inelastic skin. Since the feeding programme should be gradual, initially glucose water, fruit juice and skimmed milk powder may be given.
- Psychological factors may contribute to eating very little food (anorexia nervosa). Some mental patients reject food leading to severe weight loss. Anorexia nervosa is seen in girls between the age of 15 and 25 years. Usually it arises from a desire to lose weight. If untreated this condition may prove fatal due to progressive starvation. Electrolytes are lost in vomiting at the sight of food, or during eating. In such cases, patients must be admitted to a hospital and should be given specific directions to have high calories intake of bread, wheat and milk product.
- Many different things can cause women to be underweight, including eating habits, health problems, and medicines. Underweight can cause other health problems and problems getting pregnant. Transient stoppage of menstruation may occur which becomes normal with dietary treatment.

### 5.3.2. Complications

Being underweight can cause health problems, just as being overweight can.

Not all people who are underweight experience adverse side effects or symptoms from being underweight. However, some people, experience the following symptoms related to being underweight:

- **Osteoporosis.** According to a 2016 study, being underweight increases a woman's risk of osteoporosis, which is where the bones are brittle and more prone to breaking. Low body weight may increase your risk for low bone mineral density (BMD) and osteoporosis. There are studies which looked at BMD in 1,767 premenopausal women, and found that 24 percent of women with a BMI of 18.5 or lower had a low BMD. Only 9.4 percent of participants with a BMI higher than 18.5 had low BMD. The study results suggest that being underweight increases risk for osteoporosis.
- **Skin, hair, or teeth problems.** If a person does not get enough nutrients in their daily diet, they may display physical symptoms, such as thinning skin, hair loss, dry skin, or poor dental health.
- **Getting sick frequently.** If a person does not get enough energy from their diet to maintain a healthy body weight, they may also not be getting enough nutrients to fight off infections. As a result, a person may get sick more frequently, and common illnesses, such as a cold, can last longer than they usually would.
- **Feeling tired all the time.** Calories are a measurement of the energy a particular food can give a person. Not getting enough calories to maintain a healthy weight can make a person feel fatigued.



- **Anemia.** A person who is underweight is more likely to have low blood counts, known as anemia, which causes dizziness, headaches, and fatigue.
- **Irregular periods.** Women who are underweight may not have regular periods, they may find menstruation stops, or an adolescent's first period may be delayed or absent. Irregular or absent menstruation can cause infertility. Women with low BMIs are at increased risk for amenorrhea, which is an absence of menses, and other menstrual cycle dysfunctions. Irregular or missed menstrual cycles may be an indicator of anovulation, or that you aren't ovulating. Chronic anovulation may cause infertility.
- **Premature births.** According to a study published in *An International Journal of Obstetrics & Gynaecology*, a woman who is pregnant and underweight is at a higher risk for pre-term labor, which means having a baby before 37 weeks.
- **Slow or impaired growth.** Young people need nutrients to grow and develop healthy bones. Being underweight and not getting enough calories could mean a person may not develop as expected. Doctors call this a 'failure to thrive.'

### 5.3.2. Dietary Management

Nutrition therapy can help you eat more calories and gain weight. As you gain weight, your health may improve. A high calorie, high protein, high fat diet with liberal vitamin intake is recommended.

Just like obesity causes health concerns, having less weight than the normal also poses health issues. Being underweight can be result of poor nutrition and should be a matter of concern. If the body does not receive adequate amount of nutrients, the body fails to function to its utmost. This could result in the imbalance in the metabolism. Inculcating a healthy diet for underweight will provide the necessary nutrients for better functioning of the body.

In this case, the person suffering from underweight should include:

- 1 Heavy food items that are more in calories.
- 2 Frequent consumption of food items which are rich in nutrients, it could be snacks, shakes or juices, or proper meals.
- 3 Adding extra ingredients that are high in calories to regular diet, for example, including eggs and bananas in morning breakfast, etc., can help in increasing the weight.
- 4 Consume protein supplements along with adequate amount of vegetables and fruits.
- 5 Eating calorie dense food and maintaining a balanced diet will help in gaining the weight.
- 6 However, the diet shouldn't be started drastically and instead, should be implemented gradually so that the body is accustomed with it.

#### Check Your Progress

1. Write a short note on underweight.
2. Write about causes and dietary guidelines for underweight?

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## 5.4. ANSWERS TO CHECK YOUR PROGRESS QUESTIONS

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Weight management is the phrase used to describe both the techniques and underlying physiological processes that contribute to a person's ability to attain and maintain a certain weight. Most weight management techniques encompass long-term lifestyle strategies that promote healthy eating and daily physical activity.

Diet consists of combinations of foods, and these individual components may have interactive or synergistic effects that make studying dietary factors in isolation difficult. Dietary patterns that represent a combination of foods may be more strongly associated with disease risk than an individual food and nutrient. Previous studies have reported that dietary patterns that are high in fruits, vegetables, and fibre might be associated with a reduced risk of obesity.

Surgical treatments of obesity (bariatric surgery) techniques are divided into two groups: malabsorptive and restrictive procedures.

Malabsorptive procedures induce decreased absorption of nutrients by shortening the functional length of the small intestine. The created short-bowel syndrome leads to a negative energy balance and weight loss.

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## 5.5 SUMMARY

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This unit has thoroughly given an understanding of the sections of weight reduction and under weight. Sedentary life style, faulty cooking practices, alcoholism also aggravate the conditions. Causes- A person may be underweight due to genetics, metabolism, drug use, lack of food (frequently due to poverty), eating disorder, or illness (both physical and mental). Being underweight is associated with certain medical conditions, including anorexia, type 1 diabetes, hyperthyroidism, cancer, or tuberculosis.

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## 5.6 KEY WORDS

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- **Techniques:** a way of carrying out a particular task, especially the execution or performance of an artistic work or a scientific procedure.
- **Causes:** a person or thing that gives rise to an action, phenomenon, or condition
- **Lifestyle:** the way in which a person lives.

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## 5.7 SELF ASSESSMENT QUESTIONS

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### Short Answer Questions

1. Differentiate weight reduction and underweight.

2. Write short note on types of bariatric surgery.
3. Explain in brief about underweight.
4. Short notes on weight reduction lifestyle management.

**Long Answer Questions**

1. Summarize the surgical management of weight reduction.
2. Elaborate the causes and complication of underweight.
3. Explain the dietary management of underweight.

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**5.8 FURTHER READINGS**

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# UNIT –VI DIABETES MELLITUS

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*Diabetes Mellitus*

NOTES

## **Structure**

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## **6.0 INTRODUCTION**

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Diabetes mellitus (DM), commonly known as diabetes, is a group of metabolic disorders characterized by high blood sugar levels over a prolonged period. Symptoms of high blood sugar include frequent urination, increased thirst, and increased hunger. If left untreated, diabetes can cause many complications. Acute complications can include diabetic ketoacidosis, hyperosmolar hyperglycemic state, or death.<sup>[3]</sup> Serious long-term complications include cardiovascular disease, stroke, chronic kidney disease, foot ulcers, and damage to the eyes.

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## **6.1 OBJECTIVES**

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After going through this unit you will be able to discuss,

- The consequences of diabetes
- The different strategies to diabetes to overcome.

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## **6.2 DIABETES MELLITUS**

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### **Introduction**

Diabetes mellitus is a heterogeneous group of diseases characterized by chronic elevation of glucose in the blood. It arises because the body is unable to produce enough insulin for its own needs, either because of impaired insulin secretion, impaired insulin action, or both. Diabetes affects some 300 million people world-wide, and is on the increase. Chronic exposure to high blood glucose is a leading cause of renal failure, visual loss and a range of other types of tissue damage. Diabetes also predisposes to arterial disease, not least because it is often accompanied by hypertension, lipid disorders, and obesity. Many cases of diabetes and almost all of its unwanted long-term consequences are

*Self-Instructional Material*

potentially avoidable, but this will require intervention at a societal as well as at a medical level. This section of Diapedia offers an introduction to the history of diabetes, its clinical presentation, its current classification, and its global epidemiology. We also introduce some of the psychological and societal aspects of diabetes, including the 'hot topics' that dominate the media, and offer an overview of current areas of research interest. All these topics are considered in greater detail elsewhere in Diapedia, and we hope you will explore them further.

### 6.2.1. CLASSIFICATION

If any characteristic can define the new intentions for DM classification, it is the intention to consolidate etiological views concerning DM. The old and confusing terms of insulin-dependent (IDDM) or non-insulin-dependent (NIDDM) which were proposed by WHO in 1980 and 1985 have disappeared and the terms of new classification system identifies four types of diabetes mellitus: type 1, type 2, "other specific types" and gestational diabetes. The etiologic classifications of diabetes mellitus are listed in (Table 2).

#### Type 1 diabetes mellitus

Type 1 diabetes mellitus (juvenile diabetes) is characterized by beta cell destruction caused by an autoimmune process, usually leading to absolute insulin deficiency [20]. Type 1 is usually characterized by the presence of anti-glutamic acid decarboxylase, islet cell or insulin antibodies which identify the autoimmune processes that lead to beta cell destruction. Eventually, all type 1 diabetic patients will require insulin therapy to maintain normglycemia.

#### Type 2 diabetes mellitus

The relative importance of defects in insulin secretion or in the peripheral action of the hormone in the occurrence of DM2 has been and will continue to be cause for discussion. DM2 comprises 80% to 90% of all cases of DM. Most individuals with Type 2 diabetes exhibit intra-abdominal (visceral) obesity, which is closely related to the presence of insulin resistance. In addition, hypertension and dyslipidemia (high triglyceride and low HDL-cholesterol levels; postprandial hyperlipidemia) often are present in these individuals. This is the most common form of diabetes mellitus and is highly associated with a family history of diabetes, older age, obesity and lack of exercise. It is more common in women, especially women with a history of gestational diabetes, and in Blacks, Hispanics and Native Americans.

Ranking	2000		2030	
	Country	People with diabetes (millions)	Country	People with diabetes (millions)
1	India	31.7	India	79.4
2	China	20.8	China	42.3
3	U.S.	17.7	U.S.	30.3
4	Indonesia	8.4	Indonesia	21.3
5	Japan	6.8	Pakistan	13.9
6	Pakistan	5.2	Brazil	11.3
7	Russia	4.6	Bangladesh	11.1
8	Brazil	4.6	Japan	8.9
9	Italy	4.3	Philippines	7.8
10	Bangladesh	3.2	Egypt	6.7

### List of countries with the highest numbers of estimated cases of diabetes for 2000 and 2030. Adapted from Wild S [5].

#### Gestational Diabetes Mellitus (GDM)

Gestational diabetes mellitus is an operational classification (rather than a pathophysiologic condition) identifying women who develop diabetes mellitus during gestation. Women who develop Type 1 diabetes mellitus during pregnancy and women with undiagnosed asymptomatic Type 2 diabetes mellitus that is discovered during pregnancy are classified with Gestational Diabetes Mellitus (GDM). In most women who develop GDM; the disorder has its onset in the third trimester of pregnancy.

#### Other specific type (Monogenic diabetes)

Types of diabetes mellitus of various known etiologies are grouped together to form the classification called “Other Specific Types”. This group includes persons with genetic defects of beta-cell function (this type of diabetes was formerly called MODY or maturity-onset diabetes in youth) or with defects of insulin action; persons with diseases of the exocrine pancreas, such as pancreatitis or cystic fibrosis; persons with dysfunction associated with other endocrinopathies (e.g. acromegaly); and persons with pancreatic dysfunction caused by drugs, chemicals or infections and they comprise less than 10% of DM cases.

#### 6.2.3.SYMPTOMS

People can often have diabetes and be completely unaware. The main reason for this is that the symptoms, when seen on their own, seem harmless. However, the earlier diabetes is diagnosed the greater the chances are that serious complications, which can result from having diabetes, can be avoided. Here is a list of the most common diabetes symptoms:

- ❖ **Frequent urination:** Have you been going to the bathroom to urinate more often recently? Do you notice that you spend most of the day going to the toilet? When there is too much glucose (sugar) in your blood you will urinate more often. If your insulin is ineffective, or not there at all, your kidneys cannot filter the glucose back into the blood. The kidneys will take water from your blood in order to dilute the glucose - which in turn fills up your bladder.
- ❖ **Disproportionate thirst:** If you are urinating more than usual, you will need to replace that lost liquid. You will be drinking more than usual. Have you been drinking more than usual lately.
- ❖ **Intense hunger:** As the insulin in your blood is not working properly, or is not there at all, and your cells are not getting their energy, your body may react by trying to find more energy - food. You will become hungry.
- ❖ **Weight gain:** This might be the result of the above symptom (intense hunger).
- ❖ **Unusual weight loss:** This is more common among people with Diabetes Type 1. As your body is not making insulin it will seek out another energy source (the cells aren't getting glucose). Muscle tissue and fat will be broken down for energy. As Type 1 is of a more sudden onset and Type 2 is much more gradual, weight loss is more noticeable with Type 1.

- ❖ **Increased fatigue:** If your insulin is not working properly, or is not there at all, glucose will not be entering your cells and providing them with energy. This will make you feel tired and listless.
- ❖ **Irritability:** Irritability can be due to your lack of energy.
- ❖ **Blurred vision:** This can be caused by tissue being pulled from your eye lenses. This affects your eyes' ability to focus. With proper treatment this can be treated. There are severe cases where blindness or prolonged vision problems can occur.
- ❖ **Cuts and bruises don't heal properly or quickly:** Do you find cuts and bruises take a much longer time than usual to heal? When there is more sugar (glucose) in your body, its ability to heal can be undermined.
- ❖ **More skin and/or yeast infections:** When there is more sugar in your body, its ability to recover from infections is affected. Women with diabetes find it especially difficult to recover from bladder and vaginal infections.
- ❖ **Itchy skin:** A feeling of itchiness on your skin is sometimes a symptom of diabetes.
- ❖ **Gums are red and/or swollen - Gums pull away from teeth:** If your gums are tender, red and/or swollen this could be a sign of diabetes. Your teeth could become loose as the gums pull away from them.
- ❖ **Frequent gum disease/infection:** As well as the previous gum symptoms, you may experience more frequent gum disease and/or gum infections.
- ❖ **Sexual dysfunction among men:** If you are over 50 and experience frequent or constant sexual dysfunction (erectile dysfunction), it could be a symptom of diabetes.
- ❖ **Numbness or tingling, especially in your feet and hands:** If there is too much sugar in your body your nerves could become damaged, as could the tiny blood vessels that feed those nerves. You may experience tingling and/or numbness in your hands and feet.

#### 6.2.4. DIAGNOSIS

Keeping in mind the consequences that DM can have for the affected individual, the clinician must be certain when establishing a diagnosis of DM. In the case of florid and persistent symptoms and the presence of sufficiently elevated glycemia numbers, the diagnosis will be obvious in the majority of cases. However, it must not be forgotten that in a great many cases the diagnosis is made in asymptomatic persons following a routine analytical examination.

##### **Diabetes mellitus**

The diagnosis of DM can be made in the following situations (Table 1): a) occasional plasma glycemia  $\geq 200$  mg/dL (11.1 mmol/L) (obtained at any time of day and without regard to when food was last ingested) and symptoms of DM (polyuria, polydipsia and inexplicable weight loss); b) fasting plasma glycemia (FPA)  $\geq 126$  mg/dL (7.0 mmol/L), fasting being a period of at least 8 hours without ingestion of food), or c) plasma glycemia  $\geq 200$  mg/dL (11.1 mmol/L) at 2 hours after an oral glucose tolerance test (GTT). The test must be carried out according to

WHO criteria (published in 1985), with 75 g of anhydrous glucose dissolved in water. It should be pointed out that in the absence of unequivocal hyperglycemia with acute metabolic decompensation, the criteria must be repeated again. The change of the cut-off point for FPG to a  $\geq 126$  mg/dL (previously 140 mg/dL) is based on the fact that a) this is equivalent (in population-based studies) to the cut-off point for diagnosing diabetes by a plasma glucose  $\geq 200$  mg/dL in a GTT; b) it represents a better cut-off point for separating the bimodal distribution of fasting plasma glycemia in the population; and c) in several studies this number marked the inflection point for establishing the risk of microangiopathy. While the GTT is not recommended as a routine diagnostic method in daily practice according to ADA recommendations, WHO encourages performing it as some subjects diagnosed with FPG may be different from those in whom the diagnosis has been established by GTT. In addition, the frequency of DM is lower when the ADA criteria are applied, and, in fact, approximately 30% of subjects (in studies on the European population) with a non-diabetic FPG met the criteria for DM once a GTT was performed.

### Intermediate categories between normal clinical situations and diabetes mellitus

Clinical situations that fall between normal and DM are not classified within the classification of DM itself but as intermediate states within the natural history changes in carbohydrate metabolism. In general, they are recognized as risk situations for the development of DM and cardiovascular disease.<sup>12</sup> The fact that the category «fasting glycemia change» (FGC) has recently been created does not permit complete certainty regarding the developmental characteristics of subjects with FGC.<sup>13</sup> Within this group, 2 entities are recognized (Table 1):

TABLA 1. Diagnostic values of diabetes mellitus and other categories of hyperglycemia

	Fasting plasma glycemia (mmol/L [mg/dL])	2 hours after 75 mg overload (mmol/L [mg/dL])	Cut-off points for plasma glycemia with the risk of illness
DM	$\geq 7.0$ [126]	$\geq 11.1$ [200]	Retinopathy, nephropathy, neuropathy, CVD
DGT	$< 7.0$ [126]	7.8-11.0 [140-199]	Diabetes and CVD
FGC	6.1-6.9 [110-125]	—	Diabetes and CVD (not well studied)

DM indicates diabetes mellitus; DGT, decreased glucose tolerance; FGC, fasting glycemia change; CVD, cardiovascular disease.

1. Diminished glucose tolerance (DGT) is defined as the result of a GTT that shows a plasma glycemia at 2 hours of  $\geq 140$  and  $< 200$  mg/dL. The GTT defines as normal glucose tolerance a plasma glycemia at 2 hours of  $< 140$  mg/dL.
2. Per 1997 ADA recommendations, the category of FGC was introduced as a clinical situation in which the FPG is  $\geq 110$  and  $< 126$  mg/dL. A normal FGC would be  $< 110$  mg/dL.

Since the introduction of this new category (FGC), much has been written on the supposed concordance between FGC and DGT, and there are an increasing number of studies demonstrating that these are not equivalent entities as far as their transcendence and prognosis are concerned.<sup>11</sup> It is clear GTT response of subjects with FGC is heterogeneous (normal, DGT, and DM). It seems that an elevated percentage of individuals with FGC



have a concomitant DGT, but also that many subjects, in spite of normal glycemia ( $<100\text{mg/dL}$ ) may also present with DGT and, therefore, an increased risk of DM.<sup>14,15</sup> In summary, while the diagnostic guidelines continue to use glycemic thresholds associated with an increased risk of developing microvascular disease when defining DM, the greatest mortality-morbidity of this affliction is associated with macrovascular disease and its complications. In general, there is a current consensus that determined by glycemia a GTT is a better indicator of the risk of cardiovascular disease and that, therefore, performing only a fasting metabolic evaluation may not be sufficient.

### **Diagnosis of gestational diabetes**

Gestational diabetes (GD) is defined as all alterations in carbohydrate metabolism that are diagnosed for the first time during pregnancy. The diagnostic criteria have changed over the years and today there are various recommendations for the application of same. The Spanish diabetes and pregnancy group in 2000 adopted criteria similar to those promoted by the ADA. These criteria establish the performance of a screening test (O'Sullivan test with 50 g of glucose independent of the presence or absence of a prior period of fasting), which consists of the evaluation of glycemia upon administration of 50 g of oral glucose. The test is considered positive when plasma glucose is  $\geq 140\text{ mg/dL}$ . This test must be performed universally in the second trimester (24-28 weeks) of every pregnancy and in the first trimester if risk factors exist such as a history of fetal macrosomy, polyhydramnios, familial history of DM, previous GD, DGT, obesity, or in women  $\geq 35$  years of age. A diagnosis of GD would be confirmed by a GTT with 100 g of oral glucose (blood draw for glycemia at 0, 1, 2, and 3 hours). The test is considered positive if 2 values are  $\geq$  a 0h=105, 1h=190, 2h=165 and 3h=145 mg/dL.

There is a less-used diagnostic guideline (WHO) that does not include screening and is based on performing a GTT with 75 g of oral glucose during the 24th and 28th weeks of gestation, with blood draw for glycemia at 0 and 2 hours and values based on the GTT values given above for the diagnosis of DM or DGT in the general population (glycemia  $\geq 126$  or glycemia at 2 hours  $\geq 140\text{ mg/dL}$ ).<sup>9</sup> Taking into account that GD constitutes a risk for the later development of DM, it is also advisable, that patients with a previous history of GD undergo a glucose tolerance evaluation after pregnancy has been completed with a GTT with 75 g of glucose.

### **Causes Type 1 Diabetes**

Type 1 diabetes occurs when your immune system, the body's system for fighting infection, attacks and destroys the insulin-producing beta cells of the pancreas. Scientists think type 1 diabetes is caused by genes and environmental factors, such as viruses, that might trigger the disease.

### **Causes type 2 diabetes**

Type 2 diabetes—the most common form of diabetes—is caused by several factors, including lifestyle factors and genes.

### **Overweight, obesity, and physical inactivity**

Extra weight sometimes causes insulin resistance and is common in people with type 2 diabetes. The location of body fat also makes a

difference. Extra belly fat is linked to insulin resistance, type 2 diabetes, and heart and blood vessel disease. To see if your weight puts you at risk for type 2 diabetes, check out these Body Mass Index (BMI) charts.

### **Insulin resistance**

Type 2 diabetes usually begins with insulin resistance, a condition in which muscle, liver, and fat cells do not use insulin well. As a result, your body needs more insulin to help glucose enter cells. At first, the pancreas makes more insulin to keep up with the added demand. Over time, the pancreas can't make enough insulin, and blood glucose levels rise.

### **Genes and family history**

As in type 1 diabetes, certain genes may make you more likely to develop type 2 diabetes. The disease tends to run in families and occurs more often in these racial/ethnic groups:

- African Americans
- Alaska Natives
- American Indians
- Asian Americans
- Hispanics/Latinos
- Native Hawaiians
- Pacific Islanders

Genes also can increase the risk of type 2 diabetes by increasing a person's tendency to become overweight or obese.

### **Causes gestational diabetes**

Scientists believe gestational diabetes, a type of diabetes that develops during pregnancy, is caused by the hormonal changes of pregnancy along with genetic and lifestyle factors.

### **Insulin resistance**

Hormones produced by the placenta contribute to insulin resistance, which occurs in all women during late pregnancy. Most pregnant women can produce enough insulin to overcome insulin resistance, but some cannot. Gestational diabetes occurs when the pancreas can't make enough insulin.

As with type 2 diabetes, extra weight is linked to gestational diabetes. Women who are overweight or obese may already have insulin resistance when they become pregnant. Gaining too much weight during pregnancy may also be a factor.

Hormonal changes, extra weight, and family history can contribute to gestational diabetes.

### **Genes and family history**

Having a family history of diabetes makes it more likely that a woman will develop gestational diabetes, which suggests that genes play a role. Genes may also explain why the disorder occurs more often in African Americans, American Indians, Asians, and Hispanics/Latinas.

### **Other cause of diabetes**

Genetic mutations, other diseases, damage to the pancreas, and certain medicines may also cause diabetes.

### Genetic mutations

- **Monogenic diabetes** is caused by mutations, or changes, in a single gene. These changes are usually passed through families, but sometimes the gene mutation happens on its own. Most of these gene mutations cause diabetes by making the pancreas less able to make insulin. The most common types of monogenic diabetes are neonatal diabetes and maturity-onset diabetes of the young (MODY). Neonatal diabetes occurs in the first 6 months of life. Doctors usually diagnose MODY during adolescence or early adulthood, but sometimes the disease is not diagnosed until later in life.
- **Cystic fibrosis** produces thick mucus that causes scarring in the pancreas. This scarring can prevent the pancreas from making enough insulin.
- **Hemochromatosis** causes the body to store too much iron. If the disease is not treated, iron can build up in and damage the pancreas and other organs.

### Hormonal diseases

Some hormonal diseases cause the body to produce too much of certain hormones, which sometimes cause insulin resistance and diabetes.

- Cushing's syndrome occurs when the body produces too much cortisol—often called the “stress hormone.”
- Acromegaly occurs when the body produces too much growth hormone.
- Hyperthyroidism occurs when the thyroid gland produces too much thyroid hormone.

### Damage to or removal of the pancreas

Pancreatitis, pancreatic cancer, and trauma can all harm the beta cells or make them less able to produce insulin, resulting in diabetes. If the damaged pancreas is removed, diabetes will occur due to the loss of the beta cells.

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## 6.3. MANAGEMENT OF DIABETES

The main goal of diabetes management is, as far as possible, to restore carbohydrate metabolism to a normal state. To achieve this goal, individuals with an absolute deficiency of insulin require insulin replacement therapy, which is given through injections or an insulin pump. Insulin resistance, in contrast, can be corrected by dietary modifications and exercise. Other goals of diabetes management are to prevent or treat the many complications that can result from the disease itself and from its treatment.

### Dietary management in diabetes

The primary objectives of dietary intervention are essentially the same for both type 1 and type 2 diabetes.

- Achieve and maintain blood glucose and blood pressure levels in the normal range, or as close to the normal range as safely possible, and achieve and maintain a lipid and lipoprotein profile to reduce cardiovascular disease risk
- Achieve and maintain a healthy body weight
- Prevent, or at least slow, the development of the complications of diabetes

- Consider personal and cultural food preferences and an individual's willingness to change
- Maintain the pleasure of eating by only limiting food choices when indicated by scientific evidence.

### **Management in type 1 diabetes**

Dietetic advice is required at the initial diagnosis of type 1 diabetes, with follow up 2–4 weeks later and regular (at least annual) review thereafter. The nutritional management of children with type 1 diabetes focuses on providing adequate energy for growth and development, and may initially require additional energy intake to compensate for weight loss before diagnosis. Appetite and activity levels change as children and adolescents grow into adulthood, and dietetic advice needs to be modified accordingly. The recommended meal plan should consider usual appetite, food intake patterns, level of exercise and insulin regimen. Recommendations are based on healthy eating principles of three balanced meals per day, healthy snacks, and regular physical activity. A key aspect of MNT is advice on carbohydrate amount, type and distribution over the day, taking into account the age of the individual and their insulin regimen. When using intensive insulin therapy regimens, education about carbohydrate quantification is essential to allow adjustments in insulin dose. In clinical practice, a number of methods for carbohydrate quantification are commonly taught, including 1 g increments, 10 g portions, and 15 g exchanges.

Recent studies have demonstrated that carbohydrate counting is difficult and repeated age appropriate education by experienced health professionals is necessary to maintain accuracy in estimations. Although intensive regimens increase flexibility in food intake, regularity in meal routines and monitoring blood glucose levels at least four times daily remain important for optimal glycaemic control. Dietary advice for all people with type 1 diabetes should include education regarding the glycaemic index (GI). Low GI foods are encouraged as these foods minimize the postprandial glycaemic excursion and improve long term glycaemic outcome. Low GI foods (such as some wholegrain breads, most pasta, legumes, temperate climate fruits, milk and yoghurt) cause a gradual sustained rise in postprandial blood glucose levels and improved long term glycaemic control compared to high GI foods that produce dramatic fluctuations in postprandial blood glucose levels. Low GI foods should be incorporated at all meals and snacks and used instead of high GI foods where practical. When dealing with children, it is important to involve the whole family in making dietary changes. Advice should focus on decreasing the intake of sweetened drinks and saturated fat while increasing the intake of wholegrain breads and cereals (preferably with a low GI), fruit, vegetables and low fat dairy products (except children less than 2 years of age who require regular fat dairy foods). 'Diabetic foods' are not recommended (other than diet soft drinks) because they are not necessary, expensive, often high in fat, and may contain sweeteners with laxative effects. Maintenance of an appropriate body weight is a key strategy of care for people with type 1 diabetes.

Additional contributing factors to excessive weight gain may be:

- Over-insulinisation

- Snacking to match insulin peaks, and
- Excess energy intake to avoid or treat hypoglycaemia.

Guidance on appropriate food quantities for treatment of hypoglycaemia and food/insulin adjustment for exercise can be provided by an Accredited Practising Dietitian (APD). Disordered eating and coeliac disease are more common in individuals with type 1 diabetes than in their non-diabetic peers. These conditions require extra education and dietary intervention with more frequent dietetic review, and should be referred accordingly

### **Management in type 2 diabetes**

People with type 2 diabetes require dietetic advice at diagnosis (preferably within 1 month), a follow up visit 3 months after initial dietary intervention, and should receive ongoing MNT every 6–12 months.<sup>15</sup> Due to the high prevalence of overweight and obesity in this group, and its primary role in the aetiology of the condition, weight loss of 5–10% of initial body weight at diagnosis is a primary objective, along with management of hyperglycaemia, hyperlipidaemia and/or hypertension. This can be achieved through a diet in which energy intake is balanced with regular physical activity, and one that is low in saturated fat and sodium and high in fibre and low GI carbohydrates. It is worth noting that reducing energy intake, regardless of dietary composition, and regular dietary counselling and support are the most likely predictors of successful weight loss. It is important to limit the intake of saturated fat and avoid trans fats to assist with weight management, improve insulin sensitivity, and reduce blood lipids to decrease overall cardiovascular disease risk.

Carbohydrate intake should be spread out evenly over the day to assist with blood glucose management. For patients taking insulin and some types of oral medications, carbohydrate intake should be matched with the action of their medication. Carbohydrate should come mainly from fibre rich fruits, vegetables, whole grains and legumes, as well as low fat dairy products (milk and yoghurt), preferably with a low GI. It is generally advisable for people with diabetes to avoid high protein diets due to possible negative effects on kidney function and a lack of evidence for long term benefits. Alcohol should be limited to no more than two standard drinks per day. Regular physical activity should also accompany dietary changes and ideally should include at least 150 minutes per week of moderate intensity aerobic exercise and resistance training three times per week unless there are contraindications.

#### **Check Your Progress**

1. What is diabetes mellitus?
2. How diabetes does was diagnosed and discuss the dietary management?

## **6.4. ARTIFICIAL SWEETENERS**

### **Introduction**

The sensory properties of food is highly influenced by the sensory properties like taste smell texture and appearance. The selection and consumption of food in man play a crucial role in the regulation of human appetite and nutrient intake. A sweetener is a food additive, which mimics

the effect of sugar on taste. Therefore, they are called sugar substitutes. Consumers often select those foods, which are composed of low calorie sweetener because they want the taste of sweetness without added calories. The dietary option that such product provides may be especially helpful in the management of obesity or diabetes mellitus. One group of such sweeteners consists of substances with a very intense sweet taste and is used in small amount to replace the sweetness of a much higher amount of sugar. The sweeteners of this type currently approved for use in the United States are- Aspartame, Acesulfane-K, Neotame, Saccharin, Sucralose, Cyclamate and Alitame. Table 1 summarizes some information about high intensity sweeteners.

### **Aspartame**

Aspartame was discovered in 1965 by James Schlatter a chemist. It is an artificial, non-saccharide sweetener, L-aspartyl-L phenylalanine methyl ester that is a methyl ester of the dipeptide of the amino acids aspartic acid and phenylalanine. Under strongly acidic or alkaline conditions, aspartame may generate methanol by hydrolysis. Under more severe conditions, the peptide bonds are also hydrolyzed, resulting in the free amino acids. It is slightly soluble in water, (about 3gm per 100ml, pH 3 at room temp.). The solubility increases with higher or lower pH as well as with increased temperature. In aqueous solution the relationship between pH and stability of aspartame is a bell-shaped curve with the maximum stability at pH 4.3. This sweetener is marketed under a number of trademark names including Equal, Nutrasweet, and Candere and has a good clean sweet taste but its time-intensity profile differs from sucrose.

**Synthesis** Chemical synthesis of aspartame involves the coupling of the two amino acid units having appropriate functional group protection with conventional synthetic reagents. The two major processes are known as the Z- and F- processes named after the protecting group used on the aspartyl group. Both of these processes produce some  $\beta$ -coupled products together with the desired  $\alpha$ -aspartame. The Z-process mainly involves the dehydration of the benzyloxycarbonyl-L-aspartic acid with acetic anhydride. The anhydride is then coupled with the methyl ester of L-phenylalanine in toluene to give a mixture of benzyloxy carbonyl  $\alpha$ - and  $\beta$  aspartames. The protecting groups are removed by hydrogenolysis and resulting mixture of aspartame isomer yield aspartame upon crystallization. The F-process involves the protection of the amino group of aspartic acid with a formyl group and concomitant dehydration to form anhydride. The anhydride is then coupled either with L-phenylalanine or its methyl ester and the formyl group removed by acid hydrolysis. The resultant mixture of  $\alpha$  and  $\beta$  products are subjected to the esterification conditions of aqueous methanol and preferentially crystallizes out from this mixture and is then neutralized to yield aspartame. The application of biotechnology and biocatalysts towards the synthesis of the aspartame has been extensively explored. Due to the presence of its dipeptide structure many variations of reverse proteolysis that employ both kinetically and thermodynamically controlled approaches has been investigated with different enzymes and under various reaction condition. Two Japanese companies have reported one formation route to produce aspartame directly by incubating microorganisms with L- aspartic acid and methyl ester of phenylalanine.

### **Metabolism and health aspect**

Aspartame is a low calorie sweetener used to sweeten a variety of low and reduced calorie foods and beverages including low calorie tabletop sweetener as well as for use in gum, breakfast cereal and other dry products. Aspartame provides energy of 4 calories per gram. Aspartame is unstable if subjected to prolong heating and therefore cannot be used in baking or cooking. It also decomposes in liquids during storage.

Upon ingestion, aspartame breaks down into natural residual components, including aspartic acid, phenylalanine, methanol and further break down products including formaldehyde, formic acid and diketopiperazine. Each of which then metabolized just as it would be if derived from other dietary sources and are safe as consumed in normal diets. Aspartame has been the subject of controversy regarding its safety since its initial approval by the U.S. Food and Drug Administration (FDA) in 1974. High level of the naturally occurring essential amino acid phenylalanine is a health hazard to those born with phenylketonuria (PKU) a rare inherited disease. So the phenylalanine level statement or aspartame—sweeten products is for their benefit and has no relevance for general population. Various scientific researches concluded that the effects of aspartame are likely to be attributable to methanol or its metabolites, evidence indicating that fruits and vegetables also contain high level of methanol than aspartame sweetened food and beverage do. But high intake of fruits and vegetables are associated with decrease rather than increase in cancer risk (Heber 2004). Carcinogenicity studies of aspartame were conducted by Nalt Toxicological Programme (NTP) in 2 strains of transgenic mice, and it was concluded that aspartame exposure was associated with increase in cancer in either male or female mice (NTP 2005). Based on government research reviews and recommendations from advisory bodies such as European Commissions Scientific Committee on Food and joint FAO/WHO expert committee on food additives, aspartame has been found to be safe for human consumption by more than ninety countries worldwide (Magnuson et al. 2007).

### **Acesulfame—k**

Acesulfame—k has been developed as sweetener by Hoechst. This high intensity sweetener is potassium salt of 6-methyl-123-axathiazine-4 (3H)-one 2,2-dioxide with molecular formula  $C_4H_4KNO_4S$  and molecular weight of 201.24. It is a white crystalline powder, approximately 120 times sweeter than sucrose and has high water solubility. Acesulfame—k is heat stable, so can be used in cooking and baking. It may have a bitter after taste when used alone to sweeten food or beverage. Ace-k is often blended with other sweetener (usually sucralose or aspartame) whereby each sweetener masks the other's after taste and exhibit a synergistic effects by which the blend is sweeter than its components.

### **Synthesis**

Early methods for Ace-k synthesis used chlorosulfonyl or fluoro sulfonyl isocyanate with propyne acetone and with other chemicals give N-chloro or N- (fluoro-sulfonyl) acetoacetamide, which is then cyclized by metabolic potassium hydroxide to give Ace-k. Alternative method involves the treatment of acetoacetamide with at least two equivalents of sulfur trioxide. This results in formation of N-sulfoacetoacetamide, which is then

dehydrated by sulfur trioxide to form oxathiazinone dioxide. Neutralization with potassium hydroxide gives Ace-k..

### **Metabolism and health aspect**

Acesulfame—k is not metabolized in the human body, thus it provides no calories and does not influence potassium intake despite its potassium content. In 1988 USFDA approved the use of Ace-k in a variety of dry food products and in alcoholic beverages. In 2003 the agency approved its use as a general-purpose sweetener. One breakdown product of ace-k is acetoacetamide known to be toxic if consumed in very large doses because human exposure to this breakdown product would be negligible. The USFDA concluded that no further testing of it was necessary.

### **Sucralose**

Sucralose was discovered in 1976. This nonnutritive sweetener is made from sucrose by a process that substitutes 3 chloride atoms for 3 hydroxyl groups on the sucrose molecule (FDA 2006). Sucralose is 450–650 times sweeter than sucrose, has a pleasant sweet taste and its quality and time intensity profile is very close to that of sucrose. It has a moderate synergy with other nutritive and non-nutritive sweeteners. It is very much soluble in water and is stable over a wide range of pH and temperature. It does liberate HCl when stored at high temperature and produce some kind of discoloration.

### **Synthesis**

The synthesis of sucralose involves a series of selective protection and deprotection steps so that the 4-hydroxyl group can be converted to a chloro atom with inversion of configuration. Treatment of the free hydroxyl groups with sulfuryl chloride produce trichlorodisaccharide which is then deprotected to give the sucralose. The use of enzymes or microbial cultures to augment synthetic organic chemistry and carry out selected functionalization of complex molecule has been widely documented in the growing field of biocatalysis.

### **Metabolism and health aspect**

Although sucralose is made from sugar, the human body does not recognize it as a sugar and does not metabolize it therefore it provides no calories. The bulk of sucralose ingested does not leave the gastrointestinal tract and is directly excreted in the feces while 11–27% of it is absorbed (Knight 1993). The amount that is absorbed from the gastro intestinal tract is largely removed from the blood stream by the kidneys and eliminated in the urine. As it is an organo chloride and some of which are known to have significant toxicity but sucralose is not known to be toxic. In addition sucralose does not breakdown or dechlorinate. In determining the safety of sucralose, the FDA reviewed data from more than 110 studies in human and animals. Many of the studies were designed to identify possible toxic effects including carcinogenic reproductive and neurological effects but no such effects were found. Food and Drug Administration (FDA) approval is based on the findings that sucralose is safe for human consumption. U.S. Food and Drug Administration (USFDA) approved sucralose as a generalpurpose sweetener. The acceptable daily intake (ADI) for sucralose



in US is 5mg/kg body weight/day. The estimated daily intake for percentile consumers as calculated by USFDA is 1.6mg/kg body weight/day.

### **Saccharin**

Saccharin was discovered by Remson and Fahlberg in 1878 at the Johns Hopkins University, Baltimore. It is a non-nutritive sweetener of 1,2-benzisothiazol-3-(2H) on 1,1 dioxide. Saccharin has an unpleasant bitter or metallic off taste. As the parent compound is only sparingly soluble in water, the sweetener is usually used as the sodium or calcium salt. Both salts are highly water soluble, 0.67 gms/ml of water at room temperature (Priebem and Kauffman 1980). It is about 300 times sweeter than sucrose.

### **Synthesis**

Chemical synthesis of saccharin involves the oxidation of the o-toluenesulfonamide with variety of agents like potassium permanganate, chromic acid, electrochemically etc. to the corresponding carboxylic acid. The ortho isomer is dehydrated to give the sweetener. Another process involves diazotization of methyl anthranilate and then treatment of the diazonium salt with sulfur dioxide and chloride gas to give the sulfonyl chloride which is then treated with ammonia to give saccharin.

### **Metabolism and health aspect**

The FDA tried to ban saccharin in 1977 because animal studies had showed that it caused cancer in rat (mainly bladder cancer). Many studies have since been performed on saccharin. No study has ever shown a clear casual relationship between saccharin consumption and health risks in human at normal doses. Though some studies have shown a correlation between consumption and cancer incidence. Saccharin is currently permitted for use under an interim regulation that specifies the amounts of saccharin permitted in beverages, processed food, and sugar substitute and requires that the product level must state saccharin in the ingredient declaration and specify the amount used.

### **Cyclamate**

Cyclamate was discovered in 1937. It was used as a low calorie sweetener in the United States in 1950s and 1960s. It is a salt of cyclohexylsulfamic acid. Sodium cyclamate is used as non-nutritive sweetener and the analogous calcium salt used specially in low sodium diets. Cyclamate is 30 times sweeter than sucrose. It has a bitter off taste, but has good sweetness synergy with saccharin. It is soluble in water and its solubility can be increased by preparing the sodium or calcium salt.

### **Synthesis**

This process begins with the trisaccharide raffinose followed by chemical chlorination to form tetrachloro raffinose TCR. This TCR is then enzymatically treated with a galactosidase to move the 6-chloro-6-deoxygalactosyl moieties from the 6th position to yield cyclamate (Bennett et al. 1992). There are another two methods available for synthesis of saccharin like bioorganic synthesis (Drasar et al. 1972) and regioselective deacylation.

### **Metabolism and health aspect**

Cyclamate itself shows very low toxicity but is metabolized by the gut bacteria to cyclohexylamine which shows greater toxicity (Bopp et al. 1986) because of the nature of cyclamate metabolism. It would be

inappropriate to assume that the total daily intake of cyclamate is metabolized to cyclohexylamine. The acceptable daily intake (ADI) for cyclamate was calculated by both the scientific committee of food (SCF) and the joint expert committee on food additives (JECFA) based on the “no observed adverse effect level” (NOAEL). For cyclohexylamine in rats assuming that 18.9% of the daily intake of cyclamate is metabolized to cyclohexylamine each day (SCF 2000). The plasma concentrations of cyclohexylamine following cyclamate intake will depend on both the extent of metabolism by the intestinal flora and the extent of elimination of cyclohexylamine from the circulation.

Scientific research on cyclamate is continuing. Recent studies have provided new data on the extent to which individuals convert cyclamate to cyclohexylamine during long term consumption (Renwick et al. 2004). This study gives first true indication of possible exposure to cyclohexylamine from cyclamate metabolism in humans over a period that is toxicologically relevant to the establishment of ADI for cyclamate.

### **Neotame**

Neotame is a derivative of a dipeptide compound of the amino acids - aspartic acid and phenylalanine. Neotame has been developed as a sweetener with a high degree of sweetness and is obtained by N-alkylating aspartame. Its degree of sweetness varies according to the kind of food and blend composition. It is 7000 to 13,000 times and about 30 to 60 times sweeter than sugar and aspartame respectively (Prakash et al. 2002). Neotame is an odorless white to gray-white powder with a strong sweetness and is readily soluble in alcohols and slightly soluble in water. The 0.5% aqueous solution of neotame is weakly acidic (pH 5.8) (Prakash et al. 2002).

### **Synthesis**

A chemoenzymatic method used for preparing N-[N-(3-3dimethylbutyl)-L-aspartyl]-L-phenylamine 1- methyl ester via the enzymatic regioselective hydrolysis of neotame ester using lipases or esterases (Prakash and Zhao 2001). Another method involve the hydrogenation of L- $\alpha$ aspartyl -L- phenylalanine I methyl ester and 3-3 dimethylbutyraldehyde produced insitu by the hydrolysis or cleavage of a 3-3-dimethylbutyraldehyde precursor (Prakash 2007).

### **Metabolism and health aspect**

Neotame is rapidly metabolized, completely eliminated and does not accumulate in the body. The major metabolic pathway of neotame is hydrolysis of the methyl ester by esterase which is present throughout the body. This yields deesterified neotame, the major metabolite and a significant amount of methanol. Due to the presence of the 3-3-dimethylbutyl group, peptidases which would typically break the peptide bond between the aspartic acid and phenylalanine moieties are essentially blocked, thus reducing the availability of phenylalanine. The amount of methanol derived from neotame is exceedingly small (Neotame 2002). Neotame was approved by the USFDA as a general purpose sweetener in July 2002 (USFDA 2002). It has also been favorably evaluated by JECFA (JFECFA 2004) which established an ADI of 2mg/kg body weight/ day. The ADI for neotame in the US is 18 mg/person/day (USFDA 2002).

### Alitame

Alitame (Fig. 1g) is an intense sweetener with sweetness potency 200 times greater than that of sucrose. It is a dipeptide of L-aspartic acid and D-alanine with a terminal N-substituted tetra methylthietanyl-amine moiety.

#### Synthesis

Alitame is prepared by a multi step synthesis involving the reaction between two intermediates (S)-[2-5-dioxo-(4- thiazolidine)] acetic acid and (R) -2- amino-N-(2,2,4,4- tetramethyl-3-thietanyl) propanamides. The final product is isolated and purified by crystallization of an alitame -4-methylbenzenesulfonic acid adduct followed by additional purification steps and finally recrystallization from water.

#### Metabolism and health aspect

Alitame is readily absorbed in the GI tract and then rapidly metabolized and excreted. It has two main components, aspartic acid and alanine amide. The aspartic acid component is metabolized normally and the alanine amide passes through the body with minimal metabolic changes. In humans the glucuronic derivative of D-alanine tetramethylthietane amide is the major urinary metabolite. JEFCA reviewed safety data on alitame in 2002. The committee concluded that there was no evidence that alitame is carcinogenic. An ADI of 0–1 mg/kg body weight was allocated on the basis of the NOAEL of 100mg/kg body weight/day to an 18 month study in dogs. Alitame has already been approved in Mexico, Colombia and China as well as Australia and New Zealand (Kroger et al. 2006). Rare sugar Rare sugars, which are defined as monosaccharides and their derivatives that are rare in nature (Izumori 2002) have recently attracted a great deal of attention mainly concerning their application. This could provide an alternative to the other sweetener due to its lack of calories. Rare sugars are either not metabolized by the body or metabolized to a lesser extent than natural sugar. Due to these features, rare sugars are well tolerated by diabetes patients. Other advantage of rare sugar is the absence of any objectionable after taste.

#### D-psicose

D-psicose (Fig. 1h) (D-ribo-2 hexulose), a C-3 epimer of Dfructose is a rare sugar present in small quantities in commercial mixtures of D-glucose and D-fructose obtained from the hydrolysis of sucrose or isomerization of Dglucose (Green and Perlin 1968). D-psicose has 70% of the sweetness of sucrose and has a higher solubility that makes it easy to use for food processing. It has been reported that the addition of D-psicose in food products improve the gelling behavior and flavor as well as it increases the antioxidant property of the food products (Sun et al. 2006; Sun et al. 2007). Furthermore, food products containing D-psicose maintain a high level of antioxidant effect over a long period of storage, which is able to delay the onset of lipid auto-oxidation and extend the food storage time (Sun et al. 2008). It gives proper sweetness, smooth texture, desirable mouthfeel and great self-stability to food products. **Synthesis**

D-psicose has previously been produced by chemical methods from D-fructose using catalytic action of molybdate ions in an acidic aqueous solution (Bilik and Tihlarik 1973) it is also sometimes prepared by boiling D-fructose in ethanol and triethylamine (Doner 1979). All the chemical

methods are insufficient in terms of large-scale production. An improved process for the mass production of D-psicose was developed using D-tagatose-3-epimerase bioreactor. D-fructose solution (60%, pH 7.0) was passed at 45°C through a column filled with immobilized D-tagatose-3-epimerase (D-TE) which was produced using recombinant E. coli. and 25% of the substrate was converted to D-psicose. After epimerization, the substrate D-fructose was removed by treatment with baker's yeast. The supernatant was concentrated to syrup by evaporation under vacuum and D-psicose was crystallized with ethanol (Takeshita et al. 2002). Another work was done for mass production of D-psicose using a non-characterized gene from *Agrobacterium tumefaciens* which increase the production 586 fold higher than that of D-TE. The enzyme is D-psicose-3-epimerase. This finding has considerable importance in D-psicose production.

### **Metabolism and health aspect**

An animal study on the suppression of increase in plasma glucose concentration with D-psicose found significant drop in plasma glucose concentration when maltose and sucrose were used as substrates, but no significant drop when glucose and soluble starch were used as substrate (Matsuo 2006). Another animal study proposed that D-psicose inhibited the hydrolysis of maltose with  $\alpha$ -glucosidase in rats (Matsuo and Izumori 2006). The doses of D-psicose at 5g (around 1/15 of carbohydrate intake) would be the minimum effective dose for suppressing the elevation of plasma glucose and insulin concentration for 75g maltodextrin. This study confirmed the improving effects of glucose tolerance. D-psicose is expected to serve as a food material with low glycemic index. Another study demonstrated that D-psicose inhibits intestinal sucrase and maltase activities in an uncompetitive manner and suppress the plasma glucose increase after sucrose and maltose ingestion. Thus D-psicose may be useful in preventing postprandial hyperglycemia in diabetic patient when food containing sucrose and maltose are ingested.

### **Xylitol**

Xylitol is a naturally occurring sugar. Xylitol is a five carbon sugar that tastes and looks exactly like sugar.

### **Synthesis**

The synthesis of xylitol from natural product is based on the pentosans occurring in many plants. Xylan, a constituent of pentosan, is a polysaccharide that can be hydrolyzed into D-xylose. Xylitol can be synthesized by hydrogenation of xylose. Xylitol also can be produced from D-glucose by three steps. Xylitol production from yeast is an alternative to chemical studies.

### **Metabolism and health aspect**

Xylitol metabolises easily and independently from insulin in humans and produces very low amount energy. Xylitol has a recognized glycemic index of 8 and have a caloric value of 2.4 calories/gm. (Sellman 2003) Xylitol is non-fermentable and therefore cannot be converted to acids by oral bacteria, thus it helps to restore a proper alkaline/acid balance in mouth. Several clinical trials have indicated that xylitol products (chewing gum) are more effective in reducing dental caries. In 1996, the joint expert committee on Food Additive (JECFA) confirmed the safety of xylitol for human consumption and allocated xylitol an ADI of 'Not

specified'. The scientific committee for food of the European Union (EU) also determined xylitol 'acceptable' for dietary uses.

### **Tagatose**

The ketohexose D-tagatose is structurally similar to D-fructose except for an inverted optically active center. Because of its excellent taste and bulk properties, combined with a possibly very low energy value, D-tagatose has potential for use as a sweetener.

### **Synthesis**

D-tagatose is produced from lactose in two-step. Firstly, lactose is converted to glucose and galactose by hydrolysis and then galactose is isomerized to D-tagatose by adding calcium hydroxide.

### **Metabolism and health aspect**

The metabolism of tagatose is identical as fructose but it is incompletely absorbed. The study on small-bowel absorption of tagatose concludes that 15g tagatose/day had a high apparent absorption in the small intestine of humans. The major part of ingested tagatose is fermented in the colon by indigenous microflora, resulting in the production of short chain fatty acid. The short chain fatty acids are absorbed almost completely and metabolized. Thus it can be concluded that D-tagatose a carbohydrate with physiological properties potentially valuable for the control of both body weight and symptoms of the metabolic syndrome as seen in diabetics. Substances such as glucose and especially fructose that promote lipogenesis and have high glycosylation indices could be replaced with D-tagatose with lower fat accumulation, lower glycosylation index (Bunn and Higgins 1994) and strong antidiabetic effects found in rats.

### **D-allose**

D-allose, a cis-aldohexose is a non caloric sweetening and bulking agent which have good antioxidant properties. The mass production of D-allose mainly achieved from D-psicose in a batch reaction by crude recombinant L-rhamnose isomerase cross linked with glutaraldehyde (Menavuvu et al. 2006). Studies on D-allose supplementation on Dahl salt sensitive hypertensive rats and spontaneously hypertensive rats suggests the possibility of D-allose supplementation for prevention of salt sensitive hypertension. D-allose has been reported to inhibit segmented neutrophil production and lower platelet count in vivo without other significant detrimental clinical effects in rats. D-allose is also used as potential inhibitor of various glycosides. Other sweetener Table 2 summarizes the information about some nutritive sweetener known as sugar alcohol. They are highly soluble and non hygroscopic. The sugar alcohols are non-reducing, temperature stable and more resistant to browning reactions than sucrose.

### **Erythritol and other polyols**

Erythritol is four carbon sugar alcohol (or polyol). It is manufactured by fermentation from glucose and sucrose by *Trichosporonoides megachiliensis*. It has a sweetness approximately 60–80% of sucrose. Polyols are low digestible carbohydrates which are poorly absorbed from the small intestine. These are also used for their humectant and bulking properties. Excessive consumption has a laxative effect due to unabsorbed polyol increasing the osmotic potential of the gut lumen and other gastrointestinal effect. Erythritol is considered to be of low toxicity.

It has been assessed by JECFA which assigned it an 'ADI not specified'. Studies with human have shown them that ingested doses of erythritol is absorbed from the small intestine and excreted in the urine unchanged (Munro et al. 1998). Another polyol like sorbitol show property close to sugar.

### **Trehalose**

Trehalose is a non-reducing disaccharide that consists of two glucose units linked by a 1,1-glycosidic bond. It is with a relative sweetness of 40–45% that of sucrose. Trehalose is produced directly from food-grade starch by a multienzymatic process. This disaccharide is enzymatically hydrolyzed by the enzyme trehalase in the small intestine in to two glucose subunits which are subsequently absorbed and metabolized in a manner similar to maltose. Present study reports that adding trehalose to dehydrated pear cubes could improve aroma retention by 15% (Komes et al. 2006). It has also added advantage of being an antioxidant. Several safety studies on trehalose have been evaluated by JECFA, 2001 and allocated an ADI of 'not specified'. Trehalose is approved in Japan, Korea, Taiwan, and UK.

### **Stevia rebaudiana**

Stevia is a natural herb. This zero calorie sweetener mainly containing steviol glycoside which is 10–15 times sweeter than sucrose. Human body does not metabolize these sweet glycosides, so obtains no-calories from stevia. Unlike artificial sweetener, the sweet glycoside does not break down in heat which makes stevia an excellent sweetener for cooking and baking. Studies have indicated that stevia tends to lower the elevated blood pressure. It also significantly improves nutritional status of diabetic patients.

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## **6.5. DIET AND INSULIN AND LIFESTYLE MANAGEMENT DIETARY CONSIDERATIONS**

### **CARBOHYDRATE**

People with type 1 diabetes, because they experience absolute insulin deficiency, must use insulin to control glucose excursions after meals. Since 1994, the American Diabetes Association (ADA) has recommended that, for patients with type 1 diabetes, 60–70% of total calories come from carbohydrate and monounsaturated fat. Although some studies have considered whether a preponderance of calories from unsaturated fat or carbohydrate may be more beneficial, there is no consensus on the relative amount of each. There are demonstrated improvements, however, from adjusting the doses of prandial rapid- or short-acting insulin based on the carbohydrate content of meals for patients using basal-bolus insulin regimens involving multiple daily injections or continuous subcutaneous insulin infusion. Similarly, patients on fixed doses of rapid- or short-acting insulin should attempt to keep the amount of carbohydrate relatively constant from meal to meal. Recommendations for carbohydrate consumption for people with type 2 diabetes are similar to those for patients with type 1 diabetes. Carbohydrate and monounsaturated fat should comprise 60–70% of total calories. However, there is some concern that increased unsaturated fat consumption may promote weight

gain in obese patients with type 2 diabetes and thereby decrease insulin sensitivity. Glycemic excursions appear to be similar between starches and sucrose (“table sugar”); therefore, sucrose does not need to be eliminated from the diet. The “glycemic index” is an attempt to compare the glycemic effects of various foods to a standard, such as white bread. Although several authors have proposed its clinical usefulness in controlling postprandial hyperglycemia, prospective studies have not demonstrated a clear improvement in hemoglobin A1c (A1C) in patients using low-glycemic-index diets. One cross-sectional study<sup>14</sup> suggested a relationship between low-glycemic-index diets and low A1C levels in patients with type 1 diabetes, but it is important to note that this study did not control for patients using once-daily, twice-daily, or more intensive insulin therapy regimens to control their glucose excursions. Another more recent meta-analysis of low-glycemic-index diets did suggest a mild but significant improvement in A1C levels. Therefore, there may exist a small benefit in pursuing a low-glycemic-index diet in patients with diabetes. This benefit, however, appears to be less than the benefit of either matching insulin doses to carbohydrate consumed or controlling carbohydrate consumed when using fixed insulin doses. Many sweeteners are available to the general public; perhaps the most common is sucrose. Studies comparing the impact of sucrose versus the impact of the same amount of starch on glycemic control have shown that their impact is essentially identical. As described above, sucrose should be adequately covered by rapid- or short-acting prandial insulin but does not need to be eliminated from the diet. Fructose may cause less postprandial hyperglycemia, but there is some evidence suggesting that it may also lead to or worsen hyperlipidemia. Therefore, the addition of fructose to the diet as a sweetening agent is not recommended by the ADA; foods that contain naturally occurring fructose, such as fruits, do not need to be avoided. The Federal Drug Administration (FDA) has approved several sugar alcohols for use as sweeteners. These include products such as sorbitol, a common sweetener in chewing gum. Sugar alcohols cause less hyperglycemia than naturally occurring sugars and may also decrease the risk of dental caries. They are only partially absorbed from the intestinal tract and therefore may lead to diarrhea or gastrointestinal discomfort, especially if consumed in higher amounts. They provide approximately half the calories of natural sugars and should be included in carbohydrate counting at half the impact of sucrose. Despite a lower risk of cavities, they have not been shown to facilitate weight loss or improve glycemic control. Several nonnutritive sweeteners are also available and do not affect blood glucose levels. These include aspartame, sucralose, saccharin, neotame, and acesulfame potassium. Although at one time linked to carcinogenesis in laboratory animals at extremely high doses, saccharin is no longer considered a cancer-causing chemical by the FDA. One of the most recently released sweeteners, sucralose, has been shown to have no significant effect on blood glucose levels, and therefore may be omitted from carbohydrate calculations. These sweeteners have not been shown to facilitate weight loss or improve glycemic control. Patients should exercise caution whenever introducing artificial sweeteners into the diet or decreasing their carbohydrate consumption. Making these changes

without adjustment in diabetes medications could cause hypoglycemia, especially in patients using insulin or insulin secretagogues.

### **Protein**

Although the majority of clinical focus on the management of diabetes is on carbohydrate metabolism, protein metabolism in the state of diabetes is also abnormal. Patients with type 2 diabetes exhibit a more negative nitrogen balance than individuals without diabetes. Protein degradation appears to be exacerbated by hyperglycemia and improved by controlling glucose levels with insulin therapy.<sup>19–21</sup> These studies suggest that the protein requirements for people with type 2 diabetes may be slightly greater than those for nondiabetic individuals. Patients with type 1 diabetes can and do convert amino acids into glucose depending on the level of insulinization; therefore, protein consumption may cause hyperglycemia.<sup>12</sup> Studies of patients with type 2 diabetes, however, have demonstrated that protein consumption does not increase plasma glucose concentrations and that endogenous insulin release is, in fact, stimulated by protein consumption. There may also be an association between high-protein diets and the risk of developing diabetic nephropathy. In a cross-sectional study of patients with type 1 diabetes, patients with macroalbuminuria were more likely than those with microalbuminuria or normal albumin excretion to report consuming > 20% of their calories in the form of protein. High-protein diets are not recommended.

### **Dietary Fat**

Recommendations regarding fat in the diet of people with diabetes are similar to those for patients with coronary artery disease. This is primarily because studies have shown that the risk of myocardial infarction in diabetic patients is similar to that of nondiabetic patients who have already suffered a myocardial infarction.<sup>24</sup> Because saturated fats are the major dietary determinants of serum LDL cholesterol levels, people with diabetes should strive to keep saturated fat consumption to < 7% of total daily calories and to minimize consumption of trans-fatty acids. Cholesterol consumption should be < 200 mg/day. When incorporated into a controlled-calorie diet in which individuals are not losing weight, programs that emphasize either carbohydrate or monounsaturated fats both lower cholesterol, but the higher-carbohydrate diets may exacerbate hyperglycemia. In diets in which total calories were reduced to facilitate weight loss, however, the hyperglycemic effect of the high-carbohydrate diet appeared mitigated. Mediterranean-style diets, which are high in polyunsaturated fats, have been associated with lower mortality in elderly Europeans, but this study was not specific to people with diabetes. Diets high in fish oil may decrease the risk of cardiovascular disease and all-cause mortality.<sup>26</sup> Plant sterols are plant esters that decrease intestinal absorption of both dietary and hepatobiliary cholesterol. They have been shown in prospective studies of diabetic patients to decrease LDL cholesterol. To avoid unnecessary weight gain, the ADA recommends that, if they are used in the diet to decrease cholesterol, they should replace cholesterol sources rather than simply be added. There has been a great deal of interest in using micronutrients such as chromium, zinc, antioxidants, and herbal supplements to improve diabetes control. Although some small studies have suggested a benefit from chromium,



other studies and meta-analysis have not reached the same conclusion. Currently, there are no large convincing studies that prove benefit of micronutrients in the management of diabetes. Considerable attention and marketing has been focused on the macronutrient content of diets. A recent study suggested that a diet low in carbohydrate and high in fat and protein may yield greater weight loss than other diets in nondiabetic patients. Similar diets studied in diabetic patients have also suggested that a low-carbohydrate diet may produce similar or superior weight loss than balanced diets. Changes in triglycerides may be more favorable in low-carbohydrate diets, and A1C levels may be lower in low-carbohydrate diets. Meta-analysis of several studies, however, suggested that low-carbohydrate diets may raise LDL levels. It is important to note that the existing studies of low-carbohydrate diets are short-term studies and that the longterm effects of such diets is unknown. This is especially concerning because of their widespread use and the association of diabetic kidney disease with diets consisting of > 20% of calories from protein. For these reasons, a low-carbohydrate diet (< 130 g of total carbohydrate per day) is not recommended by the ADA.

### **Exercise**

Patients with type 1 or type 2 diabetes have an increased risk of coronary artery disease. The ADA recommends that patients who plan to begin a moderate- to high-intensity exercise program undergo screening for cardiovascular disease if they are > 35 years of age. Patients who are > 25 years of age should also be screened if they have had type 2 diabetes for > 10 years or type 1 diabetes for > 15 years, have an additional risk factor for coronary disease, or have microvascular disease, peripheral vascular disease, or autonomic neuropathy. Decisions regarding screening of patients who plan low levels of physical activity, such as walking, are left to the discretion of the treating physician. Because some activities can lead to retinal hemorrhage or detached retina in the setting of proliferative retinopathy, patients with this condition should consult their ophthalmologist before beginning an exercise regimen. People with type 1 diabetes who begin an exercise regimen should tailor their exercise regimen to their specific condition. For instance, a patient with peripheral neuropathy must take precautions to avoid blisters and abrasions and check closely for such conditions after exercising. Patients should consider delaying exercise if their blood glucose is > 250 mg/dl and ketones are present or if their blood glucose level is > 300 mg/dl. They should monitor blood glucose before and after physical activity and be cautious about hypoglycemia, which can develop during or even several hours after exercise. They should have carbohydrate sources available and consume them as necessary to avoid hypoglycemia. Although studies have not demonstrated a clear benefit of aerobic exercise on A1C levels in type 1 diabetes, aerobic exercise is clearly beneficial in controlling other risk factors for cardiovascular disease.

Physical exercise is a key component of lifestyle modification that can help individuals prevent or control type 2 diabetes. Although diet is probably more important in the initial phases of weight loss, incorporating exercise as part of a weight-loss regimen helps maintain weight loss and

prevent weight regain. Mild to moderate activity levels have been associated with a lower risk of developing diabetes or pre-diabetes. Men with low degrees of cardiorespiratory fitness may have up to a 1.9-fold increased risk of developing impaired fasting glucose compared to men with high degrees of fitness. Patients should understand that the amount of exercise that produces a beneficial effect on health is not large; as little as 30 minutes of moderate physical activity daily may offer protection from diabetes.<sup>33,35</sup> As with the lowering of A1C levels, there is a gradient of benefit with higher levels of exercise and activity. Greater levels of physical activity are associated with lower risks of developing diabetes in women compared with lesser levels of activity. These studies indicate that exercise should be a mainstay of primary prevention of diabetes. In patients with type 2 diabetes, structured regimens of physical activity for 8 weeks or longer improved A1C independent of changes in body mass. There may also be further improvement in A1C with increasing intensity of exercise. Exercise in type 2 diabetes has not been associated with peripheral neuropathy or worsening of nonproliferative retinopathy. Physical activity may cause transient increases in urinary albumin excretion, but exercise has not been shown to increase the rate of progression of diabetic kidney disease. Resistance exercises may be incorporated into a weekly exercise regimen in the absence of contraindications. As further evidence regarding the benefit of exercise, in men with diabetes, the degree of physical fitness correlated with overall mortality, and this association was independent of BMI. When physicians are confronted with newly presenting patients with diabetes or glucose intolerance/impaired fasting glucose, one of the most vexing questions posed is to what extent to rely on exercise, weight loss, and dietary measures to control the disease. Evidence supports the contention that controlling blood glucose through modification of diet and lifestyle should be a mainstay of diabetes therapy. With many oral and injectable pharmaceutical agents to help patients control their glucose levels, it is easy for a practitioner to overlook or forget to emphasize and reinforce the importance of lifestyle modification on the treatment of diabetes. Despite being one of the most time-consuming discussions to have with patients, this is probably the most important patient-physician discussion in regard to diabetes control and prevention of disease progression and complications.

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**Diet tips to improve insulin resistance**

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Insulin is a hormone that helps the body absorb glucose and keeps blood sugar levels balanced. Insulin resistance makes it harder for the body's cells to take in glucose. However, some dietary measures can improve insulin resistance.

Insulin resistance is when the cells in the body do not absorb insulin properly. Over time, insulin resistance can cause a range of problems, including permanently high blood sugar levels and cell damage to organs, muscle, limbs, and eyes.

People with insulin resistance often receive a diagnosis of prediabetes, which might lead to type 2 diabetes. People who are insulin resistant may need extra checks to make sure they do not develop type 2 diabetes.

Certain diet and other lifestyle choices can increase the risks related to insulin resistance. Making dietary changes can improve insulin sensitivity and reduce insulin resistance and the risk of developing type 2 diabetes.

In this article, we look at the dietary and lifestyle changes a person can make to increase their body's sensitivity to insulin.

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### **Foods to eat**

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*A balanced diet can help people control their blood sugar levels.*

Western diets typically lack certain nutrients, such as magnesium, calcium, fiber, and potassium. These nutrients are essential for maintaining blood sugar levels. People with insulin resistance should seek out foods that contain plenty of these nutrients.

According to the American Diabetes Association, people with insulin resistance can eat from any food group. However, it is important to understand which foods increase blood sugar and which support insulin sensitivity.

The following foods help to support insulin sensitivity and reduce the risk of developing diabetes in general:

- non-starchy vegetables, such as broccoli, dark leafy greens, and peppers
- tomatoes, which are an excellent source of vitamins C and E
- citrus fruits, such as lemons, oranges, and limes
- high-fiber foods, including beans and lentils
- some whole grains, such as oats, quinoa, and barley
- protein-rich foods, including lean meats, fish, soy, legumes, and nuts
- fish with a high omega-3 fatty acid content, such as salmon, sardines, and herring
- foods that contain antioxidants, such as berries
- sweet potatoes, which have a lower GI than regular potatoes
- water, especially as a substitute for sweetened drinks
- unsweetened teas
- unsweetened yogurt

### **Foods to avoid**

Certain foods are more likely to raise blood sugar. Regularly eating foods with high sugar content can overload the body's ability to produce enough insulin. It can also limit the ability of cells to absorb the sugar. If the cells become saturated with too much blood sugar, or glucose, they will gradually respond less and less to insulin. When this happens, the glucose remains in the blood, contributing to the health problems that accompany consistently raised blood sugar, such as damage to the kidneys (nephropathy) or limbs (neuropathy).

Avoiding or significantly limiting the following foods can help moderate blood sugar level:

- sweetened beverages, including fruit juices, soda, and fountain drinks
- alcohol, particularly beer and grain alcohol, especially in large quantities

- starchy vegetables, such as potatoes and yams (especially without skin), pumpkin, corn
- processed snacks and boxed foods
- sugary sweets, such as cupcakes, ice cream, or chocolate bars
- refined grains, such as white bread, rice, pasta, and flour-based foods, which are lower in fiber than whole grain versions
- dairy from cows, especially milk
- fried foods, even if it is a type of food that would be less harmful cooked another way, such as vegetables
- foods high in saturated fats, including chocolate, butter, and salt pork

### **Finding a healthful balance**

However, people can still eat foods on this list occasionally without causing any long-term harm to their insulin sensitivity. The key is to limit these foods and replace them with more healthful options as often as possible. Sometimes, the occasional treat can help a person satisfy their sweet tooth and focus on adjusting their diet more regularly. By sticking to a high-fiber, plant-based diet that is low in added sugars, a person can steadily improve their insulin sensitivity. Daily exercise is also a significant factor. During activity, the muscles soak up glucose from the bloodstream and do not require insulin. Taking a walk after a meal and being active throughout the rest of the day can significantly improve blood sugar management. By losing 5–10 percent of their body weight, a person can also significantly improve insulin sensitivity.

These lifestyle changes can reduce the risk of type 2 diabetes, cardiovascular disease, and other health problems.

### **Life style management**

There are a number of things you can do to manage diabetes and any complications.

### **Important Steps in Managing Your Diabetes**

Healthy eating and exercise are very beneficial for people with diabetes. They can improve your overall health, help manage your blood glucose level, and decrease your risk of the complications of diabetes.

### **Choose low calorie foods**

To help manage your blood glucose levels, you need to control the number of calories you eat each day. But you don't have to give up taste and satisfaction! Here's how to eat well and healthfully when you have diabetes:

- Eat smaller portions at each meal. If you eat out, share what you order with a friend or bring home part of your meal to eat the next day.
- Eat cereals, breads, and pasta made with whole grains instead of white flour. Substitute brown rice for white rice and sweet potatoes for white potatoes.
- Read labels on foods such as cereal, bread, and pasta. Choose those containing at least 3 grams or more of fiber per serving.
- Eat a variety of brightly-colored, low calorie fruits and vegetables. Aim for 6 to 9 servings a day. Especially good choices include leafy greens (such as spinach, chard, kale, collards, mustard greens,

and dark green or red lettuces), broccoli, broccoli rabe, red peppers, carrots, berries, cherries, apples, pears, and citrus fruits.

- Drink water or unsweetened tea instead of fruit juices, soft drinks, or other beverages high in sugar.
- Avoid eating processed and prepared foods. These foods are often loaded with fat, calories, and sodium, plus they can contain unhealthy ingredients such as trans fats and high fructose corn syrup.
- At fast food restaurants, choose the salad option (with low-fat dressing). At family-style restaurants, choose broiled poultry, fish, or lean meat entrees with steamed veggies. Skip cheesy, buttery, or creamy sauces. Choose oil and vinegar dressings on salads.
- Limit the amount of alcohol you drink.
- Less than 20-35% of your total daily calories should come from fat. The healthiest fats are found in foods such as whole grains, avocados, walnuts, almonds, sunflower seeds, peanut butter, and fatty fish (such as salmon, sardines, mackerel, herring, and trout).
- Choose olive and canola oil for cooking and salads.
- Choose foods high in protein, including eggs, low-fat dairy products, lean meats, poultry, fish, beans, and nuts.
- Your diet should also be high in fiber and appropriate vitamins and minerals.
- Save desserts and other sweet treats for special occasions. Choose fruit canned in its own juice instead of sugar syrup.

### **Exercises**

Exercise is essential for losing weight and controlling your blood glucose. Even small increases in physical activity can help.

People with pre-diabetes or diabetes should try to exercise 20 to 30 minutes at least three times a week. If you are able to, there is even greater benefit in exercising 20 to 30 minutes on most days.

Activities such as doing yard work or cleaning the house count as exercise, as long as they increase your heart rate and cause you to sweat lightly. Walking, swimming, and water aerobics are also great forms of exercise.

Exercise can be more fun if you do it with a friend or a group. Exercise classes are available at many local hospitals, community and senior centres, and adult education programs. An exercise teacher can help guide you on how to prevent injuries and modify activities for any physical limitations you may have. You may check with your insurance plan if you are eligible for a Silver Sneakers program. This program provides people 65 and over with free access to local fitness centers.

### **Diabetes education and self management**

Medicare pays for a visit with a diabetes educator once every year. Ask your healthcare provider to give you a referral. The educator will teach you how manage your diabetes and will work with you to develop a self-management program.

It is important that you know everything you can about how to manage your diabetes. Therefore, you should feel comfortable asking the diabetes educator as many questions as you need to, as often as you need to. Make

sure you fully understand the answers to all your questions, and ask the educator to explain anything you do not understand.

If you need help, your family or other caregivers should also receive training and become involved in your diabetes self-management. Caregivers may need to take over the self-management program if an older person with diabetes begins to have mental difficulties or becomes significantly disabled.

### ***Recognize the symptoms***

#### ***Very High Blood Glucose***

This is an uncommon but serious complication of diabetes that occurs most often in older adults. A spike in glucose can be caused by a sudden illness, particularly an infection, or certain medications. Watch out for these symptoms:

- Physical weakness
- Lack of energy
- Agitation
- Confusion

#### ***Low Blood Glucose***

Low blood glucose usually occurs when you take too much of your diabetes medication, or if you skip a meal. Illness and infections can also cause low blood glucose. Watch for these symptoms:

- Nervousness
- Shaking
- Sweating
- Confusion

#### ***What to Do***

If your blood sugar tests low, you should be able to raise it quickly by having some sugar. You can eat a piece of candy, have a spoonful of honey, or drink a glass of fruit juice. That's why it's a good idea to keep hard candies handy, just in case. However, this sugar boost only lasts a little while. Therefore, it's important to eat a meal as soon as possible.

If your low blood glucose is frequent or severe, your healthcare provider will need to check your diabetes treatment plan and change it if necessary. You may just need a better plan for controlling your blood glucose levels. You may also need to visit your healthcare provider more frequently for check-ups, or see a diabetes care specialist.

#### ***Managing stress***

Extreme or chronic stress can raise blood sugar levels. Though you can't avoid stress, you can learn to manage your reaction to it.

#### ***These tips can help:***

- Don't put unnecessary demands on yourself. Try to avoid being a perfectionist or workaholic.
- Schedule time for things you enjoy, such as socializing with friends or family, gardening, singing or playing an instrument, or doing crafts or other creative activities.
- Keep a positive attitude and focus on the things in your life that are going well.
- Talk to your spouse or partner, a close friend, a family member, or a counselor or clergy member about the things you find stressful.
- Exercise. It is an excellent way to relieve stress.

- Learn and use simple relaxation techniques, such as listening to calming music or sounds, deep breathing, muscle relaxation, or meditation.

**Check Your Progress**

1. What are the complications of diabetes mellitus?
2. What is artificial sweeteners?

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## 6.4 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS

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The old and confusing terms of insulin-dependent (IDDM) or non-insulin-dependent (NIDDM) which were proposed by WHO in 1980 and 1985 have disappeared and the terms of new classification system identifies four types of diabetes mellitus: type 1, type 2, “other specific types” and gestational diabetes.

Gestational diabetes mellitus is an operational classification (rather than a pathophysiologic condition) identifying women who develop diabetes mellitus during gestation.

Hormones produced by the placenta contribute to insulin resistance, which occurs in all women during late pregnancy. Most pregnant women can produce enough insulin to overcome insulin resistance, but some cannot. Gestational diabetes occurs when the pancreas can't make enough insulin.

Recommendations are based on healthy eating principles of three balanced meals per day, healthy snacks, and regular physical activity. A key aspect of MNT is advice on carbohydrate amount, type and distribution over the day, taking into account the age of the individual and their insulin regimen (Table 1). When using intensive insulin therapy regimens, education about carbohydrate quantification is essential to allow adjustments in insulin dose. In clinical practice, a number of methods for carbohydrate quantification are commonly taught, including 1 g increments, 10 g portions, and 15 g exchanges.

A sweetener is a food additive, which mimics the effect of sugar on taste. Therefore, they are called sugar substitutes. Consumers often select those foods, which are composed of low calorie sweetener because they want the taste of sweetness without added calories. The dietary option that such product provides may be especially helpful in the management of obesity or diabetes mellitus.

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## 6.5 SUMMARY

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The enormous economic, social and personal cost of type 2 diabetes make a compelling case for prevention. In recent years, there has been much new evidence demonstrating the potentially preventable nature of diabetes,

particularly by the implementation of lifestyle measures such as weight control and exercise. In view of this and the devastating health impact of the disease it seems prudent that primary prevention should be a major priority.

There is convincing evidence for a decreased risk of diabetes in adults who are physically active and maintain a normal body mass index (BMI) throughout adulthood, and in overweight adults with impaired glucose tolerance who lose weight voluntarily. An increased risk for developing diabetes mellitus is associated with overweight and obesity; abdominal obesity; physical inactivity; and maternal diabetes. It is probable that a high intake of saturated fats and intrauterine growth retardation also contribute to an increased risk, while non-starch polysaccharides are likely to be associated with a decreased risk. From existing evidence it is also possible that omega3 fatty acids, low glycaemic index foods and exclusive breastfeeding may play a protective role, and that total fat intake and trans fatty acids may contribute to the risk. However, insufficient evidence is currently available to provide convincing proof.

Based on the strength of available evidence regarding diet and lifestyle in the prevention of type 2 diabetes, it is recommended that a normal weight status in the lower BMI range (BMI 21–23) and regular physical activity be maintained throughout adulthood; abdominal obesity be prevented; and saturated fat intake be less than 7% of the total energy intake.

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## 6.6 KEY WORDS

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- **MNT**  
: Medical Nutrition Therapy.
- **Insulin**  
**n:** a hormone produced in the pancreas by the islets of Langerhans, which regulates the amount of glucose in the blood. The lack of insulin causes a form of diabetes. An animal-derived or synthetic form of insulin used to treat diabetes.

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## 6.7 SELF ASSESSMENT QUESTIONS

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### Short Answer Questions

1. What is diabetes mellitus - Justify.
2. What are the classification, symptoms and causes of diabetes?
3. What is artificial sweeteners and list them?
4. Discuss the dietary management of diabetes.

### Long Answer Questions

1. Elaborate in details about diabetes mellitus.
2. Explain the artificial sweeteners used in the treatment of diabetes.
3. Elaborate the diet, insulin and life style management of diabetes



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## **6.8 FURTHER READINGS**

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- Srilakshmi.B, 2016, Nutrition Science, New Age International Publishers, New Delhi.
- Park K., 2019, Textbook of Preventive and Social –Medicine. 25<sup>th</sup> edn. Jabalpur: BanarsidasBhanot.
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# UNIT – VII DIABETES INSIPIDUS AND GESTATIONAL DIABETES

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## **Structure**

- 7.0 Introduction
- 7.1 Objectives
- 7.2 Diabetes Insipidus
- 7.3 Gestational Diabetes
  - 7.3.1 Lifestyle Management
- 7.4 Answers to check your Progress Questions
- 7.5 Summary
- 7.6 Key Words
- 7.7 Self-Assessment Questions and Answers
- 7.8 Further Readings

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## **7.0 INTRODUCTION**

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In the previous units, we have learnt about the stress, weight management, and diabetes mellitus. It is not sufficient if we are aware about the detrimental health covers. Diabetes Insipidus is a rare complication that occurs in 2 to 4 per 100,000 pregnancies. The diagnosis of gestational DI can be challenging because it usually presents with symptoms of polydipsia and polyuria, which are often attributed to normal pregnancy. This condition usually develops between the second to third trimesters of pregnancy and remits spontaneously 4 to 6 weeks after delivery. The most common etiology for gestational DI is excessive vasopressinase activity leading to ADH degradation. The objective of this review is to consider other pathophysiological mechanisms for the development of Gestational DI, its clinical presentation and to understand different tests for diagnosis. In this context, this unit will teach about what the diabetes insipidus and gestational diabetes.

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## **7.1 OBJECTIVES**

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After studying this unit you will be able to

- Define and describe about diabetes insipidus.
- Role of dietary and life style management of diabetes insipidus and gestational diabetes.

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## **7.2 DIABETES INSIPIDUS**

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Diabetes insipidus is a rare disorder that occurs when a person's kidneys pass an abnormally large volume of urine that is insipid—dilute and odorless. In most people, the kidneys pass about 1 to 2 quarts of urine a day. In people with diabetes insipidus, the kidneys can pass 3 to 20 quarts of urine a day. As a result, a person with diabetes insipidus may feel the need to drink large amounts of liquids.

Diabetes insipidus and diabetes mellitus—which includes both type 1 and type 2 diabetes—are unrelated, although both conditions cause frequent urination and constant thirst. Diabetes mellitus causes high blood glucose, or blood sugar, resulting from the body's inability to use blood glucose for energy. People with diabetes insipidus have normal blood glucose levels; however, their kidneys cannot balance fluid in the body.

### **Types of diabetes insipidus**

The types of diabetes insipidus include

- Central
- Nephrogenic
- Dipsogenic
- Gestational

Each type of diabetes insipidus has a different cause.

### **Central Diabetes Insipidus**

Central diabetes insipidus happens when damage to a person's hypothalamus or pituitary gland causes disruptions in the normal production, storage, and release of vasopressin. The disruption of vasopressin causes the kidneys to remove too much fluid from the body, leading to an increase in urination. Damage to the hypothalamus or pituitary gland can result from the following:

- Surgery
- Infection
- Inflammation
- A tumor
- Head injury

Central diabetes insipidus can also result from an inherited defect in the gene that produces vasopressin, although this cause is rare. In some cases, the cause is unknown.

### **Nephrogenic Diabetes Insipidus**

Nephrogenic diabetes insipidus occurs when the kidneys do not respond normally to vasopressin and continue to remove too much fluid from a person's bloodstream. Nephrogenic diabetes insipidus can result from inherited gene changes, or mutations, that prevent the kidneys from responding to vasopressin. Other causes of nephrogenic diabetes insipidus include

- Chronic kidney disease
- Certain medications, particularly lithium
- Low potassium levels in the blood
- High calcium levels in the blood
- Blockage of the urinary tract

The causes of nephrogenic diabetes insipidus can also be unknown.

### **Dipsogenic Diabetes Insipidus**

A defect in the thirst mechanism, located in a person's hypothalamus, causes dipsogenic diabetes insipidus. This defect results in an abnormal increase in thirst and liquid intake that suppresses vasopressin secretion and increases urine output. The same events and conditions that damage the hypothalamus or pituitary—surgery, infection, inflammation, a tumor, head injury—can also damage the thirst mechanism. Certain

medications or mental health problems may predispose a person to dipsogenic diabetes insipidus.

### **Gestational Diabetes Insipidus**

Gestational diabetes insipidus occurs only during pregnancy. In some cases, an enzyme made by the placenta—a temporary organ joining mother and baby—breaks down the mother's vasopressin. In other cases, pregnant women produce more prostaglandin, a hormone-like chemical that reduces kidney sensitivity to vasopressin. Most pregnant women who develop gestational diabetes insipidus have a mild case that does not cause noticeable symptoms. Gestational diabetes insipidus usually goes away after the mother delivers the baby; however, it may return if the mother becomes pregnant again.

### **Complications of diabetes insipidus**

The main complication of diabetes insipidus is dehydration if fluid loss is greater than liquid intake. Signs of dehydration include

- Thirst
- Dry skin
- Fatigue
- Sluggishness
- Dizziness
- Confusion
- Nausea

Severe dehydration can lead to seizures, permanent brain damage, and even death.

### **Seek Immediate Care**

Usually, people can prevent dehydration by increasing the amount of liquids they drink. However, some people may not realize they need to drink more liquids, which can lead to dehydration. People should seek immediate care if they experience symptoms of more severe dehydration, such as

- Confusion
- Dizziness
- Sluggishness

### **How is diabetes insipidus diagnosed?**

A health care provider can diagnose a person with diabetes insipidus based on the following:

- Medical and family history
- Physical exam
- Urinalysis
- Blood tests
- Fluid deprivation test
- Magnetic resonance imaging (mri)

### **Medical and Family History**

Taking a medical and family history can help a health care provider diagnose diabetes insipidus. A health care provider will ask the patient to review his or her symptoms and ask whether the patient's family has a history of diabetes insipidus or its symptoms.

## Physical Exam

A physical exam can help diagnose diabetes insipidus. During a physical exam, a health care provider usually examines the patient's skin and appearance, checking for signs of dehydration.

## Urinalysis

Urinalysis tests a urine sample. A patient collects the urine sample in a special container at home, in a health care provider's office, or at a commercial facility. A health care provider tests the sample in the same location or sends it to a lab for analysis. The test can show whether the urine is dilute or concentrated. The test can also show the presence of glucose, which can distinguish between diabetes insipidus and diabetes mellitus. The health care provider may also have the patient collect urine in a special container over a 24-hour period to measure the total amount of urine produced by the kidneys.

## Blood Tests

A blood test involves drawing a patient's blood at a health care provider's office or a commercial facility and sending the sample to a lab for analysis. The blood test measures sodium levels, which can help diagnose diabetes insipidus and in some cases determine the type.

## Fluid Deprivation Test

A fluid deprivation test measures changes in a patient's body weight and urine concentration after restricting liquid intake. A health care provider can perform two types of fluid deprivation tests:

- **A short form of the deprivation test.** A health care provider instructs the patient to stop drinking all liquids for a specific period of time, usually during dinner. The next morning, the patient will collect a urine sample at home. The patient then returns the urine sample to his or her health care provider or takes it to a lab where a technician measures the concentration of the urine sample.
- **A formal fluid deprivation test.** A health care provider performs this test in a hospital to continuously monitor the patient for signs of dehydration. Patients do not need anesthesia. A health care provider weighs the patient and analyzes a urine sample. The health care provider repeats the tests and measures the patient's blood pressure every 1 to 2 hours until one of the following happens:
  - The patient's blood pressure drops too low or the patient has a rapid heartbeat when standing.
  - The patient loses 5 percent or more of his or her initial body weight.
  - Urine concentration increases only slightly in two to three consecutive measurements.

At the end of the test, a health care provider will compare the patient's blood sodium, vasopressin levels, and urine concentration to determine whether the patient has diabetes

insipidus. Sometimes, the health care provider may administer medications during the test to see if they increase a patient's urine concentration. In other cases, the health care provider may give the patient a concentrated sodium solution intravenously at the end of the test to increase the patient's blood sodium level and determine if he or she has diabetes insipidus.

### **Magnetic Resonance Imaging**

Magnetic resonance imaging (MRI) is a test that takes pictures of the body's internal organs and soft tissues without using x-rays. A specially trained technician performs the procedure in an outpatient center or a hospital, and a radiologist—a doctor who specializes in medical imaging—interprets the images. A patient does not need anesthesia, although people with a fear of confined spaces may receive light sedation. An MRI may include an injection of a special dye, called contrast medium. With most MRI machines, the person lies on a table that slides into a tunnel-shaped device that may be open ended or closed at one end. Some MRI machines allow the patient to lie in a more open space. MRIs cannot diagnose diabetes insipidus. Instead, an MRI can show if the patient has problems with his or her hypothalamus or pituitary gland or help the health care provider determine if diabetes insipidus is the possible cause of the patient's symptoms.

### **Treatments**

The primary treatment for diabetes insipidus involves drinking enough liquid to prevent dehydration. A health care provider may refer a person with diabetes insipidus to a nephrologist—a doctor who specializes in treating kidney problems—or to an endocrinologist—a doctor who specializes in treating disorders of the hormone-producing glands. Treatment for frequent urination or constant thirst depends on the patient's type of diabetes insipidus:

- **Central diabetes insipidus.** A synthetic, or man-made, hormone called desmopressin treats central diabetes insipidus. The medication comes as an injection, a nasal spray, or a pill. The medication works by replacing the vasopressin that a patient's body normally produces. This treatment helps a patient manage symptoms of central diabetes insipidus; however, it does not cure the disease.
- **Nephrogenic diabetes insipidus.** In some cases, nephrogenic diabetes insipidus goes away after treatment of the cause. For example, switching medications or taking steps to balance the amount of calcium or potassium in the patient's body may resolve the problem. Medications for nephrogenic diabetes insipidus include diuretics, either alone or combined with aspirin or ibuprofen. Health care providers commonly prescribe diuretics to help patients' kidneys remove fluid from the body. Paradoxically, in people with nephrogenic diabetes insipidus, a class of diuretics called thiazides reduces urine production and helps patients' kidneys concentrate urine. Aspirin or ibuprofen also helps reduce urine volume.
- **Dipsogenic diabetes insipidus.** Researchers have not yet found an effective treatment for dipsogenic diabetes insipidus. People can try sucking on ice chips or sour candies to moisten their mouths and

increase saliva flow, which may reduce the desire to drink. For a person who wakes multiple times at night to urinate because of dipsogenic diabetes insipidus, taking a small dose of desmopressin at bedtime may help. Initially, the health care provider will monitor the patient's blood sodium levels to prevent hyponatremia, or low sodium levels in the blood.

- **Gestational diabetes insipidus.** A health care provider can prescribe desmopressin for women with gestational diabetes insipidus. An expecting mother's placenta does not destroy desmopressin as it does vasopressin. Most women will not need treatment after delivery.

Most people with diabetes insipidus can prevent serious problems and live a normal life if they follow the health care provider's recommendations and keep their symptoms under control.

### **Eating, Diet, and Nutrition**

Researchers have not found that eating, diet, and nutrition play a role in causing or preventing diabetes insipidus. But the below points should be remember to aware of diabetes insipidus.

### **Points to Remember**

- Diabetes insipidus is a rare disorder that occurs when a person's kidneys pass an abnormally large volume of urine that is insipid—dilute and odorless.
- A person's body regulates fluid by balancing liquid intake and removing extra fluid. Thirst usually controls a person's rate of liquid intake, while urination removes most fluid, although people also lose fluid through sweating, breathing, or diarrhea. The hormone vasopressin, also called antidiuretic hormone, controls the fluid removal rate through urination.
- The types of diabetes insipidus include central, nephrogenic, dipsogenic, and gestational. Each type of diabetes insipidus has a different cause.
- The main complication of diabetes insipidus is dehydration if fluid loss is greater than liquid intake.
- A health care provider can diagnose a person with diabetes insipidus based on a medical and family history, a physical exam, urinalysis, blood tests, a fluid deprivation test, and magnetic resonance imaging (MRI).
- The primary treatment for diabetes insipidus involves drinking enough liquid to prevent dehydration.

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## **7.3 GESTATIONAL DIABETES**

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Gestational diabetes is a condition in which a woman without diabetes develops high blood sugar levels during pregnancy. Gestational diabetes generally results in few symptoms; however, it does increase the risk of pre-eclampsia, depression, and requiring a Caesarean section. Babies born to mothers with poorly treated gestational diabetes are at increased risk of being too large, having low blood sugar after birth, and jaundice. If untreated, it can also result in a stillbirth. Long term, children are at higher risk of being overweight and developing type 2 diabetes.

Gestational diabetes is caused by not enough insulin in the setting of insulin resistance. Risk factors include being overweight, previously having gestational diabetes, a family history of type 2 diabetes, and having polycystic ovarian syndrome. Diagnosis is by blood tests. For those at normal risk, screening is recommended between 24 and 28 weeks' gestation. For those at high risk, testing may occur at the first prenatal visit.

Prevention is by maintaining a healthy weight and exercising before pregnancy. Gestational diabetes is treated with a diabetic diet, exercise, and possibly insulin injections. Most women are able to manage their blood sugar with diet and exercise. Blood sugar testing among those who are affected is often recommended four times a day. Breastfeeding is recommended as soon as possible after birth.

Gestational diabetes affects 3–9% of pregnancies, depending on the population studied. It is especially common during the last three months of pregnancy. It affects 1% of those under the age of 20 and 13% of those over the age of 44. A number of ethnic groups including Asians, American Indians, Indigenous Australians, and Pacific Islanders are at higher risk. In 90% of cases, gestational diabetes will resolve after the baby is born. Women, however, are at an increased risk of developing type 2 diabetes.

### Classification

Gestational diabetes is formally defined as "any degree of glucose intolerance with onset or first recognition during pregnancy". This definition acknowledges the possibility that a woman may have previously undiagnosed diabetes mellitus, or may have developed diabetes coincidentally with pregnancy. Whether symptoms subside after pregnancy is also irrelevant to the diagnosis. A woman is diagnosed with gestational diabetes when glucose intolerance continues beyond 24 to 28 weeks of gestation.

The White classification, named after Priscilla White, who pioneered research on the effect of diabetes types on perinatal outcome, is widely used to assess maternal and fetal risk.<sup>[7]</sup> It distinguishes between gestational diabetes (type A) and pre-gestational diabetes (diabetes that existed prior to pregnancy). These two groups are further subdivided according to their associated risks and management.

The two subtypes of gestational diabetes under this classification system are:

- Type A1: abnormal oral glucose tolerance test (OGTT), but normal blood glucose levels during fasting and two hours after meals; diet modification is sufficient to control glucose levels
- Type A2: abnormal OGTT compounded by abnormal glucose levels during fasting and/or after meals; additional therapy with insulin or other medications is required

Diabetes which existed prior to pregnancy is also split up into several subtypes under this system:

- Type B: onset at age 20 or older and duration of less than 10 years.
- Type C: onset at age 10–19 or duration of 10–19 years.



- Type D: onset before age 10 or duration greater than 20 years.
- Type E: overt diabetes mellitus with calcified pelvic vessels.
- Type F: diabetic nephropathy.
- Type R: proliferative retinopathy.
- Type RF: retinopathy and nephropathy.
- Type H: ischemic heart disease.
- Type T: prior kidney transplant.

An early age of onset or long-standing disease comes with greater risks, hence the first three subtypes. Two other sets of criteria are available for diagnosis of gestational diabetes, both based on blood-sugar levels. Criteria for diagnosis of gestational diabetes, using the 100 gram Glucose Tolerance Test, according to Carpenter and Coustan:

- Fasting 95 mg/dl
- 1 hour 180 mg/dl
- 2 hours 155 mg/dl
- 3 hours 140 mg/dl

Criteria for diagnosis of gestational diabetes according to National Diabetes Data Group:

- Fasting 105 mg/dl
- 1 hour 190 mg/dl
- 2 hours 165 mg/dl
- 3 hours 145 mg/dl

#### **Risk factors**

Classical risk factors for developing gestational diabetes are:

- Polycystic ovary syndrome
- A previous diagnosis of gestational diabetes or prediabetes, impaired glucose tolerance, or impaired fasting glycaemia
- A family history revealing a first-degree relative with type 2 diabetes
- Maternal age – a woman's risk factor increases as she gets older (especially for women over 35 years of age).
- Paternal age – one study found that a father's age over 55 years was associated with GD<sup>[13]</sup>
- Ethnicity (those with higher risk factors include African-Americans, Afro-Caribbeans, Native Americans, Hispanics, Pacific Islanders, and people originating from South Asia)
- Being overweight, obese or severely obese increases the risk by a factor 2.1, 3.6 and 8.6, respectively.
- A previous pregnancy which resulted in a child with a macrosomia (high birth weight: >90th centile or >4000 g (8 lbs 12.8 oz))
- Previous poor obstetric history
- Other genetic risk factors: There are at least 10 genes where certain polymorphism are associated with an increased risk of gestational diabetes, most notably TCF7L2.

In addition to this, statistics show a double risk of GDM in smokers. Polycystic ovarian syndrome is also a risk factor,<sup>[12]</sup> although relevant evidence remains controversial. Some studies have looked at more controversial potential risk factors, such as short stature.

About 40–60% of women with GDM have no demonstrable risk factor; for this reason many advocate to screen all women. Typically, women with GDM exhibit no symptoms (another reason for universal screening), but some women may demonstrate increased thirst, increased urination, fatigue, nausea and vomiting, bladderinfection, yeast infections and blurred vision.

### **Prevention**

A 2015 review found that when done during pregnancy moderate physical exercise is effective for the prevention of gestational diabetes. A 2014 review however did not find a significant effect.

Diet and physical activity interventions designed to prevent excessive gestational weight gain reduce the rates of gestational diabetes. However, the impact of these interventions varies with the body-mass index of the person as well as with the region in which the studies were performed.

It has been suggested that for women who have had gestational diabetes, support between pregnancies may lower their chances of having gestational diabetes again in future pregnancies. This support might include diet and exercise, education, and lifestyle advice. However, there is no research to show whether interventions between pregnancies lower the number of women who develop gestational diabetes again. Theoretically, smoking cessation may decrease the risk of gestational diabetes among smokers.

### **7.3.1. LIFESTYLE MANAGEMENT**

Treatment of GDM with diet and insulin reduces health problems mother and child. Treatment of GDM is also accompanied by more inductions of labour. A repeat OGTT should be carried out 6 weeks after delivery, to confirm the diabetes has disappeared. Afterwards, regular screening for type 2 diabetes is advised.

Lifestyle interventions include exercise, diet advice, behavioural interventions, relaxation, self-monitoring glucose, and combined interventions.<sup>[57]</sup> Women with gestational diabetes who receive lifestyle interventions seem to have less postpartum depression, and were more likely to reach their weight loss targets after giving birth, than women who had no intervention. Their babies are also less likely to be large for their gestational age, and have less percentage of fat when they are born. More research is needed to find out which lifestyle interventions are best.

If a diabetic diet or G.I. Diet, exercise, and oral medication are inadequate to control glucose levels, insulin therapy may become necessary. The development of macrosomia can be evaluated during pregnancy by using sonography. Women who use insulin, with a history of stillbirth, or with hypertension are managed like women with overt diabetes.

### **Lifestyle**

Counselling before pregnancy (for example, about preventive folic acid supplements) and multidisciplinary management are important for good pregnancy outcomes. Most women can manage their GDM with dietary changes and exercise. Self monitoring of blood glucose levels can guide therapy. Some women will need antidiabetic drugs, most

commonly insulin therapy. Any diet needs to provide sufficient calories for pregnancy, typically 2,000 – 2,500 kcal with the exclusion of simple carbohydrates. The main goal of dietary modifications is to avoid peaks in blood sugar levels. This can be done by spreading carbohydrate intake over meals and snacks throughout the day, and using slow-release carbohydrate sources—known as the G.I. Diet. Since insulin resistance is highest in mornings, breakfast carbohydrates need to be restricted more. Ingesting more fiber in foods with whole grains, or fruit and vegetables can also reduce the risk of gestational diabetes. There is not enough evidence to indicate if one type of dietary advice is better than another.

Regular moderately intense physical exercise is advised, although there is no consensus on the specific structure of exercise programs for GDM. Pregnant women who exercise have lower blood sugar levels when fasting and after meals compared to those who do not exercise. It is not clear which form of exercise is best when pregnant.

Self monitoring can be accomplished using a handheld capillary glucose dosage system. Compliance with these glucometer systems can be low. There is not a lot of research into what target blood sugar levels should be for women with gestational diabetes and targets recommended to women vary around the world. Target ranges advised by the Australasian Diabetes in Pregnancy Society are as follows:

- fasting capillary blood glucose levels <5.5 mmol/L
- 1 hour postprandial capillary blood glucose levels <8.0 mmol/L
- 2 hour postprandial blood glucose levels <6.7 mmol/L

Regular blood samples can be used to determine HbA1c levels, which give an idea of glucose control over a longer time period. Research suggests a possible benefit of breastfeeding to reduce the risk of diabetes and related risks for both mother and child.

### **Medication**

If monitoring reveals failing control of glucose levels with these measures, or if there is evidence of complications like excessive fetal growth, treatment with insulin might be necessary. This is most commonly fast-acting insulin given just before eating to blunt glucose rises after meals. Care needs to be taken to avoid low blood sugar levels due to excessive insulin. Insulin therapy can be normal or very tight; more injections can result in better control but requires more effort, and there is no consensus that it has large benefits. A 2016 Cochrane review concluded that quality evidence is not yet available to determine the best blood sugar range for improving health for pregnant women with GDM and their babies.

There is some evidence that certain medications by mouth might be safe in pregnancy, or at least, are less dangerous to the developing fetus than poorly controlled diabetes. When comparing which diabetes tablets (medication by mouth) work best and are safest, there is not enough quality research to support one medication over another. The medication metformin is better than glyburide. If blood glucose cannot be adequately controlled with a single agent, the combination of metformin and insulin may be better than insulin alone. Another review found good

short term safety for both the mother and baby with metformin but unclear long term safety.

People may prefer metformin by mouth to insulin injections. Treatment of polycystic ovarian syndrome with metformin during pregnancy has been noted to decrease GDM levels.

Almost half of the women did not reach sufficient control with metformin alone and needed supplemental therapy with insulin; compared to those treated with insulin alone, they required less insulin, and they gained less weight. With no long-term studies into children of women treated with the drug, there remains a possibility of long-term complications from metformin therapy. Babies born to women treated with metformin have been found to develop less visceral fat, making them less prone to insulin resistance in later life.

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## **7.4 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS**

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Diabetes insipidus is a rare disorder that occurs when a person's kidneys pass an abnormally large volume of urine that is insipid—dilute and odorless. In most people, the kidneys pass about 1 to 2 quarts of urine a day.

The types of diabetes insipidus include

- central
- nephrogenic
- dipsogenic
- gestational

Each type of diabetes insipidus has a different cause.

Central diabetes insipidus can also result from an inherited defect in the gene that produces vasopressin, although this cause is rare. In some cases, the cause is unknown.

The main complication of diabetes insipidus is dehydration if fluid loss is greater than liquid intake. Signs of dehydration include

- Thirst
- Dry skin
- Fatigue
- Sluggishness
- Dizziness
- Confusion
- Nausea

A health care provider can diagnose a person with diabetes insipidus based on the following:

- Medical and family history
- Physical exam
- Urinalysis
- Blood tests
- Fluid deprivation test
- Magnetic resonance imaging (mri)

### **Dietary Recommendations**

It is important to be meet with a registered dietitian to have your diet assessed. The dietitian will calculate the amount of carbohydrates that you

need at meals and snacks. You will also be taught how to count carbohydrates.

The following are dietary recommendations that will help you maintain safe blood sugar levels:

**Distribute your foods between three meals and two or three snacks each day**

Eating too much at one time can cause your blood sugar to rise too much. It is very important that you do not skip meals. During pregnancy, you have increased nutritional needs and your baby requires balanced nutrition.

**Eat reasonable portions of starch**

Starchy foods eventually turn into glucose so it's important not to be excessive. However, starch should be included in every meal. A reasonable portion is about one cup of total starch per meal, or two pieces of bread.

**Drink one cup of milk at a time**

Milk is a healthy food and an important source of calcium. However, milk is a liquid form of carbohydrate and drinking too much at one time can raise your blood sugar.

**Limit fruit portions**

Fruit is a healthy food, but it is high in natural sugars. You may eat one to three portions of fruit per day, but only eat one at a time. A portion of fruit is either one very small piece of fruit, half of a large piece of fruit, or about one-half cup of mixed fruit. Do not eat fruit that has been canned in syrup.

**Breakfast matters**

Blood sugar can be difficult to control in the morning because of normal fluctuations in hormone levels.

Refined cereals, fruits and even milk may not be well tolerated in your morning meal. If your post-breakfast blood sugar level increases too much after having these foods, then you should not eat them for your breakfast. A breakfast that consists of starch plus protein is usually tolerated the best.

**Avoid fruit juice**

It takes several fruits to make a glass of juice. Juice is a concentrated source of carbohydrate. Because it is liquid, juice can raise blood sugar quickly.

**Strictly limit sweets and desserts**

Cakes, cookies, candies and pastries tend to have excessive amounts of carbohydrate. These foods often contain large amounts of fat and offer very little in terms of nutrition. Additionally, avoid all regular sodas and sugar-sweetened beverages.

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**7.5 SUMMARY**

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There are various types of diabetes insipidus that occur due to different pathology that occurs outside of, during, and as a result of pregnancy. Gestational diabetes insipidus is a rare, but well recognized, complication of pregnancy. It is related to excess vasopressinase enzyme activity which is metabolized in the liver. A high index of suspicion of gestational diabetes insipidus is required in a correct clinical setting especially in the presence of other risk factors such as preeclampsia, HELLP syndrome, and twin pregnancies. We are presenting a case of gestational diabetes insipidus in a patient with HELLP syndrome. The newborn in this case also had hypernatremia thereby raising possibilities of vasopressinase crossing the placenta.

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## 7.6 KEY WORDS

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- **Gestational:** Gestation is the period of development during the carrying of an embryo, fetus, or reptilian embryo inside viviparous animals. It is typical for mammals, but also occurs for some non-mammals. Mammals during pregnancy can have one or more gestations at the same time, for example in a multiple birth.
- **Diabetes Insipidus:** Diabetes insipidus (die-uh-BEE-teze in-SIP-uh-dus) is an uncommon disorder that causes an imbalance of fluids in the body. This imbalance makes you very thirsty even if you've had something to drink. It also leads you to produce large amounts of urine.

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## 7.7 SELF-ASSESSMENT QUESTIONS

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### Short Answer Questions

1. What are the complications in diabetes insipidus?
2. Write a short note on gestational diabetes.
3. What are the treatments for diabetes insipidus?
4. Discuss the risk factors for developing gestational diabetes.

### Long Answer Questions

1. Explain the life style management during GDM period.
2. Explain different types of diabetes insipidus.

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## 7.8 FURTHER READINGS

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- American Diabetes Association: Nutrition principles and recommendations in diabetes (Position Statement). Diabetes Care 27(Suppl. 1):S36–S46, 2004
- Srilakshmi B, 2014, Dietetics, 7<sup>th</sup> Edition, New Age International Publisher, New Delhi.
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# BLOCK –III: DIETETICS IN CARDIOVASCULAR DISEASES

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## UNIT – VIII

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### Structure

- 8.0 Introduction
- 8.1 Objectives
- 8.2 cardiovascular diseases
  - 8.2.1 Risk factors
- 8.3. Blood lipids
  - 8.3.1.1. Classification
  - 8.3.1.2. Assessment
  - 8.3.1.3. Dyslipidemia and hypercholesterolemia
  - 8.3.1.4. Nutritional risk factors
- 8.4. Answers to check your Progress Questions
- 8.5. Summary
- 8.6. Key Words
- 8.7. Self-Assessment Questions and Answers
- 8.8. Further Readings

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### 8.0 INTRODUCTION

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Cardiovascular diseases (CVDs) have now become the leading cause of mortality in India. A quarter of all mortality is attributable to CVD. Ischemic heart disease and stroke are the predominant causes and are responsible for >80% of CVD deaths. The Global Burden of Disease study estimate of age-standardized CVD death rate of 272 per 100 000 population in India is higher than the global average of 235 per 100 000 population. Some aspects of the CVD epidemic in India are particular causes of concern, including its accelerated buildup, the early age of disease onset in the population, and the high case fatality rate. In India, the epidemiological transition from predominantly infectious disease conditions to noncommunicable diseases has occurred over a rather brief period of time. Premature mortality in terms of years of life lost because of CVD in India increased by 59%, from 23.2 million (1990) to 37 million (2010). Despite wide heterogeneity in the prevalence of cardiovascular risk factors across different regions, CVD has emerged as the leading cause of death in all parts of India, including poorer states and rural areas. The progression of the epidemic is characterized by the reversal of socioeconomic gradients; tobacco use and low fruit and vegetable intake have become more prevalent among those from lower socioeconomic backgrounds. In addition, individuals from lower socioeconomic backgrounds frequently do not receive optimal therapy, leading to poorer outcomes. Countering the epidemic requires the development of strategies such as the formulation and effective implementation of evidence-based policy, reinforcement of health systems, and emphasis on prevention, early



detection, and treatment with the use of both conventional and innovative techniques. Several ongoing community-based studies are testing these strategies.

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## 8.1 OBJECTIVES

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After studying this unit you will be able to:

- Expla  
in the risk factors of cardiovascular diseases.
  - Expla  
in in detail blood lipids.
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## 8.2 CARDIOVASCULAR DISEASES

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### Cardiovascular Diseases

Cardiovascular disease is a dominant cause of premature mortality in people. Cardiovascular disease in its various manifestations (coronary disease, cerebrovascular disease, peripheral vascular disease) has a long presymptomatic or incubation period, possibly 30-50 years in duration. The underlying pathological process in Coronary Heart Disease or CHD (and occlusive disease elsewhere) is atherosclerosis. This may give rise to a gradual obstruction of vessels and diminution in blood flow. Alternatively, a small coronary artery plaque, perhaps blocking only 30% of blood flow, may be unstable and fracture. This leads to coronary thrombosis which becomes a myocardial infarction (i.e. a heart attack).

India is currently experiencing an epidemic of Coronary artery disease (CAD). Statistics show that 20-25% of all medical admissions<sup>19</sup> and 25% of all mortality is due to CAD. The unhealthy life style practices viz., unbalanced dietary pattern, lack of physical activity, tobacco consumption, ill effects of urbanization, psychosocial stress, all contribute to a greater risk of developing CAD in Indians. The increasing rates of CAD mortality and the projected rise in CAD mortality for 2020 in the developing world necessitate immediate prevention and control measures. Experience in the developed world has shown that significant reductions in CAD prevalence and mortality can be achieved via primary and secondary preventive efforts as well as timely intervention and medical therapy. Despite this alarming burden of CVD, there are no definite guidelines at the national level to combat this serious problem.

The term CVD refers to a number of individual diseases affecting the cardiovascular system. Cardiovascular diseases account for over half of all deaths in middle age and one-third of all deaths in old age in most developed countries. Globally CVDs account for 30% of all deaths. CVD refers to disease of the arteries supplying the heart (CHD), the brain (cerebrovascular disease), and the extremities, especially the legs (peripheral vascular disease, PVD). It involves the processes of atherosclerosis (lesions in the arterial wall) and thrombosis (blood clotting), as well as changes to the function of the arterial lining. • CVD is the leading cause of death worldwide, accounting for around 18 million deaths each year. Around 50% of these deaths are from CHD and a further 25% from stroke

**Coronary Heart Disease (CHD)**

It is a condition in which the walls of the arteries supplying blood to the heart muscle (coronary arteries) become thickened. This thickening, caused by the development of lesions in the arterial wall, is called atherosclerosis; the lesions are called plaques. Atherosclerosis can restrict the supply of blood to the heart muscle (the myocardium) and may manifest to the patient as chest pain on exertion (angina) or breathlessness on exertion. If the cap covering the plaque ruptures, exposing the contents to the circulation, the blood may clot and obstruct the flow completely, resulting in a MI or heart attack. CHD is also known as ischaemic heart disease.

**Cerebrovascular Disease**

Cerebrovascular disease involves interruption of the blood supply to part of the brain and may result in a stroke or a transient ischaemic attack. There are two main types of stroke: ischaemic stroke and haemorrhagic stroke. Ischaemic stroke involves a blockage in the blood supply to the brain. The loss of blood supply to part of the brain may lead to irreversible damage to brain tissue. High blood pressure (hypertension) is a major risk factor for haemorrhagic stroke.

**Peripheral Vascular Disease**

Peripheral vascular disease (PVD) involves atherosclerotic plaques narrowing the arteries supplying regions other than the myocardium and brain. A common form involves narrowing of the arteries supplying blood to the legs. The result may be pain on exercise (claudication). In more severe cases, impaired blood supply leads to death of leg tissues, which require amputation.

CVDs, whether affecting the coronary, cerebral, or peripheral arteries, share a common pathophysiology involving atherosclerosis and thrombosis (or clotting).

**Risk Factors**

A number of risk factors have been identified. A short list of these factors might include: cholesterol and other lipid abnormalities, elevated blood pressure, cigarette smoking, diabetes, obesity, blood coagulation abnormalities, male gender, family history of premature CHD, increasing age. The disease is also a major cause of illness and disability, including angina and heart. Conventional lifestyle-related risk factors for CVD include smoking, raised circulating cholesterol levels, particularly low-density lipoprotein (LDL)-cholesterol, raised blood pressure, physical inactivity, obesity, and diabetes. However, these 'classical' risk factors cannot fully explain the regional, gender, socioeconomic, and ethnic differences in CVD, and emerging evidence suggests that other novel risk factors may play an important role.

Risk factors for cardiovascular disease are particular habits, behaviors, circumstances or conditions that increase a person's risk of developing cardiovascular disease, including lack of exercise, unhealthy eating, smoking, diabetes, age and family history.

Cardiovascular disease is a broad, umbrella term used to describe all conditions affecting the heart and circulatory system, including coronary heart disease, stroke, heart attack and aortic disease.<sup>1</sup>

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Good to know: Cardiovascular disease is sometimes called “heart disease”, but in medical terms, they are not exactly the same thing. Heart disease is a general term for conditions affecting the structure of the heart and the way it functions. All heart diseases are cardiovascular diseases. However, not all cardiovascular diseases are heart diseases. An example is stroke, which affects blood vessels in the brain, but not the heart itself.

Cardiovascular disease risk factors can be split into two categories: modifiable and non-modifiable.<sup>3</sup> Non-modifiable cardiovascular disease risk factors are those that cannot be changed. These include a person’s age, ethnicity and family history (genetics cannot be changed), among other factors. Modifiable cardiovascular disease risk factors are those that can be reduced or controlled with altered behavior. By making certain lifestyle changes, people are able to lower their chances of developing cardiovascular disease. Examples include smoking, diet and exercise. Possessing one or more risk factors increases a person’s risk of developing cardiovascular disease; it does not, however, mean that cardiovascular disease is an inevitability. If you think that you might have signs of cardiovascular disease, you can try using the Ada app for a free assessment.

**Family history**

There is a genetic element to cardiovascular disease, meaning a family history of the condition is considered to be a risk factor. Generally, this applies if a person’s first-degree relative developed CVD at what may be considered a relatively young age. This is the case if the person’s father or brother developed cardiovascular disease before the age of 55, or their mother or sister developed it before the age of 65.

A family history of high blood pressure (hypertension), high cholesterol and type 2 diabetes can also increase one’s chances of developing these conditions, which can in turn increase the risk of cardiovascular disease.

Having a family history of heart disease does not mean CVD is inevitable, but does make it more likely. Leading a healthy lifestyle is generally recommended to help reduce the risk of cardiovascular disease in those with a genetic predisposition to the condition.

**Age**

Older people are at greater risk of developing cardiovascular disease. Although the process of aging cannot be changed, leading a generally healthy lifestyle is recommended to help reduce the likelihood of developing heart and circulatory conditions.

**Ethnicity**

Statistics suggest that people of South Asian, African or Caribbean descent have a greater risk of developing cardiovascular disease. Type 2 diabetes – a risk factor in itself for cardiovascular disease – also seems to be more prevalent among these groups. The reasons for this are difficult to define. However, leading a healthy lifestyle is generally recommended as a way for people from all backgrounds to help prevent heart and circulatory disease from developing.

**Sex**

While it may have long been seen as a man’s disease, the risk of cardiovascular disease in women has been underestimated, and symptoms

may go unrecognized, complicating diagnosis and treatment. Though CVD risk factors are shared by men and women, some may be more prevalent and/or more significant for one sex or gender; for example, having diabetes may be a stronger risk for certain types of CVD in women. Research is ongoing.

Women tend to develop cardiovascular disease at an older age than men. This later age of onset in women is thought to be linked to the hormonal changes that follow menopause.

If you are concerned about your cardiovascular disease risk profile, it is advisable to consult a doctor, who should be able to answer any questions you may have.

### **Socioeconomic status**

People who have a low socioeconomic status seem to be at a greater risk of cardiovascular disease. Although the reasons behind this are multiple and their relationships complex, diet is generally considered to be one of the biggest factors, with those from a higher socioeconomic background typically having greater access to a more nutritionally-balanced diet.

### **Cholesterol**

High levels of low-density lipoprotein (LDL) cholesterol – also known as “bad cholesterol” – are linked to a range of cardiovascular diseases. Cholesterol is a fatty substance that is carried around the body by proteins. If too much LDL cholesterol is present, it can cause fatty substances to build up in the artery walls and lead to complications.

High levels of LDL cholesterol are often caused by factors such as an unhealthy diet, smoking, physical inactivity, high alcohol intake and liver and kidney disease. To reduce LDL cholesterol levels, people can eat a balanced diet, undertake regular exercise and quit smoking. Those with extremely high levels of LDL cholesterol may be prescribed medication to lower them, most often statins.

Good to know: High-density lipoprotein (HDL) cholesterol is known as “good cholesterol”. This cholesterol transports cholesterol and fats from around the body to the liver, where they can be removed. Unlike LDL, it is generally a good thing to have a high level of HDL, as this can help lower one’s risk of developing heart disease or having a stroke. Eating healthily, staying active, avoiding tobacco and limiting alcohol intake can all help to increase HDL cholesterol levels.

### **High blood pressure (hypertension)**

High blood pressure, known as hypertension, is another contributing factor to cardiovascular disease, including heart failure, stroke and heart attack. High blood pressure is often symptomless, but can be easily diagnosed by a doctor, using a routine test.

High blood pressure is often linked to being overweight, physical inactivity, a high intake of salt or alcohol or a family history of the disorder, but in some cases may have no apparent cause. Lifestyle changes may help to reduce high blood pressure and, in severe cases, medication may be prescribed.

### **Diabetes**

Having diabetes, a condition that causes high levels of glucose in the blood, is a risk factor for developing cardiovascular disease. High

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glucose levels can damage the artery walls and make the buildup of fatty deposits (atheroma) more likely. If these fatty deposits occur in the coronary arteries, they can lead to possible coronary heart disease and heart attack.

There are two types of diabetes: type 1, which involves the body being unable to produce insulin and which usually develops in children and young adults, and type 2 diabetes, which is more likely to affect older people, though is becoming more common in younger people, and which involves the body either not making enough insulin or the body becoming resistant to insulin. Type 2 diabetes is closely associated with a lifestyle that leads to being overweight and physical inactivity.

Eating a balanced diet, taking regular exercise and leading a generally healthy lifestyle can both help manage diabetes in those who already have the condition and help prevent the onset of the condition in those that don't. In people with diabetes, careful management of blood sugar levels is also very important in helping to reduce the risk of cardiovascular disease.

### **Smoking**

Smoking tobacco significantly increases the chance of developing cardiovascular disease. Smoking damages and narrows the arteries, making angina pectoris and heart attack more likely. Angina pectoris is a condition characterized by pain or discomfort in the center of the chest, caused by the heart muscle not getting enough blood. Nicotine also makes the heart beat faster and increases blood pressure, meaning the heart has to work harder to pump blood around the body.

Soon after quitting smoking, health benefits such as improved circulation, better taste and smell and a stronger immune system can usually be noticed. Doctors and other health professionals are able to offer advice on how to quit smoking.

### **Physical inactivity**

Physical inactivity is an important risk factor for cardiovascular disease. Not exercising regularly increases a person's chances of being overweight, of having high blood pressure and of developing other conditions that make cardiovascular disease more likely.<sup>20</sup>

To see substantial health benefits, experts recommend that adults do at least 150 minutes of moderate to high-intensity exercise per week.

### **Being overweight (obesity)**

Being overweight is another leading risk factor for cardiovascular disease. Eating an unhealthy diet and being physically inactive are both contributing factors to being overweight, which is generally defined as having a body mass index (BMI) outside the normal range.

Taking steps to lose weight through lifestyle and dietary changes can help reduce the risk of a range of cardiovascular conditions, including coronary heart disease and congestive heart failure.

### **Diet**

Eating an unhealthy diet is a significant risk factor for cardiovascular disease. To lower the risk, a balanced diet made up of plenty of fruits and vegetables, complex carbohydrates and protein should be aimed at and excess fats, salts and sugars avoided.

Alcohol should also be consumed in moderation, if at all. In many countries, this is defined as a maximum of 14 units of alcohol per week, with some experts recommending half that for women. The week should include several alcohol-free days. One unit is equal to approximately one small glass of beer or wine, or one “shot” of distilled spirits or liquor, e.g. whisky, gin.

## **BLOOD LIPIDS**

### **CLASSIFICATION**

#### **Lipids and Lipoproteins**

Fats such as cholesterol and triglycerides are transported in the bloodstream as part of lipoprotein particles. The classes include Low Density Lipoprotein (LDL, which carries most of the blood cholesterol), Very Low Density Lipoprotein (VLDL, which carry most of the blood triglycerides), High Density Lipoprotein (HDL cholesterol) and Chylomicrons (recently absorbed fat). Excess LDL cholesterol is the most widely accepted risk factor for heart disease. Excess triglycerides are probably important in CHD but this is controversial. A low concentration of HDL cholesterol is also accepted as a major risk factor. Excess triglycerides may be more important when HDL cholesterol is low. Serum cholesterol has consistently been shown to be a significant risk factor for CHD and other major cardiovascular diseases as well. The impact of cholesterol on CHD risk is increased if other risk factors are simultaneously present. Cholesterol is a risk factor for a first coronary event. It appears to be highly predictive of a recurrent event.

There are four major classes of circulating lipoproteins, each with its own characteristic protein and lipid composition. They are chylomicrons, very low-density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). Within all these classes of complexes, the various molecular components are not chemically linked to each other but are simply associated in such a way as to minimize hydrophobic contacts with water. The most distinguishing feature of each class is the relative amounts of lipid and protein. Because the lipid and protein composition is reflected in the density of each lipoprotein (lipid molecules being less dense than proteins), density, an easily measured attribute, forms the operational basis of defining the lipoprotein classes. Measuring density also provides the basis of separating and purifying lipoproteins from plasma for study and diagnosis. The table gives a summary of the characteristics of the lipoprotein classes and shows the correlation between composition and density.

The principal lipid components are triglycerides, cholesterol, cholesteryl esters, and phospholipids. The hydrophobic core of the particle is formed by the triglycerides and cholesteryl esters. The fatty acyl chains of these components are unsaturated, and so the core structure is liquid at body temperature. The table gives more details about the nine different protein components, called apoproteins, of the lipoprotein classes. With the exception of LDL, which contains only one type of apoprotein, all classes have multiple apoprotein components. All the apoproteins, like phospholipids, are amphipathic and interact favourably with both lipids and water.

## NOTES

Chylomicrons are the largest lipoproteins, with diameters of 75–600 nanometres (nm;  $1 \text{ nm} = 10^{-9}$  metre). They have the lowest protein-to-lipid ratio (being about 90 percent lipid) and therefore the lowest density. Chylomicrons are synthesized by the absorptive cells of the intestinal lining and are secreted by these cells into the lymphatic system, which joins the blood circulation at the subclavian vein. The triglyceride, cholesteryl ester, and free cholesterol content of these particles is derived from the digestion of dietary fat. Their primary destinations in peripheral areas are heart muscle, skeletal muscle, adipose tissue, and lactating mammary tissue. The transfer of triglycerides and cholesteryl esters to the tissues depletes the lipid-protein. VLDL is a lipoprotein class synthesized by the liver that is analogous to the chylomicrons secreted by the intestine. Its purpose is also to deliver triglycerides, cholesteryl esters, and cholesterol to peripheral tissues. VLDL is largely depleted of its triglyceride content in these tissues and gives rise to an intermediate-density lipoprotein (IDL) remnant, which is returned to the liver in the bloodstream. As might be expected, the same proteins are present in both VLDL and IDL.

Low-density lipoproteins are derived from VLDL and IDL in the plasma and contain a large amount of cholesterol and cholesteryl esters. Their principal role is to deliver these two forms of cholesterol to peripheral tissues. Almost two-thirds of the cholesterol and its esters found in plasma (blood free of red and white cells) is associated with LDL. Lipoproteins of this class are the smallest, with a diameter of 10.8 nm and the highest protein-to-lipid ratio. The resulting high density gives this class its name. HDL plays a primary role in the removal of excess cholesterol from cells and returning it to the liver, where it is metabolized to bile acids and salts that are eventually eliminated through the intestine. LDL and HDL together are the major factors in maintaining the cholesterol balance of the body. Because of the high correlation between blood cholesterol levels and atherosclerosis, high ratios of HDL to cholesterol (principally as found in LDL) correlate well with a lower incidence of this disease in humans.

**Assessment**

Current recommendations for cholesterol testing come from the Adult Treatment Panel (ATP) III guidelines, and are based on many large clinical studies, such as the Framingham Heart Study.

For healthy adults with no cardiovascular risk factors, the ATP III guidelines recommend screening once every five years.<sup>[1]</sup> A lipid profile may also be ordered at regular intervals to evaluate the success of lipid-lowering drugs such as statins.

In the pediatric and adolescent population, lipid testing is not routinely performed. However, the American Academy of Pediatrics and NHLBI now recommend that children aged 9–11 be screened once for severe cholesterol abnormalities.<sup>[2]</sup> This screening can be valuable to detect genetic diseases such as familial hypercholesterolemia that can be lethal if not treated early.

Traditionally, most laboratories have required patients to fast for 9–12 hours before screening. However, recent studies have questioned the utility of fasting before lipid panels, and some diagnostic labs now routinely accept non-fasting samples.

Typically the laboratory measures only three quantities: total cholesterol; HDL; Triglycerides. From these three data LDL may be calculated. According to Friedewald's equation:

- $LDL = \text{Total cholesterol} - HDL - \text{Triglycerides}/5$

Other calculations of LDL from those same three data have been proposed which yield some significantly different results. <sup>[5]</sup>

VLDL may be defined as the total cholesterol that is neither HDL nor LDL. Then Friedewald's equation mentioned above yields:

- $VLDL = \text{Triglycerides}/5$

The alternative calculations mentioned above may yield significantly different values for VLDL.

### **Dyslipidemia and Hypercholesterolemia**

**Dyslipidemia:** A disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency. Dyslipidemias may be manifested by elevation of the total cholesterol, the "bad" low-density lipoprotein (LDL) cholesterol and the triglyceride concentrations, and a decrease in the "good" high-density lipoprotein (HDL) cholesterol concentration in the blood.

Dyslipidemia comes under consideration in many situations including diabetes, a common cause of hyperlipidemia. For adults with diabetes, it has been recommended that the levels of LDL, HDL, and total cholesterol, and triglyceride be measured every year. Optimal LDL cholesterol levels for adults with diabetes are less than 100 mg/dL (2.60 mmol/L), optimal HDL cholesterol levels are equal to or greater than 40 mg/dL (1.02 mmol/L), and desirable triglyceride levels are less than 150 mg/dL (1.7 mmol/L).

Hypercholesterolaemia is defined as elevated amounts of cholesterol in the blood. **Cholesterol** is a waxy, fat-like substance that is naturally found in the walls of cells. It is used by the body to produce certain hormones, vitamin D, and bile acids that help to digest fat. If the amounts of cholesterol in the blood are excessive, cholesterol can build up in arteries, which can lead to coronary heart disease and many other serious conditions.

**Nonfamilial (non-inherited) hypercholesterolaemia** is the most common form of hypercholesterolaemia. It occurs in people with a susceptible genotype which is aggravated by excessive intake of saturated fats and cholesterol.

**Familial hypercholesterolemia** is an inherited genetic disorder. The children of people with familial hypercholesterolemia may inherit either the normal gene or the defective gene, so the prevalence within that family will be approximately 50%.

### **Nutritional risk factors**



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A number of factors are associated with the build-up of fatty deposits in the coronary arteries, including cigarette smoking, lack of physical activity and a family history of the disease.

**Other risk factors include:**

- **Type of fat eaten** – saturated and trans fats increase blood cholesterol and heart attack rates. Polyunsaturated and monounsaturated fats lower the risk of heart attacks.
- **Obesity** – many overweight and obese people have diets high in fat, particularly saturated fat. A person who carries the bulk of their body fat around their stomach (an ‘apple’ shaped body) is at greater risk of heart disease than someone whose body fat tends to settle around their bottom, hips and thighs (a ‘pear’ shaped body).
- **High blood pressure (hypertension)** – blood pressure is the amount of pressure within the arteries (blood vessels that carry blood around the body). High blood pressure, or hypertension, means that the pressure in the arteries is higher than normal. This may be because the arteries are less elastic, there is more blood volume, or more blood is being pumped out of the heart.
- **Uncontrolled diabetes and impaired glucose tolerance**  
In healthy people, insulin keeps the blood sugar level relatively constant. However, for those vulnerable to type 2 diabetes, the body gradually loses its sensitivity to insulin. This leads to chronically elevated blood sugar levels, also known as impaired glucose tolerance.

Uncontrolled diabetes can damage the artery walls and contribute to coronary heart disease. People who are obese are more likely to develop type 2 diabetes than people of normal weight. Australian Aboriginal and Torres Strait Islander peoples have much higher rates of diabetes than other Australians, even at lower body weights.

**Cholesterol levels and dietary fats**

Cholesterol is a fat that is crucial to many metabolic functions and is an essential part of all the body’s cell membranes. Cholesterol is produced in the body from the food we eat and is produced in the liver.

Blood lipids (fats) that contain cholesterol include low density lipoprotein (LDL) and high density lipoprotein (HDL). LDL cholesterol can lead to plaque forming on arteries. HDL cholesterol helps the body to remove cholesterol from the body and makes it harder for plaque to form in the arteries.

Saturated and trans fats in the diet tend to increase LDL cholesterol in the blood. Common sources of saturated fats include animal products (butter, meat fat, beef, lamb, chicken skin and full cream dairy foods), and processed foods like pastries and biscuits.

Trans fatty acids and saturated fats, such as elaidic acid, are formed when monounsaturated or polyunsaturated vegetable oils are hydrogenated and hardened to form margarines. This applies particularly to the harder vegetable fat and shortening used by the food industry in products such as cakes and biscuits.

Most monounsaturated and polyunsaturated table margarines sold in Australia have very low levels of trans fatty acids. The small amounts

present in table margarines are not significant and do not negate the benefits of substituting these margarines for saturated fats like butter. Trans fatty acids also occur naturally in some meats, butter and dairy products.

These trans fats tend to increase the LDL cholesterol.

Saturated fat (e.g. found in red-meat, cheese, full fat dairy) increases the total and LDL-cholesterol, a major CVD risk factor.

Trans-fat (e.g. found in fried and baked goods) increases the total and LDL- cholesterol, as well as lowering the beneficial HDL cholesterol.

Changing the type of fat in our diet (replacing saturated fats with unsaturated fats) may reduce the risk of CVD. Unsaturated fats are found in olive oil, nuts, seeds and oily fish.

Eating foods that naturally contain cholesterol (e.g. eggs, shellfish, organ meats) have less impact on blood cholesterol levels than eating foods that are high in saturated fat.

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## 8.4 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS

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Cardiovascular disease is a dominant cause of premature mortality in people. Cardiovascular disease in its various manifestations (coronary disease, cerebrovascular disease, peripheral vascular disease) has a long presymptomatic or incubation period, possibly 30-50 years in duration. The underlying pathological process in Coronary Heart Disease or CHD (and occlusive disease elsewhere) is atherosclerosis. This may give rise to a gradual obstruction of vessels and diminution in blood flow. Alternatively, a small coronary artery plaque, perhaps blocking only 30% of blood flow, may be unstable and fracture. This leads to coronary thrombosis which becomes a myocardial infarction (i.e. a heart attack).

The term CVD refers to a number of individual diseases affecting the cardiovascular system. Cardiovascular diseases account for over half of all deaths in middle age and one-third of all deaths in old age in most developed countries. Globally CVDs account for 30% of all deaths. CVD refers to disease of the arteries supplying the heart (CHD), the brain (cerebrovascular disease) and the extremities, especially the legs (peripheral vascular disease, PVD).

It is a condition in which the walls of the arteries supplying blood to the heart muscle (coronary arteries) become thickened. This thickening, caused by the development of lesions in the arterial wall, is called atherosclerosis; the lesions are called plaques.

A number of risk factors have been identified. A short list of these factors might include: cholesterol and other lipid abnormalities, elevated blood pressure, cigarette smoking, diabetes, obesity, blood coagulation abnormalities, male gender, family history of premature CHD, increasing age. The disease is also a major cause of illness and disability, including angina and heart.

### **Nutritional risk factors**

- Saturated fat (e.g. found in red-meat, cheese, full fat dairy) increases the total and LDL-cholesterol, a major CVD risk factor .

- Trans-fat (e.g. found in fried and baked goods) increases the total and LDL- cholesterol, as well as lowering the beneficial HDL cholesterol.
- Changing the type of fat in our diet (replacing saturated fats with unsaturated fats) may reduce the risk of CVD. Unsaturated fats are found in olive oil, nuts, seeds and oily fish.
- Eating foods that naturally contain cholesterol (e.g. eggs, shellfish, organ meats) have less impact on blood cholesterol levels than eating foods that are high in saturated fat.

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## 8.5 SUMMARY

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The major cardiovascular diseases affecting the developed world have at their core atherosclerosis and hypertension, both of which are profoundly affected by diet and can be approached, at least in part, from a nutritional point of view, as can the increasing “epidemic” of obesity. Diet is a multi-component mixture of many nutrients, which may interact with one another. The definitive study of nutrients and their impact on cardiovascular disease can be a daunting enterprise. Many dietary risk factors contribute to these diseases in various environmental and ethnic settings. These risk factors are often in evidence in youth so that preventive measures must be initiated early in life. Although most of the information about nutritional risk factors and cardiovascular disease derives from studies in the developed world, the situation is rapidly evolving toward epidemic proportions in the developing world where a major burden on the economy and health services will be imposed.

Many risk factors for cardiovascular diseases are profoundly affected by diet. Although most of the information about nutritional risk factors and cardiovascular disease derives from studies in the developed world, the situation is rapidly evolving toward epidemic proportions in the developing world, leading to impending economic and health service burdens.

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## 8.6 KEY WORDS

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- **CVD:** Cardiovascular Diseases.
- **LDL:** low-density lipoprotein (LDL).
- **HDL:** high-density lipoprotein (HDL)
- **VLDL:** very low-density lipoproteins (VLDL)

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## 8.7 SELF ASSESSMENT QUESTIONS

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### Short Answer Questions

1. Write a short note on CVD.
2. Explain peripheral vascular disease.
3. Brief on blood lipids classification.
4. Discuss the Dyslipidemia and Hypercholesterolemia.

### Long Answer Questions

1. Explain food supplementation CVD.
2. Elaborate on risk factors of CVD.

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## **8.8 FURTHER READINGS**

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- Suryatapas, 2018, Textbook of Community Nutrition, 3<sup>rd</sup> Edition, Academic Publishers, West Bengal.
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*NOTES*

*Self-Instructional Material*

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# UNIT-IX ATHEROSCLEROSIS

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Atherosclerosis

NOTES

## Structure

- 9.0 Introduction
  - 9.1 Objectives
  - 9.2 Atherosclerosis
    - 9.2.1.1 Disease progression
    - 9.2.1.2 Causes
    - 9.2.1.3 Symptoms
    - 9.2.1.4 Clinical findings
  - 9.3 Management of atherosclerosis
  - 9.4 Answers to Check Your Progress Questions
  - 9.5 Summary
  - 9.6 Key Words
  - 9.7 Self-Assessment Questions
  - 9.8 Further Readings

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## 9.0 INTRODUCTION

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In previous units we have discussed about CVD and its risk factors. Atherosclerosis is a narrowing of the arteries caused by a buildup of plaque. It's also called arteriosclerosis or hardening of the arteries. Arteries are the blood vessels that carry oxygen and nutrients from your heart to the rest of your body. As you get older, fat and cholesterol can collect in your arteries and form plaque. The buildup of plaque makes it difficult for blood to flow through your arteries. This buildup may occur in any artery in your body and can result in a shortage of blood and oxygen in various tissues of your body. Pieces of plaque can also break off, causing a blood clot. Atherosclerosis can lead to heart attack, stroke, or heart failure if left untreated. Atherosclerosis is a fairly common problem associated with aging. This condition can be prevented, and many successful treatment options exist. In this unit we shall study about atherosclerosis diseases prognosis, causes, symptoms, clinical findings and management.

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## 9.1 OBJECTIVES

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After studying this unit you will know

- About atherosclerosis causes, symptoms and its management.
- Explain about the disease progression of atherosclerosis

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## 9.2 ATHEROSCLEROSIS

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Atherosclerosis is the narrowing of arteries due to plaque build-up on the artery walls. Arteries carry blood from the heart to the rest of the body. They are lined with a thin layer of cells that keeps them smooth and allows blood to flow easily. This is called the endothelium. Atherosclerosis starts when the endothelium becomes damaged, allowing the harmful type of cholesterol to build up in the artery wall.

The body sends a type of white blood cell to clean up this cholesterol, but, sometimes, the cells get stuck at the affected site. Over

Self-Instructional Material

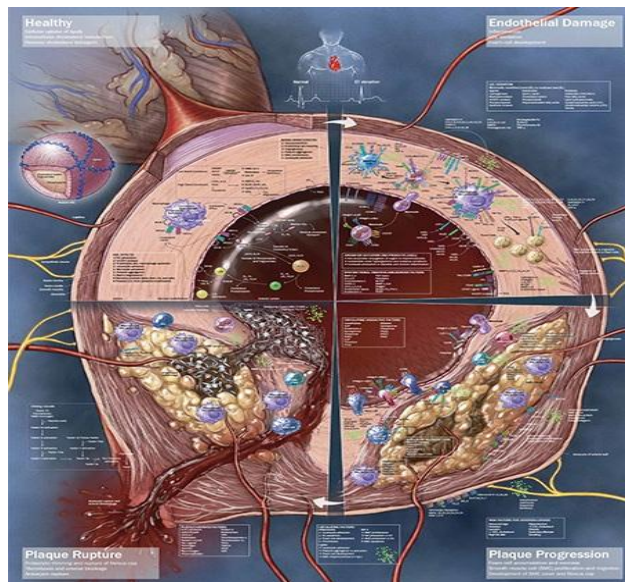
time, plaque can build up, made of cholesterol, macrophages, calcium, and other substances from the blood.

Sometimes, the plaque grows to a certain size and stops growing, causing the individual no problems. However, sometimes, the plaque clogs up the artery, disrupting the flow of blood around the body. This makes blood clots more likely, which can result in life-threatening conditions.

In some cases, the plaque eventually, breaks open. If this happens, platelets gather in the affected area and can stick together, forming blood clots. This can block the artery, leading to life-threatening complications, such as stroke and heart attack. The condition can affect the entire artery tree, but mainly affects the larger, high-pressure arteries. Let us discuss each of the heading in detail.

### 9.2.1. DISEASE PROGRESSION

Atherosclerosis is a complex multi-stage disease of the vasculature and begins with damage to the endothelial vessel lining. Vascular endothelial cells respond to the damage by upregulating luminal adhesion proteins, inflammatory mediators, and oxidative enzymes. Immune cells adhere to the activated endothelium and extravasate to the intima where they expand the inflammatory response. Lipoprotein particles are targeted by oxidative enzymes to generate pathogenic oxidized LDL. Macrophages within the intimal layer then phagocytose and accumulate oxLDL, which promotes their development into foam cells. Foam cells accumulate and give rise to fatty streaks within the vessel wall.



Vascular smooth muscle cells proliferate, migrate, and secrete matrix proteoglycans to form a protective fibrous cap around the plaque. They undergo partial transdifferentiation and adopt a bone cell-like phenotype. This leads to vessel wall calcification and loss of arterial elasticity. Intimal-medial layer thickening (IMT) of the artery is indicative of atherosclerosis progression. In addition, local upregulation of angiogenic factors induces neovascularization to supply blood to the growing plaque.

Foam cell death and lack of dead cell clearance result in the formation of a necrotic core within the plaque. Increased protease expression by both stromal and immune cells progressively weakens the fibrous cap. Fibrous cap thinning eventually leads to plaque rupture into the arterial lumen with direct exposure of the blood to the necrotic core. This triggers rapid thrombosis in the arterial lumen, blockage of blood flow (stenosis), and possible stroke or myocardial infarction. Alternatively, weakening of the arterial vessel wall contributes to aneurysm formation and potential bleeding into the extravascular space. The development of atherosclerosis is accelerated by a high fat diet, smoking, age, hypertension, and genetic mutations in molecules involved in normal vascular biology.

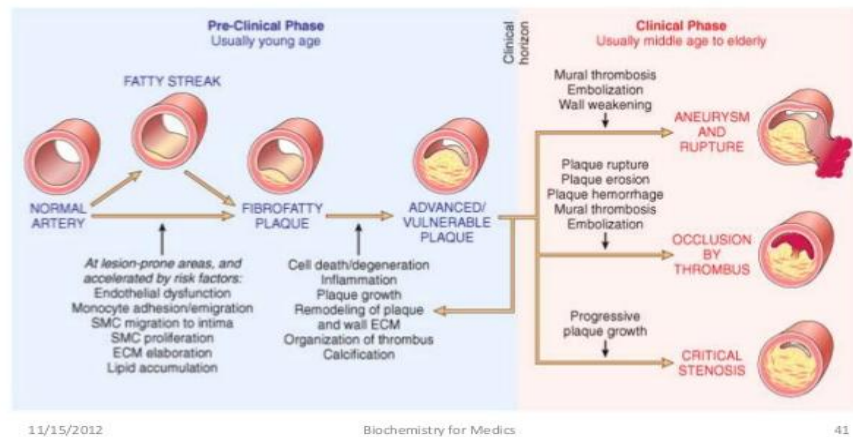
The atheromatous plaque is divided into three distinct components:

1. The atheroma ("lump of wax", from *Athera*, wax in Greek), which is the nodular accumulation of a soft, flaky, yellowish material at the center of large plaques, composed of macrophages nearest the lumen of the artery
2. Underlying areas of cholesterol crystals
3. Calcification at the outer base of older/more advanced lesions.

Atherosclerosis, produces two main problems:

First, the atheromatous plaques, though long compensated for by artery enlargement, eventually lead to plaque ruptures and clots inside the artery lumen over the ruptures. The clots heal and usually shrink but leave behind stenosis (narrowing) of the artery (both locally and in smaller downstream branches), or worse, complete closure, and, therefore, an insufficient blood supply to the tissues and organ it feeds.

## Progression of Atherosclerosis



Second, if the compensating artery enlargement process is excessive, then a net aneurysm results. These complications of advanced atherosclerosis are chronic, slowly progressive and cumulative. Most commonly, soft plaque suddenly ruptures (see vulnerable plaque), causing the formation of a thrombus that will rapidly slow or stop blood flow, leading to death of the tissues fed by the artery in approximately 5 minutes. This catastrophic event is called an infarction. One of the most common recognized scenarios is called coronary thrombosis of a coronary artery,

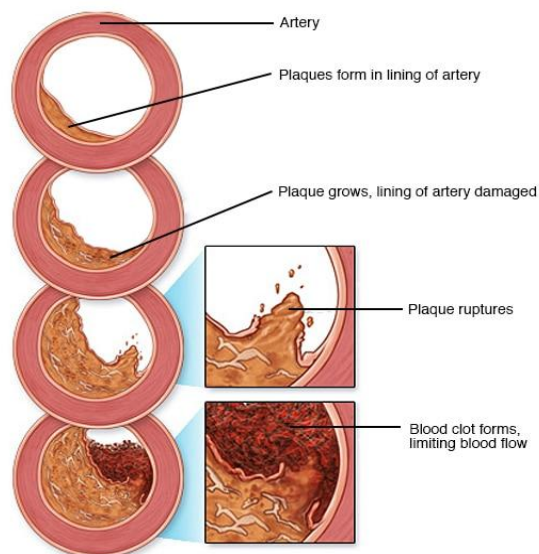
causing myocardial infarction (a heart attack). Even worse is the same process in an artery to the brain, commonly called stroke. Another common scenario in very advanced disease is claudication from insufficient blood supply to the legs, typically due to a combination of both stenosis and aneurysmal segments narrowed with clots. Since atherosclerosis is a body-wide process, similar events occur also in the arteries to the brain, intestines, kidneys, legs, etc.

### 9.2.2. CAUSES

Atherosclerosis is a slow, progressive disease that may begin as early as childhood. Although the exact cause is unknown, atherosclerosis may start with damage or injury to the inner layer of an artery. The damage may be caused by:

- High blood pressure
- High cholesterol
- High triglycerides, a type of fat (lipid) in your blood
- Smoking and other sources of tobacco
- Insulin resistance, obesity or diabetes
- Inflammation from diseases, such as arthritis, lupus or infections, or inflammation of unknown cause

Once the inner wall of an artery is damaged, blood cells and other substances often clump at the injury site and build up in the inner lining of the artery.



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Over time, fatty deposits (plaque) made of cholesterol and other cellular products also build up at the injury site and harden, narrowing your arteries. The organs and tissues connected to the blocked arteries then don't receive enough blood to function properly. Eventually, pieces of the fatty deposits may break off and enter your bloodstream.

In addition, the smooth lining of the plaque may rupture, spilling cholesterol and other substances into your bloodstream. This may cause a blood clot, which can block the blood flow to a specific part of your body, such as occurs when blocked blood flow to your heart causes a heart attack.



A blood clot can also travel to other parts of your body, blocking flow to another organ.

### 9.2.3. SYMPTOMS

Atherosclerosis typically begins in early adolescence, and is usually found in most major arteries, yet is asymptomatic and not detected by most diagnostic methods during life. Atheroma in arm, or more often in leg arteries, which produces decreased blood flow, is called peripheral artery occlusive disease (PAOD). The first symptom of atherosclerotic cardiovascular disease is heart attack or sudden cardiac death (death within one hour of onset of the symptom).

Atherosclerosis usually doesn't cause signs and symptoms until it severely narrows or totally blocks an artery. Angina may feel like pressure or a squeezing pain in your chest. You also may feel it in your shoulders, arms, neck, jaw, or back. This pain tends to get worse with activity and go away when you rest. Emotional stress also can trigger the pain. Shortness of breath, Arrhythmias (irregular heartbeats). Sudden numbness, weakness, and dizziness. Signs and symptoms of atherosclerosis are not visible until. The arteries are severely narrowed or blocked. Signs and symptoms differ depending on the arteries affected by atherosclerosis.

Most symptoms of atherosclerosis don't show up until a blockage occurs. Common symptoms include:

- Chest pain or angina
- Pain in your leg, arm, and anywhere else that has a blocked artery
- shortness of breath
- fatigue
- Confusion, which occurs if the blockage affects circulation to your brain
- muscle weakness in your legs from lack of circulation

It's also important to know the symptoms of heart attack and stroke. Both of these can be caused by atherosclerosis and require immediate medical attention.

The symptoms of a heart attack include:

- Chest pain or discomfort
- Pain in the shoulders, back, neck, arms, and jaw
- Abdominal pain
- Shortness of breath
- Perspiration
- Lightheadedness
- Nausea or vomiting
- A sense of impending doom

The symptoms of stroke include:

- Weakness or numbness in the face or limbs
- Trouble speaking
- Trouble understanding speech
- Vision problems
- Loss of balance
- sudden, severe headache

Heart attack and stroke are both medical emergencies. Call 911 or your local emergency services and get to a hospital's emergency room as soon as possible if you experience symptoms of a heart attack or stroke.

#### 9.2.4. CLINICAL FINDINGS

Atherosclerosis does not usually produce symptoms until blood circulation becomes restricted or blocked, leading to cardiovascular disease (CVD). The type of cardiovascular disease and its associated symptoms depends on where the blockage occurs. Conditions caused by atherosclerosis include peripheral arterial disease, angina, aneurysm, heart attack and stroke.

Atherosclerosis does not usually produce symptoms until blood circulation becomes restricted or blocked, leading to cardiovascular disease (CVD). The type of cardiovascular disease and its associated symptoms depends on where the blockage occurs. Conditions caused by atherosclerosis include peripheral arterial disease, angina, aneurysm, heart attack and stroke.

**Congestive Heart failure** - Congestive heart failure (CHF) is a chronic progressive condition that affects the pumping power of heart muscles. While often referred to simply as “heart failure”, CHF specifically refers to the stage in which fluid builds up around the heart causing it to pump inefficiently. CHF develops when your ventricles can’t pump blood in sufficient volume to the body. Eventually, blood and other fluids back up inside lungs, abdomen, liver, lower body.

**Myocardial Infarction** - Myocardial infarction (MI) or acute myocardial infarction (AMI), commonly known as a heart attack occurs when blood flow stops to a part of the heart causing damage to the heart muscle. The most common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck, or jaw. Often it is in the centre or left side of the chest and lasts for more than a few minutes. About 30% of people have atypical symptoms, with women more likely than men to present atypically. Among those over 75 years old, about 5% have had an MI with little or no history of symptoms. Approximately 1.5 million cases of MI occur annually in the United States.

**Arrhythmias** - Arrhythmia means irregular heartbeat either beating too fast or too slow. It just means it's out of its normal rhythm. It may feel like heart skipped a beat, added a beat, is fluttering, or is beating too fast its tachycardia or too slow its bradycardia.

**Pulmonary Embolism** - Pulmonary embolism is a blockage in one of the pulmonary arteries in lungs. In most cases, pulmonary embolism is caused by blood clots that travel to the lungs from the legs or, rarely, other parts of the body. Pulmonary embolism can be life-threatening, but prompt treatment can greatly reduce the risk of death.

**Cardiomyopathy** - Cardiomyopathy is a condition in which heart muscle becomes inflamed and enlarged. Because it is enlarged, heart muscle is stretched and becomes weak. This means it can’t pump blood as fast as it should. If heart muscle becomes too weak, may develop heart failure. Most people are only mildly affected by cardiomyopathy and can lead relatively normal lives. However, people who have severe heart failure may need a heart transplant. Cardiomyopathy is different to a heart attack. Heart

attacks also damage part of your heart muscle, but may be caused by something else.

- Congestive Heart Failure
- Cardiomyopathy
- Pulmonary Embolism
- Arrhythmias
- Myocardial Infraction

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### 9.3. MANAGEMENT

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#### Management dietary and lifestyle

The risk factors include high Cholesterol and low-density lipoprotein (LDL) in the blood, low level of high-density lipoprotein (HDL) in the blood, Hypertension (high blood pressure), tobacco smoke, Diabetes Mellitus, Obesity, inactive lifestyle, age - a family history of heart disease is also a risk factor and the one which cannot be controlled.

**Unhealthy blood cholesterol levels** - this includes high LDL cholesterol (sometimes called bad cholesterol) and low HDL cholesterol (sometimes called good cholesterol).

**High blood pressure** - blood pressure is considered high if it stays at or above 140/90 mmHg over a period of time.

Smoking - this can damage and tighten blood vessels, raise cholesterol levels, and raise blood pressure - smoking also doesn't allow enough oxygen to reach the body's tissues.

**Insulin resistance** - Insulin is a hormone that helps move blood sugar into cells where it's used and insulin resistance occurs when the body cannot use its own insulin properly.

**Diabetes** - this is a disease in which the body's blood sugar level is high because the body doesn't make enough insulin or does not use its insulin properly.

**Overweight or obesity** - overweight is having extra body weight from muscle, bone, fat, and/or water - obesity is having a high amount of extra body fat.

**Lack of physical activity** - lack of activity can worsen other risk factors for atherosclerosis.

Age - as the body ages the risk for atherosclerosis increases and genetic or lifestyle factors cause plaque to gradually build in the arteries - by middle-age or older, enough plaque has built up to cause signs or symptoms, in men, the risk increases after age 45, while in women, the risk increases after age 55. Family history of early heart disease - the risk for atherosclerosis increases if a father or a brother was diagnosed with heart disease before 55 years of age, or if a mother or a sister was diagnosed with heart disease before 65 years of age but though age and a family history of early heart disease are risk factors, it does not mean that you will develop atherosclerosis if you have one or both. Making lifestyle changes and/or taking medicines to treat other risk factors can often lessen the genetic influences and prevent atherosclerosis from developing, even in older adults.

Scientists continue to study other possible risk factors for atherosclerosis and have found that high levels of a protein called C-

reactive protein (CRP) in the blood may raise the risk for atherosclerosis and heart attack - high levels of CRP are proof of inflammation in the body which is the body's response to injury or infection - damage to the arteries' inner walls appears to trigger inflammation and help plaque grow. People with low CRP levels may get atherosclerosis at a slower rate than people with high CRP levels and research is currently under way to establish whether reducing inflammation and lowering CRP levels also can reduce the risk of atherosclerosis. High levels of fats called triglycerides in the blood also may raise the risk of atherosclerosis, particularly in women. Other factors that affect atherosclerosis other risk factors also may raise your risk for developing atherosclerosis include:

Sleep apnoea - a disorder in which the breathing stops or gets very shallow while a person is sleeping - untreated sleep apnoea can raise the chances of high blood pressure, diabetes, and even a heart attack or stroke. Stress - research shows that the most commonly reported "trigger" for a heart attack is an emotionally upsetting event-particularly one involving anger.

Alcohol - heavy drinking can damage the heart muscle and worsen other risk factors for atherosclerosis - men should have no more than two drinks containing alcohol a day, while women should have no more than one drink containing alcohol a day

The relation between dietary fat and atherosclerosis is a contentious field. The role of dietary oxidized fats / lipid peroxidation (rancid fats) in humans is not clear. Laboratory animals fed rancid fats develop atherosclerosis. Rats fed DHA containing oils experienced marked disruptions to their antioxidant systems, as well as accumulated significant amounts of peroxide in their blood, livers and kidneys. In another study, rabbits fed atherogenic diets containing various oils were found to undergo the greatest amount of oxidative susceptibility of LDL via polyunsaturated oils. In a study involving rabbits fed heated soybean oil, "grossly induced atherosclerosis and marked liver damage were histologically and clinically demonstrated". Rancid fats and oils taste very bad even in small amounts; people avoid eating them. It is very difficult to measure or estimate the actual human consumption of these substances.

### **Management, dietary and life style**

Good nutrition is important for your heart health. Healthy eating habits will help you control some of your risk factors for heart disease. Eat plenty of fruits, vegetables, and whole grains.

Choose lean proteins, such as skinless chicken, fish, and beans. Eat non-fat or low-fat dairy products, such as skim milk and low-fat yogurt.

Avoid foods that contain high levels of sodium (salt). Read food labels. Avoid foods that contain saturated fat and partially hydrogenated or hydrogenated fats. These are unhealthy fats that are often found in fried foods, processed foods, and baked goods.

- Eat fewer foods that contain cheese, cream, or eggs
- Reduce weight if obese.
- Stop smoking and alcohol consumption.
- Exercise regularly.

- Take brisk walks for 40 minutes daily morning.
- Practicing yoga and breathing exercises plays a very important role in lowering the cholesterol levels.
- Do some relaxation techniques like yoga, meditation etc to relieve the stress Change your lifestyle to reduce stress-physical or mental.

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## 9.4 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS

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**Atherosclerosis:** Atherosclerosis is a disease in which plaque builds up inside your arteries. Plaque is a sticky substance made up of fat, cholesterol, calcium, and other substances found in the blood. Over time, plaque hardens and narrows your arteries. That limits the flow of oxygen-rich blood to your body.

Atherosclerosis can lead to serious problems, including

- Coronary artery disease. These arteries supply blood to your heart. When they are blocked, you can suffer angina or a heart attack.
- Carotid artery disease. These arteries supply blood to your brain. When they are blocked you can suffer a stroke.
- Peripheral arterial disease. These arteries are in your arms, legs and pelvis. When they are blocked, you can suffer from numbness, pain and sometimes infections.

Atherosclerosis usually doesn't cause symptoms until it severely narrows or totally blocks an artery. Many people don't know they have it until they have a medical emergency.

A physical exam, imaging, and other diagnostic tests can tell if you have it. Medicines can slow the progress of plaque buildup. Your doctor may also recommend procedures such as angioplasty to open the arteries, or surgery on the coronary or carotid arteries. Lifestyle changes can also help. These include following a healthy diet, getting regular exercise, maintaining a healthy weight, quitting smoking, and managing stress.

Atherosclerosis does not usually produce symptoms until blood circulation becomes restricted or blocked, leading to cardiovascular disease (CVD). The type of cardiovascular disease and its associated symptoms depends on where the blockage occurs. Conditions caused by atherosclerosis include peripheral arterial disease, angina, aneurysm, heart attack and stroke.

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## 9.5 SUMMARY

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Atherosclerosis is the narrowing of arteries due to plaque buildup on the artery walls.

Arteries carry blood from the heart to the rest of the body. They are lined with a thin layer of cells that keeps them smooth and allows blood to flow easily. This is called the endothelium.

Atherosclerosis starts when the endothelium becomes damaged, allowing the harmful type of cholesterol to build up in the artery wall.

The body sends a type of white blood cell to clean up this cholesterol, but, sometimes, the cells get stuck at the affected site.

Over time, plaque can build up, made of cholesterol, macrophages, calcium, and other substances from the blood.

Sometimes, the plaque grows to a certain size and stops growing, causing the individual no problems. However, sometimes, the plaque clogs up the artery, disrupting the flow of blood around the body. This makes blood clots more likely, which can result in life-threatening conditions.

In some cases, the plaque eventually, breaks open. If this happens, platelets gather in the affected area and can stick together, forming blood clots. This can block the artery, leading to life-threatening complications, such as stroke and heart attack.

The condition can affect the entire artery tree, but mainly affects the larger, high-pressure arteries.

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## 9.6 KEY WORDS

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- **HDL:** High Density Lipoprotein
- **LDL:** Low Density Lipoprotein
- **VLDL:** Very Low Density Lipoprotein

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## 9.7 SELF ASSESSMENT QUESTIONS

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### Short Answer Questions

1. List the causes of atherosclerosis.
2. Discuss the symptoms of atherosclerosis.
3. Write a short note on disease progression of atherosclerosis.

### Long Answer Questions

1. Explain the role of dietary and lifestyle management of atherosclerosis.
2. Elaborate the causes, symptoms and clinical findings of atherosclerosis.

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## 9.8 FURTHER READINGS

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## UNIT-X

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### Structure

- 10.1 Introduction
- 10.2 Objectives
- 10.3 . Dietary management in angina pectoris
- 10.4 . Myocardial infarction and cardiac failure
- 10.5 . Hypertension
- 10.6 .Classification
- 10.7 . Causes
- 10.8 . Complications
- 10.9 . Dieatary managemetn
- 10.10 Answers to Check Your Progress Questions
- 10.11 Summary
- 10.12 Key Words
- 10.13 Self-Assessment Questions
- 10.14 Further Readings

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### 10.0 INTRODUCTION

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Dietary management, also known as “foodservice management”, is the practice of providing nutritional options for individuals and groups with diet concerns through supervision of foodservices. Practitioners in dietary management, known as dietary managers, work in hospitals, long-term care facilities, restaurants, school and college cafeterias, correctional facilities, and other foodservice settings, usually implementing meal plans established by a dietitian or nutritionist. They are responsible for supervising the work of other nutrition personnel such as cooks and dietary aides.

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### 10.1 OBJECTIVES

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After studying this unit you will be able to

- Discuss the dietary management of angina pectoris, mycardioa infarction and cardiac failure.
- Describe hypertension and its management.

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### 10.2 DIETARY MANAGEMENT IN ANGINA PECTORIS

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Angina pectoris is the medical term for chest pain or discomfort due to coronary heart disease. It occurs when the heart muscle doesn't get as much blood as it needs. This usually happens because one or more of the heart's arteries is narrowed or blocked, also called ischemia.

Angina usually causes uncomfortable pressure, fullness, squeezing or pain in the center of the chest. You may also feel the discomfort in your neck, jaw, shoulder, back or arm

Angina often occurs when the heart muscle itself needs more blood than it is getting, for example, during times of physical activity or strong emotions. Severely narrowed arteries may allow enough blood to reach the heart when the demand for oxygen is low, such as when you're sitting. But, with physical exertion—like walking up a hill or climbing stairs—the heart

works harder and needs more oxygen. Cut down salt (common table salt) intake in your diet to avoid hypertension. Intake should not exceed more than 2 to 2.5 gm a day. . Avoid foods rich in cholesterol: ground nut oil, ghee, butter, whole milk, eggs, lard (fats derived from pig meat), tallow (fats derived from goat or sheep mutton), oily fish, meats and red meat - all non-vegetarian food contain cholesterol in varied amount. . Avoid non-vegetarian food especially red meat. . Increase intake of foods that contain PUFA's (poly unsaturated fatty acids): flax / linseed oil is the richest source of PUFA's. Other oils that contain PUFA's are sesame oil, mustard oil and olive oil. As all oils are high in fats, its consumption should be limited. The intake of oils should not exceed 20gms a day. Sesame oil and almond oil hinders the absorption of LDL (low-density lipoproteins), so it is advisable to use replace other cooking oils by sesame oil.

Increase your intake of water. Consume whole grain cereals and whole pulses. Take high fiber diet - whole grains, bran, oat, green leafy vegetables, peas, beans, potatoes, raw vegetables, salads, dried fruits and fresh fruits. Eat fruits and vegetables with the skin. Cut down the intake of sugar and other sweeteners. Replace whole milk with semi-skimmed or skimmed milk. Avoid fat-rich portions of flesh foods. Instead have lean meat. Avoid fat-rich desserts and fried snacks like cakes, pastries, puddings etc. Instead of frying food boil, steam, grill or roast food. Avoid coffee and caffeinated drinks, junk food - pizza, burgers etc. Consume food rich in magnesium: Nuts and sea food, green leafy vegetables, sea plants like Japanese sea plants, whole grains Peas, lotus stem, pulses, legumes, and oil seeds. Increase intake of vitamin C, it maintains the elasticity and integrity of the artery walls. Citrus fruits, green leafy vegetables. Eat vegetables- cabbage, broccoli, tubers- potatoes, and sweet potatoes. Reduce calcium intake (reduce but do not completely avoid calcium as it is needed for maintenance of our body):- milk and sea food, nuts, green leafy vegetables, whole grains, peas, lotus stem, pulses, legumes and oil seeds - Should avoid custard apple and banana as they are high in calcium.

### **10.3. MYOCARDIAL INFARCTION AND CARDIAC FAILURE**

Myocardial infarction (MI) (ie, heart attack) is the irreversible necrosis of heart muscle secondary to prolonged ischemia. Patients with typical myocardial infarction may have the following prodromal symptoms in the days preceding the event. Myocardial infarction, commonly known as a heart attack, is the irreversible necrosis of heart muscle secondary to prolonged ischemia. This usually results from an imbalance in oxygen supply and demand, which is most often caused by plaque rupture with thrombus formation in a coronary vessel, resulting in an acute reduction of blood supply to a portion of the myocardium.

Myocardial infarction is considered part of a spectrum referred to as acute coronary syndrome (ACS). The ACS continuum representing ongoing myocardial ischemia or injury consists of unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). Patients with ischemic discomfort may or may not have ST-segment or T-wave changes denoted on the electrocardiogram (ECG). ST elevations seen on the ECG reflect active and ongoing transmural myocardial injury. Without immediate reperfusion therapy, most persons with STEMI develop Q waves,



NOTES

reflecting a dead zone of myocardium that has undergone irreversible damage and death. Those without ST elevations are diagnosed either with unstable angina or NSTEMI—differentiated by the presence of cardiac enzymes. Both these conditions may or may not have changes on the surface ECG, including ST-segment depression or T-wave morphological changes. Myocardial infarction may lead to impairment of systolic or diastolic function and to increased predisposition to arrhythmias and other long-term complications. Coronary thrombolysis and mechanical revascularization have revolutionized the primary treatment of acute myocardial infarction, largely because they allow salvage of the myocardium when implemented early after the onset of ischemia. (See Treatment Strategies and Management.) The modest prognostic benefit of an opened infarct-related artery may be realized even when recanalization is induced only 6 hours or more after the onset of symptoms, that is, when the salvaging of substantial amounts of jeopardized ischemic myocardium is no longer likely. The opening of an infarct-related artery may improve ventricular function, collateral blood flow, and ventricular remodeling, and it may decrease infarct expansion, ventricular aneurysm formation, left ventricular dilatation, late arrhythmia associated with ventricular aneurysms, and mortality

- Fatigue
- Chest discomfort
- Malaise Typical chest pain in acute myocardial infarction has the following characteristics:
  - Intense and unremitting for 30-60 minutes
  - Retrosternal and often radiates up to the neck, shoulder, and jaw and down to the ulnar aspect of the left arm
  - Usually described as a substernal pressure sensation that also may be characterized as squeezing, aching, burning, or even sharp
  - In some patients, the symptom is epigastric, with a feeling of indigestion or of fullness and gas The patient's vital signs may demonstrate the following in myocardial infarction:
    - The patient's heart rate is often increased secondary to sympathoadrenal discharge
    - The pulse may be irregular because of ventricular ectopy, an accelerated idioventricular rhythm, ventricular tachycardia, atrial fibrillation or flutter, or other supraventricular arrhythmias; bradyarrhythmias may be present
    - In general, the patient's blood pressure is initially elevated because of peripheral arterial vasoconstriction resulting from an adrenergic response to pain and ventricular dysfunction
    - However, with right ventricular myocardial infarction or severe left ventricular dysfunction, hypotension is seen
    - The respiratory rate may be increased in response to pulmonary congestion or anxiety
    - Coughing, wheezing, and the production of frothy sputum may occur
    - Fever is usually present within 24-48 hours, with the temperature curve generally parallel to the time course of elevations of creatine kinase (CK) levels in the blood. Body temperature may occasionally exceed 102°F

## **10.4. HYPERTENSION**

Hypertension (HTN or HT), also known as high blood pressure (HBP), is a long-term medical condition in which the blood pressure in the arteries is persistently elevated.<sup>[10]</sup> High blood pressure typically does not cause symptoms.<sup>[1]</sup> Long-term high blood pressure, however, is a major risk factor for coronary artery disease, stroke, heart failure, atrial fibrillation, peripheral arterial disease, vision loss, chronic kidney disease, and dementia.

Hypertension is classified as either primary (essential) high blood pressure or secondary high blood pressure. About 90–95% of cases are primary, defined as high blood pressure due to nonspecific lifestyle and genetic factors.<sup>[5][6]</sup> Lifestyle factors that increase the risk include excess salt in the diet, excess body weight, smoking, and alcohol use.<sup>[1][5]</sup> The remaining 5–10% of cases are categorized as secondary high blood pressure, defined as high blood pressure due to an identifiable cause, such as chronic kidney disease, narrowing of the kidney arteries, an endocrine disorder, or the use of birth control pills.

Blood pressure is expressed by two measurements, the systolic and diastolic pressures, which are the maximum and minimum pressures, respectively. For most adults, normal blood pressure at rest is within the range of 100–130 millimeters mercury (mmHg) systolic and 60–80 mmHg diastolic. For most adults, high blood pressure is present if the resting blood pressure is persistently at or above 130/80 or 140/90 mmHg. Different numbers apply to children. Ambulatory blood pressure monitoring over a 24-hour period appears more accurate than office-based blood pressure measurement.

Lifestyle changes and medications can lower blood pressure and decrease the risk of health complications. Lifestyle changes include weight loss, physical exercise, decreased salt intake, reducing alcohol intake, and a healthy diet. If lifestyle changes are not sufficient then blood pressure medications are used. Up to three medications can control blood pressure in 90% of people. The treatment of moderately high arterial blood pressure (defined as >160/100 mmHg) with medications is associated with an improved life expectancy. The effect of treatment of blood pressure between 130/80 mmHg and 160/100 mmHg is less clear, with some reviews finding benefit and others finding unclear benefit. High blood pressure affects between 16 and 37% of the population globally. In 2010 hypertension was believed to have been a factor in 18% of all deaths (9.4 million globally).

## **10.5. CLASSIFICATION**

Hypertension is a major health problem throughout the world because of its high prevalence and its association with increased risk of cardiovascular disease. Advances in the diagnosis and treatment of hypertension have played a major role in recent dramatic declines in coronary heart disease and stroke mortality in industrialized countries. The continuous relationship between the level of blood pressure and cardiovascular risk makes any numerical definition and classification of hypertension somewhat arbitrary. Therefore, a definition of hypertension is usually taken as that level of arterial blood pressure associated with doubling of long-term cardiovascular risk.

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The classification is based on the mean of two or more properly measured seated blood pressure readings on two or more office visits.

Normal blood pressure is defined as levels <120/80 mmHg. Systolic blood pressure of 120–139 mmHg or diastolic blood pressure 80–89 mmHg is classified as prehypertension. These patients are at increased risk for progression to hypertension. Hypertension is defined as systolic blood pressure  $\geq 140$  mmHg or diastolic blood pressure  $\geq 90$  mmHg.

Hypertension is divided into two stages.

- Stage 1 includes patients with systolic blood pressure 140–159 mmHg or diastolic blood pressure 90–99 mmHg.
- Stage 2 includes patients with systolic blood pressure  $\geq 160$  mmHg or diastolic blood pressure  $\geq 100$  mmHg.

Isolated systolic hypertension is defined as systolic blood pressure  $\geq 140$  mmHg and diastolic blood pressure <90 mmHg. Accelerated hypertension is characterized by markedly elevated blood pressure (diastolic blood pressure usually >120 mmHg) associated with retinal haemorrhage and exudates. If untreated, it commonly progresses to malignant hypertension, which is characterized by papilloedema. Both accelerated and malignant hypertensions are associated with widespread degenerative changes in the walls of resistance vessels including hypertensive encephalopathy, haematuria, and renal dysfunction.

A more elaborate classification of blood pressure is provided by the European Society of Hypertension and the European Society of Cardiology (ESH/ESC) (Table 1). The diagnosis of hypertension in adults is made when the average of two or more diastolic blood pressure measurements on at least two subsequent visits is  $\geq 90$  mmHg, or when the average of multiple systolic blood pressure readings on two or more subsequent visits is  $\geq 140$  mmHg. Patients should be clearly informed that a single elevated reading does not constitute a diagnosis of hypertension but is a sign that further observation is required.

**Classification of blood pressure for adults (Table 1)**

Category	Systolic BP (mmHg)	Diastolic BP (mmHg)
Optimal	<120	<80
Normal	120–129	80–84
High normal	130–139	85–89
Grade 1 hypertension (mild)	140–159	90–99
Grade 2 hypertension (moderate)	160–179	100–109
Grade 3 hypertension (severe)	$\geq 180$	$\geq 110$
Isolated systolic hypertension	$\geq 140$	<90

**Causes, Complications and dietary management**

**10.6. CAUSES**

*Stress can increase the risk of high blood pressure.* The cause of hypertension is often not known. In many cases, it is the result of an underlying condition. Doctors call high blood pressure that is not due to another condition or disease primary or essential hypertension. If an underlying condition is the cause of increasing blood pressure, doctors call

this secondary hypertension. Primary hypertension can result from multiple factors, including:

- blood plasma volume
- hormone activity in people who manage blood volume and pressure using medication
- environmental factors, such as stress and lack of exercise

Secondary hypertension has specific causes and is a complication of another health problem.

Chronic kidney disease (CKD) is a common cause of high blood pressure, as the kidneys no longer filter out fluid. This excess fluid leads to hypertension.

Conditions that can lead to hypertension include:

- Diabetes, due to kidney problems and nerve damage
- Kidney disease
- Pheochromocytoma, a rare cancer of an adrenal gland
- Cushing syndrome that corticosteroid drugs can cause
- Congenital adrenal hyperplasia, a disorder of the cortisol-secreting adrenal glands
- hyperthyroidism, or an overactive thyroid gland
- Hyperparathyroidism, which affects calcium and phosphorous levels
- Pregnancy
- Sleep apnea
- obesity

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## 10.7. RISK FACTORS

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A number of factors increase the risk of hypertension.

- **Age:** Hypertension is more common in people who are more than 60 years of age. Blood pressure can increase steadily with age as the arteries stiffen and narrow due to plaque buildup.
- **Ethnicity:** Some ethnic groups are more prone to hypertension than others. African Americans have a higher risk than other ethnic groups, for example.
- **Size and weight:** Being overweight or obese is a primary risk factor.
- **Alcohol and tobacco use:** Regularly consuming large quantities of alcohol or tobacco can increase blood pressure.
- **Sex:** According to a 2018 review, males have a higher risk of developing hypertension than females. However, this is only until after women reach menopause.
- **Existing health conditions:** Cardiovascular disease, diabetes, chronic kidney disease, and high cholesterol levels can lead to hypertension, especially as people age.

Other risk factors include:

- sedentary lifestyle
- salt rich, high fat diet
- low potassium intake

Poorly managed stress and a family history of high blood pressure can also contribute to the risk of developing hypertension

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## 10.8. MANAGEMENT

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**Regular physical exercise**

*Self-Instructional Material*

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People can measure blood pressure using a sphygmomanometer.

Current guidelines recommend that all people, including those with hypertension, engage in at least 150 minutes of moderate intensity, aerobic exercise every week, or 75 minutes a week of high intensity exercise.

**Stress reduction**

Avoiding or learning to manage stress can help a person control blood pressure.

Meditation, warm baths, yoga, and simply going on long walks are relaxation techniques that can help relieve stress.

People should avoid consuming alcohol, recreational drugs, tobacco, and junk food to cope with stress, as these can contribute to elevated blood pressure and the complications of hypertension.

Smoking can increase blood pressure. Avoiding or quitting smoking reduces the risk of hypertension, serious heart conditions, and other health issues.

**Medication**

People can use specific medications to treat hypertension. Doctors will often recommend a low dose at first. Antihypertensive medications will usually only have minor side effects.

Eventually, people with hypertension will need to combine two or more drugs to manage their blood pressure.

Medications for hypertension include:

- Diuretics, including thiazides, chlorthalidone, and indapamide
- Beta-blockers and alpha-blockers
- Calcium-channel blockers
- Central agonists
- Peripheral adrenergic inhibitor
- Vasodilators
- Angiotensin-converting enzyme (ace) inhibitors
- Angiotensin receptor blockers

The choice of medication depends on the individual and any underlying medical conditions they may experience.

Anyone on antihypertensive medications should carefully read the labels of any over-the-counter (OTC) drugs they may also take, such as decongestants. These OTC drugs may interact with the medications they are taking to lower their blood pressure.

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**10.9. DIET MANAGEMENT**

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People can prevent high blood pressure by following a heart-healthy diet.

**Reducing salt intake**

People's average salt intake is between 9 grams (g) and 12 g per day in most countries around the world.

The World Health Organization (WHO) recommend reducing intake to under 5 g a day to help decrease the risk of hypertension and related health problems.

Lowering salt intake can benefit people both with and without hypertension.

**Moderating alcohol consumption**

Moderate to excessive alcohol consumption can increase blood pressure.

### **Eating more fruit and vegetables and less fat**

People who have high blood pressure or people at high risk of developing high blood pressure should eat as little saturated and total fat as possible.

Instead, experts recommend:

- whole grain, high fiber foods
- a variety of fruit and vegetables
- beans, pulses, and nuts
- fish rich in omega-3 twice a week
- nontropical vegetable oils, for example, olive oil
- skinless poultry and fish
- low fat dairy products

It is important to avoid trans fats, hydrogenated vegetable oils, and animal fats, as well as large portion sizes.

Some fats, such as those in oily fish and olive oil, have protective effects on the heart. However, these are still fats. While they are typically healthful, people with a risk of hypertension should still include them in their total fat intake.

### **Managing body weight**

Excess body weight can contribute to hypertension. A fall in blood pressure usually follows weight loss, as the heart does not have to work so hard to pump blood around the body.

A balanced diet with a calorie intake that matches the individual's size, sex, and activity level will help.

### **The DASH diet**

The U.S. National Heart, Lung, and Blood Institute (NHLBI) recommend the DASH diet for people with high blood pressure. DASH stands for "Dietary Approaches to Stop Hypertension."

DASH is a flexible and balanced eating plan with a firm grounding in research by the NHLBI who advise that the diet:

- Lowers high blood pressure
- Improves levels of fats in the bloodstream
- Reduces the risk of cardiovascular disease

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## **10.10 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS**

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Much of the disease burden of high blood pressure is experienced by people who are not labeled as hypertensive. Consequently, population strategies are required to reduce the consequences of high blood pressure and reduce the need for antihypertensive medications. Lifestyle changes are recommended to lower blood pressure, before starting medications. The 2004 British Hypertension Society guidelines proposed lifestyle changes consistent with those outlined by the US National High BP Education Program in 2002 for the primary prevention of hypertension:

- Maintain normal body weight for adults (e.g. Body mass index 20–25 kg/m<sup>2</sup>)

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- Reduce dietary sodium intake to <100 mmol/ day (<6 g of sodium chloride or <2.4 g of sodium per day)
- Engage in regular aerobic physical activity such as brisk walking ( $\geq 30$  min per day, most days of the week)
- Limit alcohol consumption to no more than 3 units/day in men and no more than 2 units/day in women
- Consume a diet rich in fruit and vegetables (e.g. At least five portions per day)

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## 10.11 SUMMARY

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Elevated blood pressure arises from a combination of environmental and genetic factors and the interactions of these factors. A substantial body of evidence from animal studies, epidemiologic studies, meta-analyses, and randomized controlled trials has demonstrated that certain dietary patterns and individual dietary elements play a prominent role in the development of hypertension. Changes in diet can lower blood pressure, prevent the development of hypertension, and reduce the risk of hypertension-related complications. Dietary strategies for the prevention of hypertension include reducing sodium intake, limiting alcohol consumption, increasing potassium intake, and adopting an overall dietary pattern such as the DASH (Dietary Approaches to Stop Hypertension) diet or a Mediterranean diet. In order to reduce the burden of blood pressure-related complications, efforts that focus on environmental and individual behavioral changes that encourage and promote healthier food choices are warranted.

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## 10.12. KEY WORDS

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- **FAO** : Food and Agricultural Organization
- **WHO** : World Health Organization
- **UNICEF** : United Nations International Children Emergency Fund
- **CARE** : Cooperative American Relief Everywhere
- **AFPRO** :Action for Food Production
- **CWS** : Church World Service

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## 10.13 SELF-ASSESSMENT QUESTIONS

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### Short Answer Questions

1. Discuss the dietary management in angina pectoris.
2. Explain the myocardial infarction and cardiac failure.

### Long Answer Questions

1. Explain hypertension and its causes and complications.
2. Discuss the dietary management of hypertension.

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## 10.14 FURTHER READINGS

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- Suryatapas, 2018, Textbook of Community Nutrition, 3<sup>rd</sup> Edition, Academic Publishers, West Bengal.
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# **BLOCK-IV-DIETETICS IN CANCER, DISEASE OF NERVOUS SYSTEM AND MUSCULO SKELETAN SYSTEM**

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## **Structure**

- 11. Introduction
  - 11.1. Objectives
  - 11.2. Classification
  - 11.3. Development of cancer
  - 11.4. Risk factors
    - 11.4.1. Environmental
    - 11.4.2. Hereditary
    - 11.4.3. Nutritional
  - 11.5. Answers to Check Your Progress Questions
  - 11.6. Summary
  - 11.7. Key Words
  - 11.8. Self-Assessment Questions
  - 11.9 Further Readings

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## **11.0 INTRODUCTION**

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Cancer occurs when cells become abnormal and keep dividing without control or order. Most cancers are named for the type of cell or organ in which they begin. Screening for cancer includes physical examination, laboratory tests and procedures, and the use of imaging modalities to look at internal organs. The most common detection and diagnostic tools are CT (or CAT) scans, MRI, ultrasonography, endoscopy, and biopsy. Common tests include blood and urine tests, Pap smears, mammograms, fecal occult blood, and others as needed. Following the results of the screening, a determination is made of the size and extent of the cancer, and a treatment plan is developed. This process is called staging. Tolerance and response to therapy. Individuals who do not lose weight have significantly longer survival time than those who do. Malnourished individuals are most susceptible to infection and less likely to tolerate or derive optimal benefits from therapy. Malnutrition is also an important issue in the quality of life of individuals diagnosed with cancer. Many studies indicate that more cancer patients die of malnutrition than from the disease. Cancer leads to malnutrition on the disease prognosis, progression, response to therapy and reduces the quality of life. Death in the individual Cancer is correlated with the degree of loss of lean body mass, and sustained weight loss is a predictor of progression of disease. There are myriad nutritional and metabolic changes characteristic of cancer. These changes are directly related to the body's response to the disease, treatment methods, surgical procedures, and psychological and emotional responses of the individual. A number of emotional factors contribute to nutritional status, such as depression, guilt, fear, denial, pain, conditioned aversions, and reaction to drugs. Loss of independence creates a major trauma. Formidable

challenges face care providers and caregivers of individuals who have cancer. This chapter deals with the nutritional aspects of care.

Depending upon the site of the cancer, digestion and absorption of nutrients may be disturbed (e.g. owing to diarrhea, malabsorption). Additionally, their metabolism may be altered. Proteolysis and lipolysis are accelerated, while muscle protein synthesis is depressed, resulting in a loss of lean body mass and fat tissue. Additionally, carbohydrate metabolism is modified by tumour growth (e.g. the hepatic glucose production and Cori cycle activity are increased while the insulin sensitivity of peripheral tissues is reduced). These alterations contribute to an increase in energy expenditure and may result in progressive wasting. However, in spite of hypermetabolism and weight loss (exacerbated by stress, pain, infection, surgical procedures), patients' food intake is usually not increased, thus further promoting wasting. Furthermore, cancer cachexia – characterized by weight loss, reduction of fat and lean body masses, anorexia with reduced food intake, early satiety, dysgeusia, dysphagia, fatigue, anemia, hypoalbuminaemia, progressive debilitation – also deteriorates nutritional status. Nonetheless, besides the physical effects of cancer, patients are often suffering from psychological disturbances as well, and particularly depression, whereas oncology treatment such as surgery, chemotherapy and radiotherapy produce additional acute and chronic symptoms which increase energy expenditure and jeopardize food intake (e.g. by changes in taste, dry mouth, nausea, vomiting) and nutritional status.

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## 11.1 OBJECTIVES

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After studying this unit you will be able to:

- Define cancer.
- Explain the risk factors of cancer.

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## CANCER

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Cancer is a term used to refer malignant neoplasm or tumors. Neoplasm means cell is in a tissue proliferate without the normal controls on growth. In malignant neoplasm the cells spread to adjacent tissues and interfere with the function and often has undesirable systemic effects.

Cancer is the most serious disease among chronic disorder and is the second leading cause of death in most developing and developed countries. Cancer is the major public health challenge to scientist and public health personnel now a day.

The term cancer is a disease in which abnormal cell divide without control. In simple words it is defined as uncontrolled growth of cells. These cells has the ability to multiply and spread without restraint, that these cancer cells can invade nearby tissues and can spread through the blood stream and lymphatic system to other parts of the body.

The extra cells form a mass of tissues called a growth or tumor. These tumors may be benign (not cancerous) or malignant (cancerous).

Benign tumors can be often removed, and it cannot re-grow. Cells from benign tumor do not spread to other parts of the body, but rarely pose a threat to life.

*Self-Instructional Material*

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Malignant tumor are cancer cells, divide abnormally, invade and damage nearby tissues and organs , when cancer cells breaks away from the malignant tumor, enters into blood stream or the lymphatic system by which cancer spreads into new organ and develops causing cancer. Thus, the spread of cancer is called as metastasis.

Cancer, abnormal division and reproduction of cells that can spread throughout the body, is usually thought of as a single disease but consists of almost 100 disorders caused by nearly 300 different growths. Carcinogenesis the origin or development of cancer is thought to be a biologic, multistage process that proceeds on a continuum but is often described in three progressive phases: initiation, promotion, and tumor progression.

Benign tumors represent the accumulation of cells which have been transformed to reproduce Cancer is caused by mutation or abnormal activation of cellular genes that control cell grow and cell mitosis. The abnormal genes are called oncogenes It is now recognized that there exists regulatory genes known as tumour suppressor genes whose normal function is to prevent malignant transformation. The latter might be triggered by exposure to a carcinogen (eg: tobacco or by a spontaneous mutation Conversely normal cells contain growth promoting genes known as proto-oncogenes where inappropriate activation (by a carcinogen or mutation) to produce oncogenes that could result in malignant transformation. The probability of mutations can be increased manifold when a person is exposed to certain chemical, physical or biological factors

Cancer can occur at any age and in any part of the body in abnormal numbers but remain within the tissue of origin. Cancer is not a single disorder. There are many different kinds of cancers which take different courses and require different treatments Among the various cancers, the uterine cervix, breast, oro-pharyngeal, large bowel (colorectal), stomach, ovarian oesophageal, endometrium and prostate gland are the most likely to be influenced by diet These are also common type of cancers About 1/3 of all cancers are directly or indirectly related to diet.

India is undergoing demographic, epidemiological and nutrition transition fuelling an epidemic of chronic diseases and obesity especially in urban areas Some of these may lead to cancer The nutritional transition to a more 'western diet would in future alter the frequencies of the organ affected with prostate and colon being more frequent in men and breast, uterine and colon being more frequent in women.

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## **11.2. CLASSIFICATION OF CANCER**

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Usually cancer are classified in two ways, by the tissue in which the cancer originates (histological types)

By primary site or the location in the body where the cancer developed

Thus the cancer is classified majorly into 6 types, carcinoma, sarcoma, myeloma, leukemia, lymphoma and mixed types.

### **i) CARCINOMA**

Carcinoma refers to malignant neoplasm of epithelial origin or cancer of the internal or external lining of the body. Epithelial tissues are found throughout the body. It is present in skin, as well as the covering and lining of organs and internal passage ways, such as the gastrointestinal tract.

Carcinoma are divided into two major subtypes;  
Adenocarcinoma -- develops in an organ or gland  
Squamous cell carcinoma -- which originates in the squamous epithelium

Adenocarcinoma generally occur in mucus membrane, and are first seen as a thickened plaque like white mucosa, are often spread through the soft tissues

Squamous cell carcinoma occurs in the many areas of the body. Mostly carcinoma affects organs or glands capable of secretion, such as the breast which produce milk or the lungs, which secretes mucus, or colon or prostate or bladder.

## ii) **SARCOMA**

Sarcoma refers to cancer that originates in supportive and connective tissues such as bones, tendons, cartilage, muscle and fat. Mostly sarcoma develops as a painful mass on the bone. Sarcoma tumors usually resemble the tissues in which they grow.

Examples of sarcoma are;

- Osteosarcoma (bones)
- Chondrosarcoma (cartilage)
- Leiomyosarcoma (smooth muscle)
- Rhabdomyosarcoma (skeletal muscle)
- Mesothelioma (membranous lining of body cavities)
- Fibrosarcoma (fibrous tissues)
- Angiosarcoma
- Liposarcoma (adipose tissues)
- Glioma (neurogenic connective tissues found in brain)
- Myxosarcoma (primitive embryonic connective tissue)
- Mesodermal tumor (mixed connective tissues types)

## iii) **MYELOMA**

It is the cancer that originates in the plasma cells of bone marrow. The plasma cells produce some of the proteins found in blood.

## iv) **LEUKEMIA**

Leukemia's (liquid cancer or blood cancer) are cancers of the bone marrow (the site of blood cell production). The disease is often associated with overproduction of immature, WBC. These immature WBC do not perform but it also affects red blood cells and can cause poor blood clotting and fatigue due to anemia.

Leukemia includes,

- Myelogenous or granulocytic leukemia - Malignancy of the myeloid and granulocytic WBC series.
- Lymphatic, lymphocytic or lymphoblastic leukemia- Malignancy of the lymphoid and lymphocytic blood cell series
- Polycythemia vera or erythemia- Malignancy of various blood cells, but red cells predominating.

## v) **LYMPHOMA**

It develops in the gland or nodes of the lymphatic system, a network of vessels, nodes, organs (specifically the spleen, tonsils and thymus) that purify bodily fluids and produce infection-fighting WBC or lymphocytes.

Lymphoma are also called as solid cancer.

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Lymphoma may occur in specific organs such as stomach, breast or brain. These lymphomas are referred to as extra nodal lymphoma. Hodgkin lymphoma and non-Hodgkin lymphoma, the presence of Reed-Sternberg cells in Hodgkin lymphoma from non-Hodgkin lymphoma.

### **MIXED TYPES**

The type components may be within one category or different categories. Some examples are adenosquamous carcinoma, mixed mesodermal tumor, carcinoma sarcoma and teratocarcinoma.

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## **11.3. DEVELOPMENT OF CANCER**

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Cancer is usually by mutation or abnormal activation of cellular genes that control cell growth and cell mitosis. Thus, the abnormal genes are called as oncogenes and there exist the regulatory genes known as tumor suppressor genes, whose normal function is to prevent malignant transformation.

Thus, the cancer pathogenesis was studied using a model as mouse skin carcinogenesis. There are three stages that are involved in cancer development as initiators, promoters, and progressors.

The initial experimental studies of carcinogenesis were conducted in animals. Chemicals able to react with DNA and non-reactive compounds were both tested for their ability to cause cancer. The model used was mouse skin carcinogenesis. In this system, researchers painted test chemicals on the skin and observed the growth of tumours. Researchers found that application of a DNA-reactive substance only resulted in tumour formation when the animals were further treated with another non-reactive substance.

A compound that reacts with DNA and somehow changes the genetic makeup of the cell is called a mutagen. The mutagens that predispose cells to develop tumours are called initiators and the non-reactive compounds that stimulate tumour development are called promoters. Approximately 70% of known mutagens are also carcinogens—cancer-causing compounds. A compound that acts as both an initiator and a promoter is referred to as a 'complete carcinogen' because tumour development can occur without the application of another compound.

### **Initiation**

Initiation is the first step in the two-stage model of cancer development. Initiators, if not already reactive with DNA, are altered (frequently they are made electrophilic) via drug-metabolizing enzymes in the body and are then able to cause changes in DNA (mutations).

Since many initiators must be metabolized before becoming active, initiators are often specific to particular tissue types or species. The effects of initiators are irreversible; once a particular cell has been affected by an initiator, it is susceptible to promotion until its death. Since initiation is the result of permanent genetic change, any daughter cells produced from the division of the mutated cell will also carry the mutation.

In studies of mouse skin carcinogenesis, a linear relationship has been observed between the dose of initiator and the quantity of tumors that can be produced, thus any exposure to the initiator increases risk and this risk increases indefinitely with higher levels of exposure.

### **Promotion**

Once a cell has been mutated by an initiator, it is susceptible to the effects

of promoters. These compounds promote the proliferation of the cell, giving rise to a large number of daughter cells containing the mutation created by the initiator.

Promoters have no effect when the organism in question has not been previously treated with an initiator.

Unlike initiators, promoters do not covalently bind to DNA or macromolecules within the cell. Many bind to receptors on the cell surface in order to affect intracellular pathways that lead to increased cell proliferation.

There are two general categories of promoters: specific promoters that interact with receptors on or in target cells of defined tissues and nonspecific promoters that alter gene expression without the presence of a known receptor.

Promoters are often specific for a particular tissue or species due to their interaction with receptors that are present in different amounts in different tissue types.

While the risk of tumor growth with promoter application is dose-dependent, there is both a threshold and a maximum effect of promoters. Very low doses of promoters will not lead to tumor development and extremely high doses will not produce more risk than moderate levels of exposure.

### **Progression**

In mice, repeated promoter applications on initiator-exposed skin produces benign papillomas. Most of these papillomas regress after treatment is stopped, but some progress to cancer.

The frequency of progression suggests that the papillomas that progress to cancer have acquired an additional, spontaneous, mutation. The term progression, coined by Leslie Foulds, refers to the stepwise transformation of a benign tumour to a neoplasm and to malignancy.

Progression is associated with a karyotypic change since virtually all tumours that advance are aneuploid (have the wrong number of chromosomes). This karyotypic change is coupled with an increased growth rate, invasiveness, metastasis and an alteration in biochemistry and morphology.

From there progression, the phase in which tumor cells aggregate and grow, proceeds, leading eventually to a fully malignant neoplasm or a tumor with the capacity for tissue invasion that may eventually spread to distant tissues and organs, a process known as metastasis.

The classification of tumors is based on their tissue of origin, their growth properties, and their invasion of other tissues. Tumors that are not malignant are typically described as benign. Because cancer occurs in cells that are replicating, the patterns of cancer are quite different in children and adults. In early life the brain, nervous system, bones, muscles, and connective tissue are still growing. Thus in children these tissues are more commonly involved with cancerous lesions than they are in adults. Conversely, common adult tumors involve epithelial linings. Leukemias and lymphomas which are cancers of the immune system, occur in both children and adults.

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## **11.4. RISK FACTORS**

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Two components are inevitably involved in the etiology of cancer. They are genetic and environment factors. In the vast majority of the population, the two have to synergies to induce cancer. Different cancer have different risk factors.

### **11.4.1. ENVIRONMENT FACTORS**

There are many environment factors that are responsible for causing cancers, which includes tobacco, alcohol; dietary factors, occupational exposure, viruses,, parasites, lifestyle others such as sunlight, pollution, drugs.

#### **Ionizing radiation**

X-rays, gamma rays and particle radiations from radioactive substances, even ultraviolet rays can predispose to cancer by rupturing DNA strands thus causing mutations.

#### **Chemical Substances**

Chemical substances that can cause mutation are called carcinogens Benzene and asbestos are considered as carcinogens. The carcinogens that cause by tar the greatest number of deaths are those in cigarette smoke Tobacco is the most clearly identified cause of cancer Cancers associated with tobacco are lung mouth larynx, esophagus and bladder According to National Cancer Registry Programme of the Indian Council for Medical Research nearly a third of all cancer incidence in the country are attributable to the use of tobacco Consuming pan with tobacco causes leukoplakia and erythroplakia which are precancerous lesions that occur among 70 per cent of tobacco users Persistent use of such products could also cause genetic deformities Cancers of head and neck correlate strongly with use of alcohol and tobacco.

#### **Tobacco**

Tobacco in various forms of usage can cause lung cancer, and also cancer of larynx, mouth, pharynx, esophagus, bladder pancreas and probably kidney. Cigarette smoking is now responsible for more than million deaths in each year.

#### **Alcohol**

Excess intake of alcohol can cause esophageal and liver cancer. Beer consumption may be associated with rectal cancer. Alcohol contribute to all cancer death.

### **11.4.2. HEREDITY**

In many families there is a strong hereditary tendency to cancer. This probably results from the fact that most cancer require not one mutation but two or more mutation before cancer occurs. In those families that are particularly predisposed to cancer the genes are already mutated in the inherited genome. Breast ovarian and colon caners are mostly familiar.

### **11.4.3. NUTRITIONAL FACTORS**

Foods may cause cancer by being direct carcinogen or carcinogen may be produced cooking. Sometimes microorganisms may produce carcinogens in stored foods. Food stuff may also act as substrate for the formation of carcinogen in the body or food stuff may alter the bacterial origin and affects the bowel thereby producing carcinogen.

## **Meat**

Meat intake has been positively associated with risk of digestive tract cancers and be cancer. Intake of red meat (beef, lamb and pork), and processed meat (ham, salami and bacon increase the risk of colon cancer.

Cancers of prostate are significantly more likely in non vegetarians and frequent beef consumers also have higher risk of bladder cancer. High intake of fish sauces a risk factor for gastric cancer.

Intake of red meat cooked by broiling or barbecuing tend to be positively related to risk of non-Hodgkin's lymphoma. Cooking meat, poultry and fish at high temperatures causes carcinogens to form on food surfaces.

## **Energy balance:**

The relationship between body weight, body mass index or relative body weight and site-specific cancer has been widely investigated and in most epidemiologic studies, a positive association has been seen with cancers of the breast, endometrium, gall bladder and kidney.

In breast cancer, a positive association with weight gain and increased waist/hip ratio is seen in post-menopausal women, especially those who do not use hormone replacement therapy. Physical inactivity, high energy intake and large body mass are associated with increased risk developing colon cancer in men and women.

## **Sugars:**

Consumption of simple sugars (mono and disaccharides) may be related to colorectal cancers. Excessive starch foods may predispose to gastric cancers.

## **Fat:**

Epidemiologic studies have not provided conclusive evidence of an association between dietary and breast cancer. A high intake of saturated fat increases the risk of prostate cancer. Fat intake may be related to colorectal cancer. Intervention studies in connection with heart disease suggest that very high intakes of polyunsaturated fatty acids are associated with increased cancer deaths. Studies have shown that intake of saturated fat tended to be positively related to risk of non-hodgkin's lymphoma.

## **Protein:**

Increased meat intake has been found to be associated with an increased risk of colon Cancer and possibly with advanced prostate cancer.

## **Vitamins and Minerals:**

Low blood carotenoid levels and lung cancer, low dietary vitamin C and oro-pharyngeal, stomach and esophageal cancer and low dietary vitamin E and long, cervix and colorectal cancer have been shown to be related in several studies. Deficiency of folic acid and vitamins A and C have been associated with the development of cervical dysplasia and cervical cancer. Homocysteine levels which increase due to folate deficiency, increases the risk of colon cancers. Selenium and zinc deficiency may also increase the risk of cancer

Studies have shown that people who consume low amounts of fruits and vegetables have more chances of getting cancer.

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#### **11.4.4. OTHER FACTORS**

##### **Nitrates:**

Nitrates are present in a variety of foods, but the main dietary sources are vegetables and drinking water. Sodium and potassium nitrates are used in the processes of salting, pickling and curing foods; they are also added to hot dogs and meat to get pink color. Nitrosamines are present in tobacco and tobacco smoke. Nitrosamines related to nitrates which are potent carcinogens and may cause nasopharyngeal, stomach and colorectal cancers..

##### **Aflatoxins**

The fungi which grow on cereals and groundnuts can cause liver cancer.

##### **Estrogen**

These have been given extensively for the relief are post-menopausal symptoms and for the prevention of osteoporosis. A major uncertainty concerns the role of estrogens in the production of cancer of the breast and endometrial cancer.

##### **Viruses**

Indirect evidence suggests that viruses may act as cofactors in the development of some malignant diseases Epidemiological studies have suggested a possible role for Hepatitis B virus in human primary liver cancer Human papilloma virus and the Epstein Barr virus are considered oncogenic Vaccines for cervical cancer are under trial.

##### **Stress**

Stress may cause damage to the thymus gland and the immune system and hormonal effects mediated through the hypothalamus pituitary and adrenal cortex This Cascade of Physiologic events may provide the neurologic currency that converts anxiety to malignancy Stress may also influence the integrity of the immune system tood behaviour and the nutrition status.

##### **Age**

The risk of developing colorectal cancer (incidence) increases with age The incidence is 6 times higher among persons aged 65 years when compared with those persons aged 40-64 years It has been suggested that the increasing risk of cancer with age reflects the accumulation of critical genetic mutations over a time that ultimately culminate in neoplastic transformation. Factors that have been associated with an increased likelihood of cancer include exposure to exogenous mutagens, altered host immune function and certain inherited genetic syndromes and disorders.

##### **Physical Activity**

Men and women whose lifestyles include regular, vigorous physical activity have the lowest risk of colon cancer. Physical activity may also protect against breast cancer by reducing body weight and the other mechanisms unrelated to body weight.

##### **Immune Factors**

A healthy immune system recognizes foreign cells and destroys them Ineffective immune system may not recognize tumor cells as foreign thus allowing unchecked growth. Ageing affects immune function There is

increased cancer risk when immune system suppressed Patients who take immune suppressants are at risk of developing cancer.

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## **11.4 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS**

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Cancer is a term used to refer malignant neoplasm or tumors Neoplasm means cell is in a tissue proliferate without the normal controls on growth In malignant neoplasm the cells spread to adjacent tissues and interfere with the function and often has undesirable systemic effects.

Cancer is the most serious disease among chronic disorder and is the second leading cause of death in most developing and developed countries. Cancer is the major public health challenge to scientist and public health personnel now a day. Usually cancer are classified in two ways, by the tissue in which the cancer originates (histological types)

By primary site or the location in the body where the cancer developed

Thus the cancer is classified majorly into 6 types, carcinoma, sarcoma, myeloma, leukemia, lymphoma and mixed types. Cancer is usually by mutation or abnormal activation of cellular genes that control cell growth and cell mitosis. Thus, the abnormal genes are called as oncogenes and there exist the regulatory genes known as tumor suppressor genes, whose normal function is to prevent malignant transformation.

Thus, the cancer pathogenesis was studied using a model as mouse skin carcinogenesis. There, are three stages that are involved in cancer development as initiators, promoters and progressors.

Two components are inevitably involved in the etiology of cancer. They are genetic and environment factors. In the vast majority of the population, the two have to synergies to induce cancer. Different cancer have different risk factors.

Foods may cause cancer by being direct carcinogen or carcinogen may be produced cooking. Sometimes microorganisms may produce carcinogens in stored foods. Food stuff may also act as substrate for the formation of carcinogen in the body or food stuff may alter the bacterial origin and affects the bowel thereby producing carcinogen.

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## **11.5 SUMMARY**

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It has been estimated that 30–40 percent of all cancers can be prevented by lifestyle and dietary measures alone. Obesity, nutrient sparse foods such as concentrated sugars and refined flour products that contribute to impaired glucose metabolism (which leads to diabetes), low fiber intake, consumption of red meat, and imbalance of omega 3 and omega 6 fats all contribute to excess cancer risk. Intake of flax seed, especially its lignan fraction, and abundant portions of fruits and vegetables will lower cancer risk. Allium and cruciferous vegetables are especially beneficial, with broccoli sprouts being the densest source of sulforophane. Protective elements in a cancer prevention diet include selenium, folic acid, vitamin B-12, vitamin D, chlorophyll, and antioxidants such as the carotenoids ( $\alpha$ -carotene,  $\beta$ -carotene, lycopene, lutein, cryptoxanthin). Ascorbic acid has limited benefits orally, but could be very beneficial intravenously. Supplementary use of oral digestive enzymes and probiotics also has merit

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as anticancer dietary measures. When a diet is compiled according to the guidelines here it is likely that there would be at least a 60–70 percent decrease in breast, colorectal, and prostate cancers, and even a 40–50 percent decrease in lung cancer, along with similar reductions in cancers at other sites. Such a diet would be conducive to preventing cancer and would favor recovery from cancer as well.

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## 11.6 KEY WORDS

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- **Environmental factor:** An **environmental factor**, ecological **factor** or **eco factor** is any **factor**, abiotic or biotic, that influences living organisms.
- **Hereditary:** the passing on of physical or mental characteristics genetically from one generation to another.

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## 11.7 SELF ASSESSMENT QUESTIONS

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### Short Answer Questions

1. Write a short note on classification of cancer
2. Explain the risk factors of cancer.
3. Give any two risk factors of cancer.

### Long Answer Questions

1. Explain the development of cancer.
2. Discuss the environmental, hereditary and nutritional risk factors of cancer.

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## 11.8 FURTHER READINGS

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- Srilakshmi.B, 2016, Nutrition Science, New Age International Publishers, New Delhi.
- Srilakshmi.B, 2018, Dietetics, New Age International Publishers, New Delhi.

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## UNIT -XII

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### Structure

- 12.0 Introduction
- 12.1 Objectives
- 12.2 Nutritional effects of cancer
  - 12.2.1. Cachexia
  - 12.2.2. Energy metabolism
  - 12.2.3. Substrate metabolism and abnormalities in metabolism
- 12.3. Answers to Check Your Progress Questions
- 13.1 . Summary
- 12.5. Key Words
- 12.6. Self-Assessment Questions
- 12.7. Further Readings

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### 12.0 INTRODUCTION

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Patients undergoing cancer treatment experience a multitude of symptoms that can influence their ability to complete treatment as well as their quality of life during and after treatment. This cross-sectional study sought to describe the dietary changes experienced by cancer patients and to identify associations between these changes and common treatment symptoms. Public health policy with respect to nutrition and cancer should be based on the best available scientific research. In the previous section, we concluded that few dietary effects on cancer risk have been established. Avoiding overweight/obesity, limiting alcohol intake, and increasing physical activity will reduce cancer risk, as will limiting consumption of Chinese-style salted fish and minimising dietary exposure to aflatoxin in populations where these dietary factors are important. In this unit, we shall discuss about cachexia, energy metabolism, substrate metabolism, and abnormalities in metabolism.

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### 12.1 OBJECTIVES

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After studying this unit, you will be able to:

- Explain the nutritional effects of cancer.
- Understand the impacts in cachexia, energy metabolism, substrate metabolism, and abnormalities in metabolism.

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### 12.2 NUTRITIONAL EFFECTS OF CANCER

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The adverse nutritional effects of cancer can be severe and may be compounded by the effects of the treatment regimens and the psychological impact of cancer (Schattner and Shike, 2006). The result is often a profound depletion of nutrient stores. Anorexia, weight loss, and poor nutrition status are found in many individuals at the time of diagnosis, even in children (Goldman et al., 2006). Even small amounts of weight loss (less than 5% of

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body weight) before treatment are associated with a poor prognosis, thus reinforcing the importance of early nutrition assessment and intervention.

### **12.2.1. CACHEXIA**

A common secondary diagnosis in patients with advanced cancer is a variant of protein-energy malnutrition. This syndrome is termed cancer cachexia and is characterized by progressive weight loss, anorexia, generalized wasting and weakness, immune suppression, altered basal metabolic rate, and abnormalities in fluid and energy metabolism. The etiology of this complex metabolic derangement is not entirely understood and can manifest both in individuals with metastatic disease and in individuals with localized disease. Cytokines, protein mediators produced by inflammatory cells through broad physiologic actions, produce metabolic changes and wasting in the tumor-bearing host that are similar but not identical to those seen in sepsis and inflammation (Tisdale, 2003). Cytokines that are thought to play a role in cancer cachexia include tumor necrosis factor (TNF- $\alpha$  and TNF- $\beta$ ) cachectin, interleukin-1, interleukin-6, and interferon- $\alpha$ . These cytokines have overlapping physiologic activities, which makes it likely that no single substance is the sole cause of cancer cachexia. A pool of anticytokine antibodies or other cytokine inhibitors might be considered as a potential intervention for the treatment of cancer cachexia.

The cancer cachexia syndrome, characterized by marked weight loss, anorexia, asthenia and anemia, is invariably associated with the presence and growth of the tumour and leads to a malnutrition status owing to the induction of anorexia or decreased food intake. In addition, the competition for nutrients between the tumour and the host leads to an accelerated starvation state that promotes severe metabolic disturbances in the host, including hypermetabolism, which leads to decreased energetic efficiency. The predominant manifestation of cancer cachexia is the steadily progressive depletion of muscle mass, which is not substantially reversible with any of the currently available nutritional, metabolic or pharmacological approaches. Cancer cachexia mainly results from circulating factors produced by the tumour or by the host's immune system in response to the tumour, such as cytokines released by lymphocytes and/or monocytes/ macrophages. A number of pro-inflammatory cytokines, including interleukin (IL)-1, IL-6, tumour necrosis factor (TNF)- $\alpha$ , interferon (IFN)- $\gamma$  and IFN- $\beta$ , have been implicated in the pathogenesis of cachexia associated with human cancer.

The complications associated with the appearance of the cachexia syndrome affect both the physiological and biochemical balance of the patient and influence the efficiency of anticancer treatment, resulting in a considerably decreased survival time. At the metabolic level, cachexia is associated with the loss of body lipid stores. Alterations in lipid metabolism are partially mediated by changes in circulating hormone concentrations (insulin, glucagon and glucocorticoids, in particular) or in their effectiveness. However, a large number of observations point towards cytokines, polypeptides released mainly by immune cells, as the molecules responsible for the abovementioned metabolic derangements. The most important carbohydrate abnormalities are insulin resistance, increased glucose

synthesis, gluconeogenesis and Cori cycle activity, and decreased glucose tolerance and turnover.

The main pathological changes of protein metabolism include increased protein turnover, muscle catabolism, and liver and tumour protein synthesis, while muscle protein synthesis is decreased. The main abnormalities found in lipid metabolism are enhanced lipid mobilization, decreased lipogenesis, decreased lipoprotein lipase activity, elevated triglycerides and decreased high-density lipoproteins, increased venous glycerol and decreased glycerol clearance from the plasma.

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### **12.2.2. ENERGY METABOLISM**

In chronic starvation the resting energy expenditure (REE) is reduced as the body adapts to conserve energy and preserve body tissue. However, in comparison with control groups, hospitalized cancer patients were reported to be hypometabolic, normometabolic, or hypermetabolic (Knox et al., 1983). The difference in findings is most likely a result of the stages of illness and of nutrition status among the subjects, differing because of methods used in accurately measuring acutely ill individuals. Researchers have found that the site of cancer or tumor does not predictably increase energy needs or REE.

**Protein, fat, and Carbohydrate:**Metabolism Energy metabolism is intimately related to carbohydrate, protein, and lipid metabolism, all of which are altered by tumor growth. Tumors exert a consistent demand for glucose.

Neoplastic cells exhibit a characteristically high rate of anaerobic metabolism and yield lactate as the end product. This expanded lactic acid pool requires an increased rate of host gluconeogenesis via Cori cycle activity, which is increased in some patients with cancer but not in others. Both protein breakdown and lipolysis take place at increasing rates to maintain high rates of glucose synthesis. A relative state of insulin resistance characterized by excess fatty acid oxidation and decreased uptake and use of glucose, especially in muscle, may develop. Alterations in protein metabolism appear to be directed toward providing adequate amino acids for tumor growth. Most notable is the loss of skeletal muscle protein. Other Metabolism Hlpercalcemia can occur in individuals with bone metastases and is caused by the osteolytic activity of tumor cells releasing calcium into the extracellular fluid. Hypercalcemia is a potentially fatal condition and is most commonly seen in metastatic breast cancer and multiple myeloma; symptoms include nausea, weakness, fatigue, lethargy, and confusion.

Current medical management of hypercalcemia includes rehydration and use of biphosphonates and other antihypercalcemic agents (Percherstorfer et al., 2003) Health care professionals concur that restricting the intake of foods containing calcium is not indicated since the consumption of these foods has little effect in the overall management of hypercalcemia; common sense dictates that oral calcium supplements should be avoided. Critical imbalances

*Self-Instructional Material*

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in fluid and electrolytes can occur in individuals who have cancers that promote excessive diarrhea or vomiting. Profuse and often severe diarrhea can result from partial bowel obstructions; endocrine-secreting tumors such as those secreting serotonin (carcinoid syndrome), calcitonin, or gastrin (Zollinger-Ellison syndrome); and steatorrhea. The use of certain chemotherapy agents (e.g., anti-metabolites, alkylating agents) and antibiotics is associated with the development of sometimes severe diarrhea (Grant, 2006). In some instances immune-compromised individuals may experience profuse diarrhea that is caused by *Clostridium difficile*. Persistent vomiting is associated with intestinal obstruction, radiation therapy to the abdomen or whole brain, highly emetogenic chemotherapy agents, intracranial tumors, and terminal cancer (Grant, 2006). Careful assessment and evaluation of the etiology of the diarrhea or vomiting is critical for effective management. The activities of several enzyme systems can be affected, as can certain endocrine functions. The nature of the alterations varies by tumor type. The individual's immunologic function can be impaired, apparently as the result of both the neoplasm and progressive malnutrition. In addition to the cancer-induced metabolic effects, the mass of the tumor may anatomically alter the normal physiology of specific organ systems.

### **Loss of Appetite and Sensor Changes**

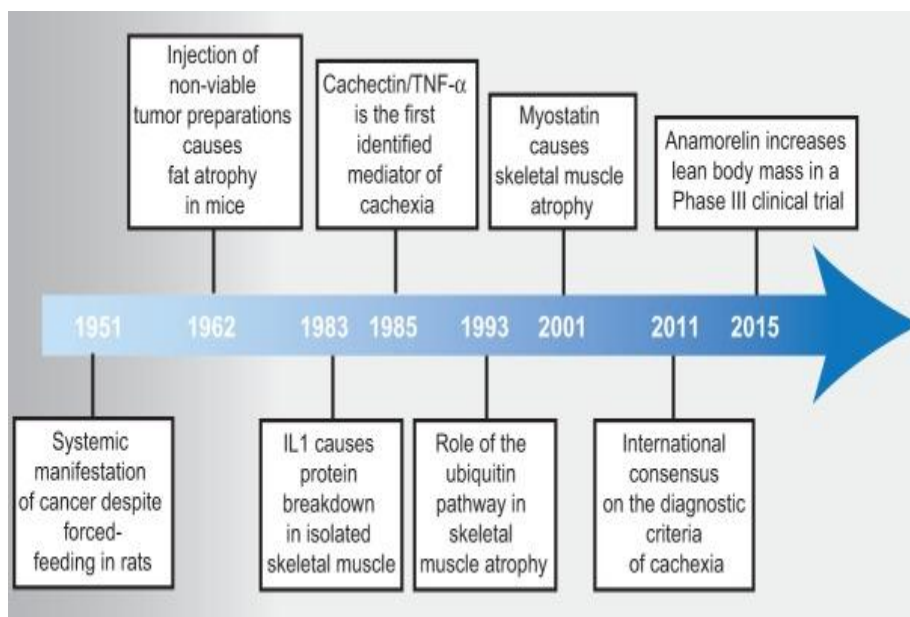
During the course of disease many individuals report a loss of appetite and a decreased voluntary food intake. Alterations in taste and smell are common, and they can contribute to the anorexia commonly seen in individuals with cancer. Taste alterations are associated with the disease itself, certain chemotherapy agents, and radiation therapy or surgery of the head and neck. Chemotherapy-induced, learned taste aversions have been reported in both adults and children. Individuals may also experience a heightened sense of smell that results in sensitivity to food preparation odors and aversions to nonfood items such as soaps or perfumes. Dietary interventions that decrease the aroma of foods such as serving foods cold instead of hot may be helpful. These sensation abnormalities do not consistently correlate with the tumor site, extent of tumor involvement, tumor response to therapy, or food preferences and intake.

### **Research milestones in cancer cachexia**

For centuries, the concept that a local malignant growth could be responsible for systemic effects has been under debate. Rather than a specific disease, the wasting associated with cancer was attributed to nonspecific pathological complications of the tumor, such as anorexia, hemorrhage, infection, or ulceration of the neoplastic tissue. In contrast, those in favor of systemic alterations produced by the tumor on the host considered cachexia the result of either direct secretion by the tumor of some substances active in distant organs or uptake by the tumor of components from the blood that are essential for the correct functioning of distant organs. Evidence supporting one hypothesis or the other was slim, and the contention was disputed on the basis of small clinical case series and anecdotal post-mortem findings (Donovan 1954).

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During the past decades, a vast body of investigation has reshaped our understanding of CAC. Experimental work with animal models of cancer rather than observations in the clinical setting led to the recognition of CAC as a legitimate entity independent of the effects of anorexia or mechanical interference of the tumor with the surrounding tissues. When tumor-bearing rats were force-fed a high-fat diet, weight loss was prevented. However, the development of anemia and the enlargement of the adrenal glands were not affected, thus showing the existence of systemic manifestations of cancer independent of nutritional intake. Following the kinetics of tissue loss in tumor-bearing mice, it was noticed that adipose tissue wasting was an early event, occurring at a time when the tumor was barely palpable. Surprisingly, fat loss could also be induced by nonviable tumor preparations, indicating that soluble components of tumor extracts can induce cancer cachexia. The causes for the systemic effects associated with cancer were more sophisticated than just reduced food intake and needed to be sought in the complex relationship between the host and the tumor. Analogies between systemic responses to infectious agents and cancers were noted, including fever, leukocytosis, and increased serum levels of acute phase response proteins. The first evidence that inflammatory mediators—namely, cytokines—were involved in the process of protein breakdown in isolated skeletal muscle and a potential role for interleukin-1 (IL-1) in muscle degradation during fever was published in 1983 (Baracos et al. 1983). Particular attention was received by the somewhat paradoxical increase in serum lipid levels despite the obvious loss of body weight in severely sick patients. It was found that such hypertriglyceridemia could be induced experimentally in animals by either injection of infective agents or transplantation of tumors. Hypertriglyceridemia was the result of lipoprotein lipase (LPL) inhibition and could be reproduced by injecting animals with conditioned medium from inflammatory cells incubated with endotoxin (Kawakami and Cerami 1981).





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Timeline of discoveries in cancer cachexia. In 1951, the first systemic manifestation of cancer was described in rats. In 1962, it was observed that injection of tumor preparations in mice was sufficient to induce fat atrophy. In 1983 and 1985, the first candidate molecules were identified. Seminal publications in 1993 and 2001 described a role for the ubiquitin pathway and myostatin in skeletal muscle atrophy. It was not until some years ago that an international consensus on the diagnostic criteria of CAC was reached. Promising results have been reported in late 2015 from the first phase III clinical trial targeting CAC with the ghrelin receptor agonist anamorelin.

In 1985, Bruce Beutler in Anthony Cerami's group (Cerami et al. 1985) provided definitive proof that circulating mediators could cause cachexia, showing that culture medium from endotoxin-activated macrophages caused body weight loss when injected into mice. The molecule in the conditioned medium causing cachexia was purified and termed "cachectin" (Beutler et al. 1985). Subsequent determination of the complete primary structure of cachectin revealed its identity with tumor necrosis factor- $\alpha$  (TNF $\alpha$ ) (Fransen et al. 1985; Pennica et al. 1985). It should be noticed that these early preparations of conditioned medium contained multiple macrophage products, and it is therefore erroneous to attribute all of the cachexiogenic action to the effect of TNF $\alpha$  alone. Furthermore, TNF $\alpha$  causes systemic shock and the release of other cytokines, further confounding the attribution of the observed phenotype to a single identifiable factor. However, despite the technical limitations, these early studies contributed to a conceptual evolution in the field of cachexia research, and the wasting syndrome was finally regarded as the result of the host response to the tumor. Clinical evidence confirmed the conclusions of preclinical investigations showing that intravenous hyperalimentation could not alleviate cachexia in cancer patients. Remarkably, the focus of research had gradually shifted from the nature of the invasive agent (infection or cancer) to the quality of the response elicited in the organism. The immune system was the likely source of all mediators responsible for the systemic changes, and a variety of cytokines joined TNF $\alpha$  in the ability to cause systemic alterations. However, despite the clear role played by cytokines in experimental cachexia, their involvement in human disease was less obvious, and clinical translation yielded ambiguous results. Perhaps as a consequence of the disappointment generated by the lack of clinical benefits from basic findings, the pace of research in the field of cancer cachexia progressively reduced. An additional explanation could be the rapid progress of molecular biology of cancer in the mid-1980s. That was the time when the first oncogenes were discovered, and the simplistic view of reducing cancer to a single base mutation was occupying the entire scene (Weinberg 2014). The war on cancer seemed to be close to a favorable ending, and the focus of research zoomed back to the tumor itself rather than the response that the tumor ignites in the organism.

**Cancer cachexia is an energy balance disorder linked to inflammation**

Weight loss is a cardinal sign of cachexia and represents the main independent predictor of mortality in cancer patients. The mechanisms for weight loss in cancer are multiple, including decreased nutrient intake, systemic metabolic dysfunction, and increased energy expenditure.

Inflammation represents a common denominator in the pathophysiology of energy imbalance during cachexia. In mice, the peritoneal injection of cancer cells expressing TNF $\alpha$  has been shown to cause weight loss and cachexia. In contrast, mice injected with the same cells without TNF $\alpha$  do not lose body weight. Similar results have been obtained with IL-6 in preclinical models. Both host- and tumor-derived cytokines cooperate in a complex way with the tumor microenvironment to sustain tumor growth and cachexia. In support of a role of cancer-derived cytokines, it has been shown recently that expression of the cytokine TNF-related weak inducer of apoptosis (TWEAK) by cancer cells causes cachexia, and the effect is similar in wild-type and *TWEAK*-deficient mice (Johnston et al. 2015).

As the cancer persists, it is assumed that ongoing local inflammation may reach a threshold when cytokines spill into the circulation, thus transforming the cancer disease from a localized tumor to a systemic impairment. Unfortunately, such a simplistic view does not stand present experimental validation. The levels of serum cytokines do not correlate with the appearance of cachexia in cancer patients. Furthermore, treatments with antibodies targeting a single cytokine have failed so far to prevent or significantly ameliorate the wasting syndrome. Very recent data emphasize the multifactorial etiology of CAC, showing now that a combination of cytokines and/or additional mediators is responsible for the cachectic phenotype.

Despite the absence of a simplistic threshold model linking cytokine levels to cachexia development, a rich body of evidence supports their causal role in the metabolic dysfunction observed in CAC. Mechanistically, cytokines were shown to increase the metabolic rate through activation of thermogenesis, inhibit adipocyte and skeletal myocyte differentiation, and reduce food intake. However, weight loss in cancer patients cannot be attributed solely to decreased food intake, since dietary supplements fail to reverse cachexia. In contrast, a recent study in mice expressing high levels of the proinflammatory cytokine IL-18 suggests that high caloric feeding in the context of metabolic dysfunction may exacerbate weight loss and cause fatal cachexia. In the context of cancer, metabolic dysfunction is caused by deregulated carbohydrate and lipid metabolism.

#### **Altered carbohydrate metabolism in cancer cachexia**

Carbohydrate intolerance in cancer patients has long been noted. While fasting blood sugar concentration between control and cancer groups did not differ significantly, intravenous glucose tolerance tests showed significantly decreased disappearance of glucose in cancer patients. In the first half of the last century, compared glucose levels in the venous blood from the tumor-bearing arm and the unaffected arm of a patient with a sarcoma on the forearm. Glucose levels from the tumor-bearing arm were reduced, thus confirming in vivo the increased rate of tumor glycolysis. Since tumor tissue takes up glucose, the decreased disappearance of glucose observed in the tolerance test must be sought in metabolic alterations in the host tissues associated with cancer development. Either increased hepatic glucose production or a decrease in peripheral utilization could account for the reduced glucose tolerance observed in cancer patients. Despite decreased hepatic glycogen stores, endogenous glucose production is increased in cachectic patients due to increased hepatic glucose recycling

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via lactate, a phenomenon termed the Cori cycle. Apart from these studies, clinical investigations on glucose metabolism in cachectic patients are noticeably thin. While one study suggests that glucose intolerance may worsen with the development of cachexia, other studies found that glucose intolerance did not correlate with body weight loss. Very recently, elegant genetic studies in the fruit fly *Drosophila* have identified an important role of insulin signaling in inducing a cachexia-like systemic wasting following transplantation of *Drosophila* tumors. Both studies have identified a tumor-secreted factor, *ImpL2/IGFBP* (an insulin-binding protein and antagonist of insulin/insulin-like growth factor [IGF] signaling), that is responsible for the wasting phenotypes in organs distant from the transplanted tumors.

### 12.2.3. ABNORMALITIES IN MECHANISM

#### Role of lipids, burning fat, and white adipose tissue (WAT) browning

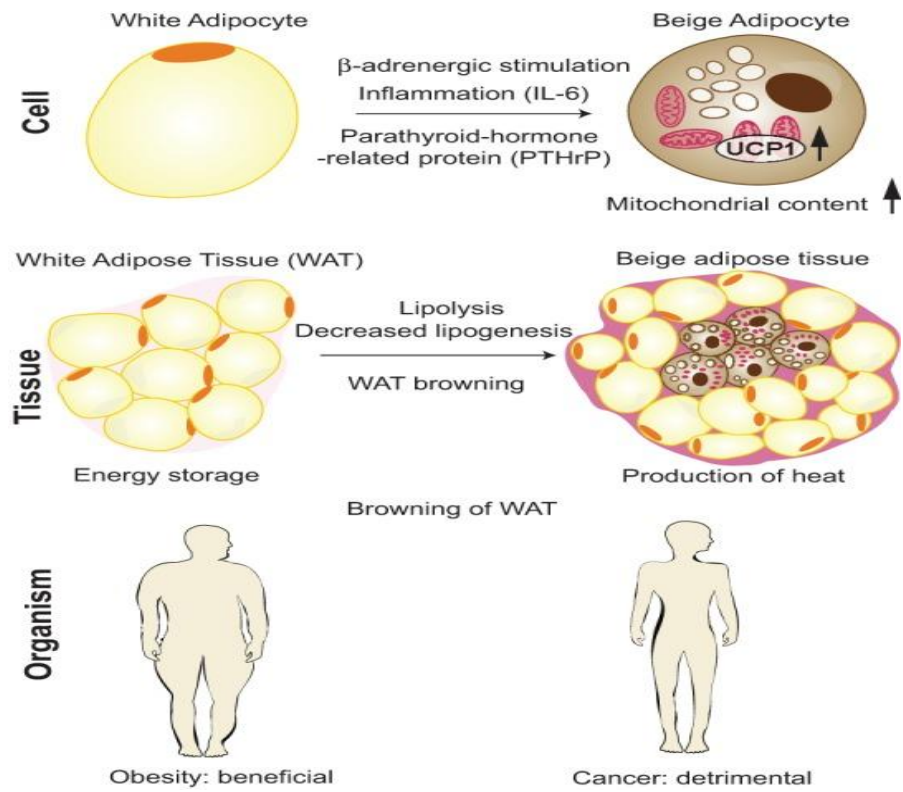
Besides changes in carbohydrate metabolism, the handling of lipids between tissues is severely impaired in cancer patients. The deposition of triglycerides (TGs) in cytoplasmic lipid droplets represents the most efficient form to store lipids in WAT and many other cell types. Already in 1848, the French physiologist Claude Bernard discovered that TGs, commonly called fat, are digested in the gut before they can be absorbed. The hydrolysis of TGs, designated lipolysis, generates glycerol and fatty acids (FAs). The enzymes mediating intracellular lipolysis include adipose TG lipase (ATGL) and the hormone-sensitive lipase (HSL), while LPL is responsible for the hydrolysis of plasma TGs of lipoproteins in the vascular system. FA uptake and TG synthesis decline in WAT in murine cancer models, whereas, in human CAC, it is associated with normal lipid synthesis but elevated lipolysis in WAT. This suggests that lipid catabolism is more relevant than inhibition of lipid synthesis for the loss of WAT in CAC. These findings were corroborated in an elegant study demonstrating that WAT lipolysis in cancer patients is increased due to elevated enzyme activities of ATGL and HSL (Das et al. 2011). Importantly, genetic deletion of *Atgl* in mice prevented increased lipolysis and the reduction of WAT and skeletal muscle mass in certain models of CAC. Similar results were also observed, although to a lesser extent, when HSL was inactivated. Lipolysis in CAC is induced by many serum factors secreted by tumor or host cells, including hormones such as glucocorticoids and catecholamines; cytokines like TNF $\alpha$ , IL-1 $\beta$ , IL-6, prostaglandins; and a zinc-glycoprotein, ZAG, also called lipid-mobilizing factor. How functional lipolysis impacts the development of cancer cachexia is the focus of ongoing investigations in several laboratories.

While quantitative changes in WAT content during cancer cachexia have long been recognized, only recently a qualitative change in the morphology and function of white adipocytes has been described. During the progression of cancer cachexia in preclinical models, WAT cells gradually convert to brown adipose tissue (BAT)-like cells, also called “beige” cells, in a process termed “browning”. Beige cells are characterized by high mitochondrial content and increased expression of uncoupling protein 1 (UCP1), which is responsible for uncoupling the use of mitochondrial electron transport from ATP synthesis to thermogenesis. The phenomenon of browning was initially described as an adaptive response to prolonged exposure to cold environments. When exposed to cold

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temperatures, mice deficient in the ability to activate thermogenesis rapidly lose core body temperature and are more susceptible to cold-induced damage. The induction of browning in humans was initially hypothesized on the basis of increased fluorodeoxyglucose (FDG) uptake in WAT depots using positron emission tomography (PET) and later confirmed at the histological level. Recent investigations have shown that the role of browning is not limited to cold acclimatization. In preclinical models of diet-induced obesity, browning promotes systemic energy expenditure, which results in body weight loss and improved insulin sensitivity. The protection conferred by browning against high-fat diet-induced obesity suggests pharmacological enhancement of browning as a promising therapeutic strategy for metabolic disorders due to excess of nutrients. While the effect of browning is identical in both obesity and cancer, the metabolic result is the opposite. Increased lipid mobilization and energy expenditure are favorable in obesity while being deleterious in cancer. In fact, different from obesity and the metabolic syndrome, browning in the context of cancer exacerbates the metabolic dysfunction, enhancing energy dissipation and contributing to the progression of CAC. Browning in cancer-bearing mice is a systemic event manifested in multiple WAT depots. It precedes the onset of skeletal muscle atrophy and determines a hypermetabolic state characterized by high resting energy expenditure. Notably, browning is not restricted to one experimental model and is not associated with one specific cancer type, since it was documented in complementary model systems, including genetically engineered mouse models (GEMMs), carcinogen-induced cancers, syngeneic transplants of murine cancer cells, and xenogeneic transplants of human cancer tissue. Recently, browning of WAT has been shown to take part in the pathogenesis of hypermetabolism commonly observed in other morbid conditions, like post-burn injury, severe adrenergic stress, and kidney failure. Treatment of mice with a synthetic thyroid hormone receptor agonist induces adaptive thermogenesis in subcutaneous WAT, thus suggesting a role for WAT browning also in hyperthyroidism.

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Mechanisms and consequences of WAT browning in cancer cachexia. At the “cell” level, beige adipocytes are induced in WAT by a combination of signaling pathways, including  $\beta$ -adrenergic stimulation, inflammation mediated by IL-6, and the presence of parathyroid-related peptide (PTHrP); as a result, UCP1 levels and mitochondrial content are increased. At the “tissue” level, CAC is associated with the appearance of islets of beige adipocytes in WAT, surrounded by white adipocytes of reduced size due to ongoing lipolysis. WAT browning and lipolysis result in decreased energy storage and increased production of heat. In the context of obesity, WAT browning is beneficial, while in cancer patients, it is detrimental.

Tumors can directly activate thermogenesis in beige cells through the secretion of parathyroid-related peptide (PTHrP), which has been identified in the supernatants from a murine lung carcinoma cell line and shown to drastically induce the expression of *UCP1*. At the molecular level, transformation of white adipocytes into beige cells requires the function of the transcriptional coregulator PRDM16. Interestingly, fat-specific *Prdm16*-deficient mice challenged in a model of cancer cachexia showed a significant reduction of browning, thermogenic activity, and WAT atrophy. Importantly, injection of cachectic xenotransplant mice with a neutralizing antibody specific for PTHrP was beneficial, reducing the intensity of cancer cachexia and skeletal muscle atrophy. In lung and colorectal cancer patients, higher plasma PTHrP concentrations are associated with increased energy expenditure and enhanced lean tissue wasting, thus confirming the therapeutic potential of inhibiting PTHrP in human cancer. While treatment of cachectic mice with a PTHrP antibody ameliorated the severity of cachexia, it did not inhibit it completely, thus suggesting that other tumor-

derived or host-derived molecules collaborate with PTHrP in the induction of browning and systemic wasting.

Next to direct activation of browning through tumor-derived PTHrP, systemic inflammation and activation of the  $\beta$ -adrenergic pathway represent complementary mechanisms involved in the pathogenesis of browning during CAC. Plasma levels of IL-6 are increased in cachectic mice, and genetic blockade of IL-6 by stable incorporation of a shRNA led to a drastic reduction of the severity of cancer cachexia in a xenogeneic cancer model. In addition, IL-6 receptor (*IL-6-R*) knockout mice implanted with melanoma cells displayed reduced browning when compared with control mice, further corroborating the role of IL-6 in the activation of the thermogenic program in white adipocytes. While the direct induction of UCP1 expression by incubation of adipocytes in the presence of recombinant IL-6 is modest, indirect mechanisms are likely to enhance IL-6-induced browning, such as alternative activation of macrophages. These cells have been shown to sustain adaptive thermogenesis by means of enhanced recruitment of  $\beta$ -adrenergic fibers. Indeed, macrophages infiltrate the WAT of cachectic mice and express markers of alternative activation. The link between the immune system and adipose tissue biology is further supported by recent investigations showing WAT browning following microbiota depletion. Interestingly, colonization of the intestine by different strains of bacteria has been shown to modulate disease severity and cachexia development in mouse models. A role for therapeutic agents targeting intestinal function in CAC remains largely speculative at present. Whether inhibition of browning may indirectly affect tumor metabolism is not known. Systemic alterations of the metabolism in the host are predicted to affect local metabolic pathways of cancer cells, although more experimental data are needed.

In addition to browning, many studies using murine cancer models have demonstrated that lipolysis induces the activation of interscapular BAT during cancer cachexia, further contributing to energy uncoupling in mitochondria with the subsequent worsening of the negative energy balance. BAT has a key role in thermogenesis and energy balance and therefore may well participate in energy expenditure in cancer patients. BAT has been shown to be present in adult humans, and a role for BAT in CAC is possible but is by no means definitively proven.

#### **Muscle wasting in cancer cachexia**

CAC is characterized by muscle atrophy, which severely impairs the patient's mobility because of fatigue and weakness (Cohen et al. 2015). Early labeling experiments have shown that different mechanisms cause skeletal muscle atrophy in different conditions. Increased myofibrillar degradation was observed in skeletal muscle atrophy caused by denervation, while a combination of decreased synthesis and increased degradation was responsible for cortisone-induced muscle atrophy (Goldberg and Goodman 1969). In animal models, glucocorticoids did not cause skeletal muscle atrophy at physiological concentrations but only at increased concentrations under pathological conditions. Therefore, increased adrenal activity and glucocorticoid levels in cancer patients were hypothesized as likely to be responsible for skeletal muscle-wasting during cancer cachexia. However, adrenalectomy did not prevent skeletal muscle atrophy in tumor-bearing

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animals, thus arguing against a role for adrenal hyperfunction in muscle atrophy during experimental CAC. Conversely, microscopic examination of skeletal muscle from cancer cachexia patients did not show evidence of degeneration of muscular or intramuscular nerve bundles, thus excluding also a role for denervation. The factors responsible for skeletal muscle atrophy in CAC remained elusive until the important role of cytokines was finally identified.

Administration of TNF $\alpha$  or IL-1 in mice was found to cause loss of skeletal muscle mass similar to what was observed in cachectic cancer patients. However, while treatment of tumor-bearing rats with anti-cytokine immunoglobulins reduced skeletal muscle atrophy, the protection against systemic wasting was only partial. Evidence accumulated pointing to the idea that, in cachexia, the synergistic action of multiple cytokines and other mediators was responsible for skeletal muscle atrophy and likely most of the other components of the wasting syndrome. At the molecular level, the ubiquitin-dependent proteasome pathway (UPP) was identified as one important mechanism underlying muscle breakdown in pathologic states, such as prolonged fasting and metabolic acidosis. Similarly, activation of the UPP was observed in preclinical models of cancer cachexia. At the genetic level, deletion of muscle-specific E3 ligases *Atrogin-1/MAFbx* or *Murf1* (muscle RING finger protein 1) protected skeletal muscle against experimental atrophy. In contrast, muscle-specific activation of *NF-kB* caused skeletal muscle wasting. In vitro studies have shown a role for TNF $\alpha$  in the activation of NF-kB, which results in inhibition of myocyte differentiation. In addition, cytokines cause a reduction in myofibrillar protein by decreasing the expression of nuclear transcription factor MyoD and through activation of UPP. A large body of evidence implicates the FOXO family of transcription factors as key mediators of skeletal muscle atrophy during CAC as well as during fasting and other pathological conditions. The catabolic effects of FOXO transcription factors are mediated by induction of the atrophy-related ubiquitin ligase *Atrogin-1/MAFbx* and *Murf1*. A third E3 ligase, *Mul1*, has been shown to be involved in the reduction of oxidative capacity in cachectic muscles by controlling mitochondrial protein degradation.

Compelling evidence shows that the atrophy-related genes, also called atrogenes, are directly responsible for skeletal muscle atrophy due to conditions different from CAC, such as denervation, diabetes, or renal failure. Therefore, this points to a concept that a common transcriptional program underlies the loss of skeletal muscle mass independently of the triggering factor (Lecker et al. 2004; Sandri et al. 2006). Skeletal muscle activation of atrogenes in experimental cachexia may also be the result of cross-talk mechanisms between distant organs. As previously noticed, genetic inhibition of lipolysis ameliorates skeletal muscle atrophy in mouse models of CAC (Das et al. 2011). Lipolysis determines an elevated flux of FAs from adipose tissue, and increased FA uptake in the skeletal muscle leads to ceramide synthesis, reduced mTOR activity, and *Atrogin* and *Murf* expression. In this regard, intramyocellular lipid droplets have been described in skeletal muscle of cancer patients, and its overall content was associated with the extent of body weight loss.

While there is considerable experimental evidence for the contribution of atrophy-related UPP in preclinical models, its direct role in human disease and human CAC in particular is, at present, controversial. Conflicting evidence comes from studies that have measured the expression levels of the different UPP components in cancer cachexia patients. Arguing against a direct role, individual components of the UPP were actually found to be unchanged or even down-regulated in cancer patients with suppression of both anabolic and catabolic processes, indicative of reduced muscle turnover that was restored to normal levels following tumor resection. On the contrary, different studies reported an increase in the expression levels of proteasome subunits in skeletal muscle of cancer patients with weight loss. Besides overexpression of the ubiquitin gene, direct measurement of the proteasome proteolytic activity showed enhancement in skeletal muscles of patients with gastric cancer when compared with noncancer surgical controls and was associated with advanced tumor stage and poor nutritional status. These conflicting data comparing animal models and patients with CAC may be due to differences in timing of examination of the skeletal muscle. The analysis in rodents was performed during or at the end of rapid skeletal muscle wasting, while, in cachectic humans, it was performed in the final stage that follows the period of dramatic wasting. It has been shown that changes in expression levels of atrogenes are maximal during the periods of rapid changes in skeletal muscle mass, while further weight loss is associated with reduced gene expression. Multiple time points during skeletal muscle atrophy in human cachexia must be measured before conclusions can be drawn. Additional mechanisms of muscle atrophy in cachexia have been suggested, including activation of the JAK/STAT3 pathway, induction of apoptosis, mitochondrial dysfunction, and the direct effect of cancer chemotherapy.

Besides the factors responsible for skeletal muscle atrophy, studies on factors relevant for muscle hypertrophy have also provided important insights into the mechanisms underlying muscle wasting in CAC. Insulin is the main anabolic factor opposing the catabolic effects of glucocorticoids, and the absence of insulin in rats contributes to skeletal muscle atrophy. At the molecular level, IGF-1 activates insulin receptor substrate 1, which signals through PI3K–AKT to induce protein synthesis by activating mTOR. Skeletal muscle hypertrophy is also observed in the presence of inactivating mutations in Myostatin, while forced expression of Myostatin causes muscle atrophy in adult mice (Zimmers et al. 2002). The muscle hypertrophy observed in myostatin-deficient mice is abolished after inhibition of bone morphogenetic protein (BMP) signaling, which results in up-regulation of the muscle ubiquitin ligase of the SCF complex in atrophy-1 (*MUSAI*). Myostatin and Activin are members of the transforming growth factor  $\beta$  (TGF $\beta$ ) family that were shown to be involved in skeletal muscle atrophy by binding to the Myostatin/Activin type II receptor B (ActRIIB). Interestingly, expression of a dominant-negative *ActRIIB* in transgenic mice results in skeletal muscle hypertrophy. Furthermore, expression of *Myostatin* is increased upon inflammatory signaling, whereas it inhibits myoblast differentiation and increases Foxo activation and the expression of ubiquitin ligases. A recently identified PGC1 $\alpha$  isoform, Pgc1 $\alpha$ 4, has been shown to be highly expressed in exercised muscle and was able to prevent



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skeletal muscle atrophy by repressing Myostatin activity. Notably, mice with skeletal muscle expression of *Pgc1 $\alpha$ 4* were protected from CAC. As an additional mechanism for skeletal muscle dysfunction in cancer, TGF $\beta$  release from bone metastasis has been demonstrated to lower intracellular calcium signaling and reduce the force of muscle contraction.

From a therapeutic perspective, recent clinical trials have provided proof of principle that it is possible to promote skeletal muscle anabolism in cancer patients. A high-protein diet supplemented with leucine has been shown to increase muscle fractional synthetic rate in a small randomized trial in cancer patients. However, it has been reported that leucine supplementation increases pancreatic cancer growth in mice, a mechanism mediated by activation of mTOR . The landmark study by Zhou et al. (2010) has shown that pharmacological blockade of ActRIIB in mouse models of CAC ameliorates skeletal muscle atrophy and prevents atrophy of cardiac muscle. Importantly, ActRIIB blockade significantly prolonged survival even in the absence of direct effects on tumor growth and cytokine secretion. At present, it is not clear whether Myostatin inhibition may also ameliorate skeletal muscle atrophy by direct stimulation of stem cell proliferation. Protection against skeletal muscle atrophy and regrowth of skeletal muscle myocytes are observed after ActRIIB blockade, although a causative role of Myostatin inhibition has yet to be proven. Impaired regenerative capacity of myogenic cells has been recently described in CAC, a process mediated by NF-kB-dependent expression of the self-renew.

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## 12.3 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS

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A common secondary diagnosis in patients with advanced cancer is a variant of protein-energy malnutrition. This syndrome is termed cancer cachexia and is characterized by progressive weight loss, anorexia, generalized wasting and weakness, immune suppression, altered basal metabolic rate, and abnormalities in fluid and energy metabolism. The etiology of this complex metabolic derangement is not entirely understood and can manifest both in individuals with metastatic disease and in individuals with localized disease. Cytokines, protein mediators produced by inflammatory cells through broad physiologic actions, produce metabolic changes and wasting in the tumor-bearing host that are similar but not identical to those seen in sepsis and inflammation

In chronic starvation the resting energy expenditure (REE) is reduced as the body adapts to conserve energy and preserve body tissue. However, in comparison with control groups, hospitalized cancer patients were reported to be hypometabolic, normometabolic, or hypermetabolic. The difference in findings is most likely a result of the stages of illness and of nutrition status among the subjects, differing because of methods used in accurately measuring acutely ill individuals. Researchers have found that the site of cancer or tumor does not predictably increase energy needs or REE.

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## **12.4SUMMARY**

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Nutrition is an important factor in the treatment and progression of cancer. The majority of cancer patients experience weight loss as their disease progresses and, in general, weight loss is a major prognostic indicator of poor survival and impaired response to cancer treatment. The incidence of malnutrition among patients with cancer has been estimated at between 40 and 80%; its prevalence depends on the tumour type, location, stage and treatment. The consequences of malnutrition may include an increased risk of complications, decreased response, and tolerance to treatment, a lower quality of life, reduced survival, and higher healthcare costs. Cancer cachexia has been implicated in the deaths of 30–50% of all cancer patients, as many die from the wasting associated with the condition.

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## **12.5KEY WORDS**

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- **Degradation:**The condition or process of degrading or being degraded.
- **Myofibrillar:** any of the elongated contractile threads found in striated muscle cells.

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## **12.6SELF-ASSESSMENT QUESTIONS**

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### **Short Answer Questions**

1. Discuss the about nutritional effects of cancer.
2. Explain the cachexia.
3. Write a short note on energy metabolism.
4. Explain the milestones in cancer cachexia.

### **Long Answer Questions**

1. Elaborate on substrate metabolism and abnormalities in metabolism.
2. Discuss in details about nutritional effects of cancer.

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## **12.7. FURTHER READINGS**

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- <https://touchoncology.com>
- <https://www.ncbi.nlm.nih.gov>
- <https://www.mercyhealth.com>
- <https://www.cancer.org>

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## **UNIT -XIII**

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### **Structure**

- 13.0 Introduction
- 13.1 Objectives
- 13.2 . Cancer therapy
  - 13.2.1. Chemotherapy
  - 13.2.2. Radiation therapy
  - 13.2.3. Immunotheraphy
  - 13.2.4. Bone marrow transplant
  - 13.2.5. Hematopoietic Stem Cell Transplantations
- 13.3. Dietary management
- 13.4. Planning diet therapy
- 13.5. role of food in the prevention of cancer
- 13.6. Answers to Check Your Progress Questions
- 13.7. Summary
- 13.8. Key Words
- 13.9. Self-Assessment Questions
- 13.10. Further Readings

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### **13.0 INTRODUCTION**

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Cancer therapy describes the treatment of cancer in a patient, often with surgery, chemotherapy, and/or radiotherapy. Targeted therapies are also available for some cancer types. A cancer patient might receive many different types of therapy, including those aimed at relieving the symptoms of cancer, such as pain. Chemotherapy is the use of any drug to treat any disease. But to most people, the word chemotherapy means drugs used for cancer treatment. It's often shortened to "chemo." Surgery and radiation therapy remove, kill, or damage cancer cells in a certain area, but chemo can work throughout the whole body. In previous units, we have discussed about the cancer and its nutritional effects.

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### **13.1 OBJECTIVES**

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After studying this unit you will be able to,

- Discuss the cancer therapy.
- Explain the impact of therapy.
- Describe the dietary management of cancer prevention.

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## 13.2 CANCER THERAPY

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Obstruction by a tumor may interfere with nutritional intake absorptive function. Thus, the nutritional status may be further compromised by surgery, radiation therapy, Chemotherapy or immunotherapy.

### 13.2.1. CHEMOTHERAPY

Chemotherapy is the use of chemical agents or medications to treat cancer. The objective of this chemotherapy is to destroy cancer cells throughout the body without causing serious permanent damage to the host.

Chemotherapy is done with the help of some chemotherapeutic agents, and the target of action of these agents is not limited to malignant cells. It affects the normal cell as well. Cells of the body with a rapid turnover such as bone marrow, hair follicles, and the mucosa of the alimentary tract are typically the most affected.

Commonly experienced nutrition impact symptoms include myelosuppression (suppression of bone marrow production), anemia, fatigue, nausea and vomiting, loss of appetite, mucositis, changes in taste and smell, xerostomia (mouth dryness), dysphagia, and changes in bowel function. As a result, dietary intake and nutrition status can be adversely affected.

Classifications of chemotherapy agents include alkylating agents, nitrosources, antitumor antibiotics, hormones, hormone antagonists, anti-metabolites, vinca alkaloids, taxanes, camptothecins, epipodophyllotoxins and immunologics.

Routes of administration for chemotherapy include oral (capsule, pill or liquid), IV (delivery of medication via an injection or an indwelling catheter into a vein), intraperitoneal (delivery of medication via a catheter directly into the abdominal cavity), intravesicular (delivery of medication via a Foley catheter directly into the bladder), or intrathecal (delivery of medication via an injection into the central nervous system using an Ommaya reservoir or a lumbar puncture).

The severity of the side effects depends on the specific agent(s) used, dosage, duration of treatment, number of treatment cycles, accompanying drugs, individual response, and current health status. The timely and appropriate use of supportive therapies such as anti-emetics, anti-diarrheal, hematopoietic agents, and antibiotics, as well as dietary changes, is important to the effective management of treatment-related side effects, especially those that have a nutrition impact. The reality is that, despite the supportive care, many patients still experience significant side effects, especially in "dose-intensive" multiple-agent chemotherapy regimens; neutropenia (reduced white blood cells or neutron/neutrophils) and myelosuppression are the primary factors limiting chemotherapy administration.

Commonly experienced chemotherapy-induced toxicities for the gastrointestinal system include diarrhea, constipation, or adlmamic ileus (inhibition of bowel motility). Chemotherapy-related taste abnormalities can lead to anorexia and oligophagy (eating few foods). Symptoms of gastrointestinal toxicity are usually not long lasting; however some multi-agent chemotherapy regimens have severe and prolonged gastrointestinal

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effects. Some agents, especially corticosteroids, can cause tissue breakdown and promote excessive urinary loss of protein, potassium, and calcium. The intestinal mucosa and digestive processes are affected, thus altering digestion and absorption to some degree. Protein, energy, and vitamin metabolism may be impaired, although the consequences of this are not known. Total lymphocyte count is depressed and does not accurately reflect nutrition status after chemotherapy administration.

Health care professionals should be alert to possible drug-nutrient interactions since some chemotherapy agents can cause potentially severe adverse events. For example, individuals with certain types of lung cancer who are being treated with pemetrexed (Alimta) require vitamin B12 and folic acid supplementation to avoid significant anemia associated with this chemotherapy agent; or a severe hypertensive event is possible when thiamine-rich foods and beverages are consumed while taking procarbazine (Mutalane), chemotherapy agent commonly used to treat brain cancer. Health care professionals can gain valuable insights regarding drug-nutrient interactions and contraindications by reviewing product medication inserts, pharmacy resource books, and medication databases or by consulting with pharmacy personnel. And has many side effects with nutritional implications, and in order to increase the effectiveness of chemotherapy, high dose of 2 or more antineoplastic agents are often given together.

Commonly experienced nutrition impact symptoms includes myelosuppression (suppression of bone marrow production), anemia, fatigue, nausea and vomiting, loss of appetite, mucositis, changes in taste and smell, xerostomia (mouth dryness), dysphagia and changes in bowel function

Cancer chemotherapeutic Agents includes alkylating agents, nitro sources, antitumor antibiotics, hormones, hormone antagonists, anti metabolites, alkaloids, taxanes, camptothecins, epipodophyllo toxins, and immunologist. Route of administration for chemotherapy includes oral (capsule, pill or liquid); IV (delivery of medication via an infusion or an indwelling catheter into vein); intraperitoneal delivery of medication via a catheter directly into abdominal cavity); intravesical (delivery of medication via a foley catheter directly into the bladder); or by Intrathecal (delivery by injecting the central nervous system using an ommaya reservoir or a lumbar puncture)

Examples of chemotherapeutic agents include

Methotrexate, fludarabine, etc.... ---- Antimetabolite

Cisplatin, Busulfan ---- alkylating agent

Vinorelbine, sometimes, Etc.... ---- antimitotic agents

These are some of the cytotoxic drugs, that are mostly toxic to rapidly dividing cells, thus they cause the most common side effects as nausea and vomiting. Sometimes the antibiotics cause significant side effects such as irritability, dizziness, dry mouth. On chemotherapy often results in diarrhea and reduced absorption of nutrients from the small intestine.

### **13.2.2. Radiation therapy**

Radiation therapy uses high-energy rays (ionizing radiation) in multiple fractionated doses to cure, control, or palliate cancer. Radiation therapy can be delivered externally into the body from a megavoltage machine (e.g., linear accelerator, cyclotron, or cobalt-60 unit) or with brachytherapy by placing a radioactive source (implant) in or near the treatment tumor volume to deliver a highly localized dose. Advances in technology to deliver radiation therapy with extreme accuracy include stereotactic radio surgery (Gamma knife) and intensity-modulated radiation therapy. Whereas chemotherapy is a systemic therapy, radiation therapy affects only the tumor and the surrounding area. The side effects of radiation therapy are usually limited to the specific site being irradiated. Chemotherapy agents may also be given in combination with radiation therapy to produce a radiation-enhancing effect. Patients receiving multimodality therapy often experience more toxic side effects sooner. The acute side effects of radiation therapy when used alone generally manifest around the second or third week of treatment and usually resolve within 2 to 4 weeks after the radiation therapy has been completed. Late effects of radiation therapy may occur several weeks, months, or even years after treatment. Regardless of the specific area being irradiated, commonly experienced nutrition impact symptoms include fatigue, loss of appetite, skin changes, and hair loss in the area being treated. Radiation therapy to the head and neck can cause a variety of acute nutritional symptoms: sore mouth, altered taste and smell, dysphagia and odinophagia, mucositis, xerostomia, anorexia, fatigue, and weight loss. Current multimodality protocols for treatment of head and neck cancer that use chemotherapy and surgery as well as radiation therapy, should not be used without aggressive enteral nutrition.

Late effects of radiation therapy may include dental caries, permanent xerostomia, trismus (lockjaw), and osteoradio necrosis. Before beginning therapy, individuals should undergo a dental evaluation and thorough teeth cleaning and receive instruction in good oral hygiene and care, including daily brushing and rinsing. After therapy has been completed, individuals should continue to have close dental monitoring and follow-up. Individuals may also benefit from a referral to a speech pathologist for assessment and evaluation of swallowing function. Nutrition impact symptoms of radiation therapy to the thorax can include heartburn and acute esophagitis characterized by dysphagia and odinophagia. Late effects include possible esophageal fibrosis and stenosis. When this occurs, individuals are generally only able to swallow liquids; esophageal dilations and nutrition support (enteral nutrition) may be necessary to meet nutritional needs. Radiation therapy to the abdomen may produce acute gastritis or enteritis accompanied by nausea, vomiting, diarrhea, and anorexia. Late effects can include severe gastrointestinal damage that is manifested by malabsorption of disaccharides, fats, and electrolytes. Radiation-induced enteritis can develop into a chronic form of the condition, with symptoms of ulceration or obstruction intensifying the risk of malnutrition. Chronic radiation enteritis combined with massive bowel resection, which results in extensive bowel dysfunction, is called short bowel syndrome. The severity of this condition depends on the length and location of the nonfunctional or

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restricted bowel; it is generally diagnosed when the individual has less than 150 cm of remaining small intestine. The sequence include maldigestion, malabsorption, malnutrition, dehydration, and potentially lethal metabolic aberrations. Initially TPN is required, and frequent monitoring of fluids and electrolytes may be required for weeks or months. The diet may need to be restricted to defined formula tube feedings or to frequent small meals high in complex carbohydrate and protein, low in fat and oxalate, and lactose free. Medications such as anti-diarrheal can be given to decrease intestinal motility. Multivitamin supplements that include vitamin B12; folic acid; and vitamins d E, and K should be given to prevent deficiencies. Serum concentrations of various minerals should be monitored and adjusted as needed. Total-body irradiation (TBI) is a technique of radiation therapy that is used in hematopoietic stem cell transplantation to eradicate malignant cells, to ablate the bone marrow to make room for the engraftment of the infused hematopoietic cells, and to suppress the immune system of the recipient to decrease the risk of graft rejection in allogeneic transplants. Acute effects of TBI are often difficult to discern since it is given in conjunction with conditioning chemotherapy (Lawon, 2003). Commonly encountered side effects are fever, nausea, vomiting, headache, parotitis (inflammation of the parotid glands), xerostomia, diarrhea, and fatigue.

### 13.2.3. Surgery

The surgical resection or removal of any part of the alimentary tract, as well as the malignant disease process itself, can impair digestion and absorption significantly. Surgery may be used as the only mode of cancer treatment, or it may be combined with preoperative or postoperative adjuvant chemotherapy or radiation therapy. After surgery individuals commonly experience fatigue, temporary changes in appetite and bowel function caused by anesthesia, and pain. They require additional energy and protein for wound healing and recovery. Most side effects are temporary and dissipate after a few days following surgery. However, some surgical interventions have long-lasting nutritional implications.

Individuals with head and neck cancer often have impaired mastication and swallowing caused by the tumor mass or the specific surgical intervention required. These patients also present additional problems because of their frequent history of smoking, alcohol use, and poor dietary intake. They are at high risk for malnutrition and postoperative complications. Surgery often necessitates temporary or permanent reliance on enteral nutrition, including percutaneous endoscopic gastrostomy or nasogastric tube feedings (see Chapter 20). Individuals who resume oral intake often have prolonged dysphagia and require modifications of food consistency and extensive training in chewing and swallowing. Referrals to a speech therapist can yield dramatic positive results through evaluation and individualized instruction in swallowing and positioning techniques. Surgical treatment of esophageal tumors may require partial or total removal of the esophagus. The stomach is commonly used for esophageal reconstruction. A feeding nasojejunostomy or jejunostomy tube can be placed at the time of surgery permitting early postoperative tube feedings. Usually the individual is able to progress to oral intake with specific dietary recommendations to minimize nutrition impact symptoms, which

include dumping syndrome, dysmotility, gastroparesis, early satiety, vomiting, and fluid and electrolyte imbalances. Postsurgical dietary recommendations include a low-fat diet with small, frequent feedings of nutrient-dense foods and avoidance of large amounts of fluids at any one time. Surgery is the most common treatment for cancer of the stomach, although chemotherapy and radiation therapy can be used before or after surgery to improve survival and control disease. Surgical interventions include partial or subtotal gastrectomy or total gastrectomy.

Placement of a jejunostomy feeding tube at surgery is advisable, and enteral nutrition support is generally feasible within a few days after surgery. Postgastrectomy syndrome encompasses a myriad of nutritional intolerances and deficiencies. Its symptoms include dumping syndrome, fat malabsorption, gastric stasis, lactose intolerance, anemias, and metabolic bone disease (e.g., osteoporosis, osteopenia, osteomalacia). Dumping syndrome is a common complication of gastric surgery and it is manifested by the rapid transit of foods or liquids (especially those high in simple carbohydrate content) and the dilutional response of the small remaining stomach to highly osmotic bolus feedings. Individuals may experience gastrointestinal and vasomotor symptoms such as abdominal cramps, diarrhea, nausea, vomiting, flushing, faintness, diaphoresis, and tachycardia.

Malabsorption is another complication of gastric surgery; deficiency of iron, folate, and less commonly vitamin B12 can lead to anemia. Micronutrient deficiencies of calcium and fat-soluble vitamins are also common. Individuals benefit from consumption of six to eight small meals per day, with fluids taken between meals. Fat intolerance may also be experienced, especially if the vagal nerve is severed. Administration of pancreatic enzymes with meals may be beneficial for patients for whom the mixing of food and pancreatic juices is inadequate. Pancreatic cancer, with its attendant surgical resection, has significant nutritional consequences. The Whipple procedure and the pylorus-sparing pancreatic duodenectomy are the most commonly used pancreatic cancer surgeries. Postsurgical complications include delayed gastric emptying, early satiety, glucose intolerance, bile acid insufficiency, diarrhea, and fat malabsorption. Pancreatic enzyme replacement may be used to aid digestion and absorption, and a fat-restricted diet may be indicated. Individuals benefit from the use of small, more frequent feedings and, if indicated, avoidance of simple carbohydrates. Partial or total resections of the intestinal tract may induce profound losses of fluid and electrolytes, the severity of which is related to the length and site of the resection. Resections of as little as 15 cm of the terminal ileum can result in bile salt losses that exceed the liver's capacity for resynthesis, and vitamin B12 absorption is affected. With depletion of the bile salt pool, steatorrhea develops. Nutrition support consists of a diet low in fat, osmolality, lactose, and oxalate



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#### **13.2.4. Immunotherapy**

Immunotherapy involves using response modifiers that all natural products that are made in quantities through cloning and genetic engineering. It is either used directly as cytotoxic agents or indirectly as stimulators of the individual own defense, where the biologic agents can kill the tumor cells

Alpha interferon is used to treat hairy cell leukemia.

Interleukin-2 is used in the treatment of individuals with malignant melanoma and renal cell carcinoma. Immunotherapy includes supportive care agents (hematopoietic) such as colony stimulating factors (cytokines that stimulate the marrow to develop faster) that are used to shorten periods of neutropenia (reduced with WBC or neutrophils) and thrombocytopenia for individual to enrich the graft of myelitis precursor before harvest of marrow from donors.

Some agents are interleukin, monoclonal antibodies, filgrastim (granulocyte colony stimulating factor G-CSF), and erythropoietin (EPO) when these are used, some individuals will experience fatigue, chills, fever, flu like symptoms and decreased food intake.

#### **13.2.5. Bone marrow transplant**

Bone marrow is the soft spongy tissue that fills the cores of a larger bones. It serves an active function in the body by producing all three types of blood cells as well as lymphocytes, which supports the immune system.

Bone marrow transplant is a procedure used to treat patients with life threatening blood, immune or genetic disorder. This includes leukemia, bone marrow cancer. A bone marrow transplant replaces the unhealthy blood forming cells with healthy ones.

Bone marrow transplant is done to treat a number of cancerous and noncancerous conditions such as

Cancerous condition ---- leukemia, lymphoma, multiple myeloma, Myelodysplasia

Non cancerous condition ----- aplastic anemia, hemoglobinopathy, immunodeficiency disorder and condition affecting blood from birth

Transplant process occurs in five steps as

Conditioning, stem cell infusion, neutropenic phase, engraftment phase, post engraftment phase.

Graft versus host disease occurs with allogeneic transplantation other than these chest pain, fever, chills, nausea, shortness of breath occurs.

#### **13.2.6. Hematopoietic Stem Cell Transplantations**

Hematopoietic stem cell transplantation (HSCT) is performed for the treatment of certain hematologic malignant diseases such as leukemia and lymphoma, malignant solid tumors, and autoimmune disorders. The stem cells used for HSCT arise from bone marrow, peripheral blood, or umbilical cord blood. The preparative regimen includes cytotoxic chemotherapy, with or without total-body irradiation, to suppress immunologic reactivity and eradicate malignant cells. This treatment regimen is followed by IV infusion of hematopoietic cells from the individual (autologous) or from a histocompatible related or unrelated donor (allogeneic) or from an identical twin (syngeneic). HSCT procedures can significantly affect nutrition status. There should be a thorough nutrition assessment of the patient before the initiation of therapy

and reassessments and monitoring throughout the entire transplant course. The acute toxicities of immunosuppression that can last for 2 to 4 weeks after the transplant include nausea, vomiting, anorexia, dysgeusia, stomatitis, oral and esophageal mucositis, fatigue and diarrhea. In addition, immunosuppressive medications can also adversely affect nutrition status (Charuhas, 2006). Individuals typically have little or no oral intake during the first few weeks following transplant; therefore enteral or parenteral nutrition support is usually considered and has become a standardized component of care. Because the function of the gastrointestinal tract is compromised, TPN is often used. Gastrostomy tubes are useful for long-term nutrition support, and TPN should be reserved for individuals who are unable to tolerate oral or enteral feeding (Sheehan, 2005). However, the administration of optimal levels of TPN is complicated by the frequent need to interrupt it for the infusion of antibiotics, blood products, and medications. This in turn necessitates careful monitoring and the use of more concentrated nutrient solutions, increased flow rates, and double- or triple-lumen catheters.

Autologous HSCT involves the use of the individual's own stem cells to reestablish hematopoietic stem cell function after the administration of high-dose chemotherapy. In some cases the use of mobilized stem cell progenitors has replaced autologous bone marrow as the source of hematopoietic progenitors for transplantation. Their use has shortened the period of pancytopenia (reduction in the cellular components of the blood), when patients are at risk for bleeding and serious infections that may lead to sepsis. These advances, along with improved prophylactic antibiotic regimens that are relatively easy to administer, have allowed patients to receive autologous marrow transplantation in the outpatient setting. This change has substantially reduced the cost of transplantation and thus has made it available to an increased number of patients. However, because a majority of these patients receive much of their care outside the hospital, nutrition assessment and monitoring are of critical importance.

The HSCT procedure is associated with severe nutritional consequences and requires prompt, aggressive intervention. Nausea, vomiting, and diarrhea are caused by the cytotoxic conditioning regimen and may later accompany antibiotic administration. Antiemetics may be helpful. Complications of delayed onset include varying degrees of mucositis, xerostomia, and dysgeusia. Mucositis, which is often severe and extremely painful, develops in more than 75% of transplant patients.

Bland liquids and soft solids are usually better tolerated in individuals with treatment-related mucositis. Strong flavored, acidic, or spicy foods should also be avoided. Herpes simplex virus and *Candida albicans* account for most oral infections. Salivary stimulants and substitutes are beneficial for temporary relief of dry mouth; in addition, liquids and foods with sauces and gravies are usually well tolerated.

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### **13.3. DIETARY MANAGEMENT**

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Nutrition therapy for cancer patients is highly individualized, depending on the body's response to the disease, the site of the cancer, the type of treatment, and the specific physical and psychological responses of the patient. Myriad metabolic and nutritional changes are characteristic of nearly all cancer patients. These include fatigue, asthenia, cachexia,

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anorexia, anemia, fluid and electrolyte imbalances, hypogeusia or dysgeusia, xerostomia, dysphagia, esophagitis, malabsorption, stomatitis, nausea and vomiting, fever, altered metabolic rate, negative nitrogen balance, and edema. Infection is not uncommon.

### **13.4. THE BODY'S RESPONSE TO CANCER**

The specific type of cancer, and the disease process itself, has profound effects on the entire body system and cause primary nutritional deficiencies. Some examples of the body's responses to several types of cancer are given in the following paragraphs. Cancers occurring in the gastrointestinal tract or adjacent tissue cause difficulty in ingestion and use of nutrients.

Obstruction curtails intake, and malabsorption interferes with digestion of fats and fat-soluble vitamins, especially vitamin D, which in turn leads to decreased metabolism and absorption of calcium, causing osteomalacia. Abdominal tumors may cause fistulas to develop, leading to bypass of the small intestine and consequent malabsorption. Adenocarcinoma of the colon leads to severe electrolyte imbalance. General malabsorption also contributes to fluid and electrolyte imbalance. Vomiting and diarrhea result in loss of water-soluble vitamins. Intestinal malignancies contribute to hypokalemia. Cancer of the bone, or breast cancer with metastasis to the bone, also lead to hypokalemia. Cancer within the thyroid gland will result in hormonal imbalances. Pancreatic cancer and resulting pancreatectomy lead to the loss of digestive enzymes and diabetes mellitus. Anorexia, the most common symptom, is related to altered metabolism, type of treatment, or emotional distress. Increased hemolysis, bleeding of lesions, fistulas, and malabsorption of nutrients needed for hemoglobin formation (iron, protein, folic acid, vitamin B12, and vitamin-C) lead to severe anemia.

Oral and other enteral feeding modes pose fewer problems than do alternative means. The spectrum of feeding modalities Oral diet amplified with nutrient supplement for increased protein, calories, vitamin and minerals. Enteral tube feeding with several routes of entry. Parenteral nutrition through central and peripheral veins. Feeding the patient by normal ingestion of food and nutrient supplements is most desirable. Based on individual nutritional assessment, a personal food plan is developed with the patient, incorporating desired food forms and family food patterns. Often the diet of the hospitalised patient can be supplemented with familiar foods from home as the clinical nutritionist plans with the family.

A number of adjustment in food texture, temperature, amount, timing, taste appearance and form can be made to help alleviate symptoms. With support, the patient and family are better able to build a positive mental attitude toward the diet as an integral part of the treatment. Food should be nutrient dense. Texture can be varied as tolerated, with appeal to sensory perceptions of colour, aroma and taste to enhance the desire to eat.

Often a series of mini meals using a wide variety of food items is better tolerated than regular large meals. If appetite is better in the morning, meals and maintaining surroundings that reduce stress may also help in the

eating process appealing in aroma and appearance and in small amounts should be continued.

Since zinc deficiency is related to diminished taste, sometimes a zinc supplement may be indicated. Salivary secretions are also affected by cancer therapy should be used good breakfast should be emphasised. Getting some exercise before so foods with a high liquid content.

Nausea is often enhanced by foods that are hot, sweet, fatty or spicy. So these can be avoided according to individual tolerance. Frequent small feedings of liquid in texture can be given. Eating dry foods such as crackers and dry toast on waking in the morning may be helpful.

Sore mouth often results from chemotherapy or radiation to the head and neck area. It is increased from any state of malnutrition or from infections such as candidiasis (thrush with numerous ulcerations of the oral and throat mucosa).

Frequent small meals and snacks, soft it texture, bland in nature and cool to cold in temperature are often better tolerated.

Cancer patients can be benefitted from early and continuing individualised nutrition intervention. The intervention may start before therapy begins, continuing throughout therapy and after completion for atleast 3 years.

The diet should be supplemented with medium chain triglycerides if there is fat malabsorption.

### **Common Nutritional Problems Occurring in Cancer Patients with Three Major Treatment Modes**

Radiation Therapy (effects depend upon site of irradiation)

#### **Head, neck, or esophagus**

1. Anorexia
2. Impaired taste acuity
3. Reduced food intake
4. Tooth decay and gum disease
5. Difficulty swallowing
6. Decreased salivary secretions and taste sensations
7. Sensitivity to texture and temperature of food
8. Inflamed oral mucosa

#### **Abdomen**

1. Loss of intestinal villi and absorbing surfaces
2. Vascular changes
3. Inflammation
4. Obstructions
5. Strictures, fistulas
6. Anorexia and nausea
7. Malabsorption
8. Diarrhea

#### **Chemotherapy**

1. Interference with production of both white blood cells and red blood cells
2. Nausea, vomiting, stomatitis, anorexia, ulcers, and diarrhea; response of the GI system similar to those that occur in radiotherapy
3. Body fluid and electrolyte disturbances
4. Hair follicle loss

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Surgical Therapy (effects site dependent)

### **GI Tract**

1. Impaired food ingestion
2. Malabsorption
3. Potential dumping syndrome
4. Possible low blood glucose following gastric resection
5. Insulin deficiency from resection of the pancreas (diabetes mellitus)
6. Fluid and electrolyte imbalances
7. Head and neck surgery or resection poses special feeding problems: different feeding methods (enteral or parenteral) and feeding intervals, and modifications in oral food preparation.

### **13.6. PLANNING DIET THERAPY**

The objectives of diet therapy are to do the following:

1. Meet the increased metabolic demands of the disease and prevent catabolism of the body tissues.
2. Alleviate symptoms of the disease and its treatment by adapting the food and feeding methods to the individual.

The basis for planning care includes:

1. Thorough personal nutrition assessment
2. Vigorous nutrition therapy to maintain good nutritional status and support
3. Revision of care plan as individual status changes Major eating problems, as discussed earlier, are:

1. Appetite problems include anorexia caused by systemic effects of cancer and treatment modalities, depression, anxiety, and stress. These problems lead to cancer cachexia.

2. Mouth problems caused by stomatitis, sore mouth, dysgeusia, hypogeusia, low salivary production, and candidiasis often occur.

3. Gastrointestinal problems, in the upper intestine, include nausea, vomiting, bloating, postgastectomy dumping syndrome, and so on. In the lower intestine, diarrhea, constipation, lactose intolerance, and so on occur. Each of the following factors is related to tissue protein synthesis and energy metabolism. Increased needs for all major nutrients, including fluids, are based on the demands of the disease and treatment. Individual needs may vary, but the general guidelines are the same.

1. Energy: Increase total energy value to prevent excessive weight loss and meet increased metabolic demands. An adult in good nutritional status requires less than 2000 kcalories per day for maintenance. A severely malnourished patient may require 3000 to 4000 kcalories. Carbohydrates should supply most of the energy intake with fat restricted to about 30% of total calories.

2. Protein: Provide additional amino acids and nitrogen for healing and tissue regeneration. An adult in good nutritional status requires less than 80–100 g for maintenance and anabolism. A malnourished patient will need more, depending on individual requirement and treatment(s).

3. Vitamins and minerals: Key vitamins and minerals control energy, protein, and amino acid metabolism. Review Chapters 2 through 6 for specifics. Some characteristics are given here. The B-complex vitamins are coenzymes in protein and energy metabolism. Vitamins A and C are

components of tissue structure. Vitamin C is also an antioxidant and functions in immune and enzyme reactions. Vitamin A functions in cell differentiation and protective immunity. Vitamin D has a vital role in the metabolism of calcium and phosphorus in bone and blood serum. Vitamin E protects the integrity of cell walls. Many minerals have structural and/or enzymatic roles in metabolic and tissue building processes.

4. Water is second only to oxygen as the most important nutrient in the human body, and maintenance of the fluid and electrolyte balance is especially crucial in cancer. Review Chapter 6 for the functions and distribution of body water.

Many individuals with cancer or AIDS subscribe to unproven nutritional therapies, from personal beliefs that it will help them take control of their disease, on the advice of family and friends, or information found on Web sites and other media. Herbal remedies, macrobiotic diets, metabolic therapy, and thymus gland extracts are often encountered by the healthcare professional when taking diet histories. Megavitamin and mineral therapies (taking 10 times the RDAs/DRIs) are among the most often used. Vitamins that are popular are A, C, B12, and thiamine, and the minerals iron, zinc, and selenium. These therapies and others can be harmful, and more details are described in Chapter 12 on alternative medicine.

Special considerations in feeding a cancer patient include the following:

1. Do not provide drinks during meal time if the patient experiences nausea. Separate liquid from solid foods.
2. If the patient has diarrhea, avoid the following:
  - a. Vitamin C supplements in high dosage
  - b. Laxative teas
  - c. Foods containing sorbitol such as sugar-free candy and gums
  - d. Dairy products rich in lactose
  - e. Caffeine
3. If the patient has a decreased appetite, do not recommend large meals.
4. If the patient has oral thrush, avoid the following:
  - a. Salty, hot, and/or spicy foods
  - b. Acidic foods such as citrus fruits, tomato-based products, vinegar or vinegar-based foods
5. If the patient has difficulty in swallowing, avoid foods that are difficult to swallow. Examples include sticky foods such as peanut butter.
6. If a patient is insulin resistant, avoid a low-fiber diet.
7. If the patient experiences a change in taste sensation, do not use oral supplements in metallic cans.

The effectiveness of cancer treatments and patient's subsequent recovery depend in large part upon adequate nutrition. Both are affected by nutrition intake and utilization.

1. Malnutrition in a cancer patient is not inevitable. Most patients can be adequately nourished, if properly planned and executed nutrition therapy is provided.
2. Be aware that nutrition therapy must be proactive. Early assessment, intervention, and continuing preventive measures to prevent malnutrition are mandatory.

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3. Nutrition therapy is designed for specific physical and psychological needs and is highly individualized, depending upon the response of each body system to the disease and treatment modality.
4. Nutrition care plans are patient centered: patients need to have some control in planning during disease stages and therapy effects.
5. Anticipate psychosocial situations that relate to appetite, various foods, drug effects, lifestyle, and beliefs of the client.
6. Provide the patients with information regarding symptoms they are experiencing, actions of their drug regimes, and mouth care tips they can do themselves.
7. Make a thorough assessment of energy, protein, electrolyte, fluid, and micronutrient needs of the patient to use as a baseline for planning diet.
8. Nutritional assessment includes physical examination, lab measurements (albumin, lymphocyte count, CBC, nitrogen balance, others), past medical history, present dietary intake (24-hour recall), and any other factors affecting intake.
9. Make revisions in the patient's diet as situations change.
10. Encouragement and support are very helpful. These have a positive effect on a patient's emotional status. They denote caring, comfort, and concern. Emphasize eating to get well, and health and wellness instead of illness.
11. Investigate the use of enteral and/or parenteral methods of feeding if they become necessary. Oral intake is preferred but may not be feasible in some cases.
12. Client education, with the nurse either as the primary teacher or as support teacher in a team effort, is effective in gaining desired goals.
13. Frequent follow-up teaching is desirable.

### **Nutrient Supplements**

Nutrient supplements in a variety of forms and flavours may be used in different ways to enhance nutrient density. When the patient is unable to eat but the gastrointestinal tract can still be used, tube feedings may be needed to provide the necessary nutritional support.

### **ROLE OF FOOD IN THE PREVENTION OF CANCER**

Diet is an important area of intervention for primary cancer prevention. Foods containing antimutagens and anti promoters of cancer are recommended. Consumption of vegetables and fruits (more than 400 g/day) can prevent at least 20 per cent of all cancer incidence.

### **Nutrients as Cancer Prevention Agents**

Epidemiological studies strongly suggest that high intakes of food rich in  $\beta$ -carotene as well as dose rich in vitamin E or C decrease the risk of some cancers.

Vitamin E or tocopherol occur widely in nature and help to stabilise most of the oils derived from plants. The antioxidant activity decreases from delta to alpha tocopherol. Vegetable foods contain considerable amounts of different tocopherols and tocotrienols in their lipid fraction. In soybean oil the active antioxidant is tocopherol.

Whole grains are concentrated sources of antioxidant nutrients, vitamin E. It is an intracellular antioxidant that protects polyunsaturated fatty acids in cell membranes from oxidative damage. It also inhibits the formation of

nitrosamines, especially at low pH levels. Vitamin E prevents the formation of carcinogens from precursor compounds.

Deficiencies of folic acid and vitamins A and C have been associated with the development of cervical dysplasia and cervical cancer.

Dark green leafy vegetables and some citrus fruits also good sources of folic acid. This may have a protective role at the molecular level in cancer development.

Selenium, manganese, zinc, copper and iron are components of the antioxidant enzyme glutathione peroxidase (GSH-Px) is selenium dependent.

Manganese superoxide dismutase and zinc superoxide dismutase (SOD) and catalase are enzyme antioxidants.

Copper, like iron is generally considered as a prooxidant. It is an essential nutrient and a part of copper, zinc - SOD and also ceruloplasmin.

Nutritional copper deficiency may impair antioxidant status by decreasing the activity of these enzymes.

Levels 25-35 per cent below these thresholds predict at least 2-fold high risk. Suboptimal levels of any single antioxidant may increase the relative risk independently.

Dietary deficiencies of selected antioxidant micro-nutrients (B-carotene and vitamins A, C and E) are known risk factors in the pathogenesis of cancer to stop further development. Concurrent correction of suboptimal plasma antioxidant levels is an important part of optimal nutrition at the early stages of cancer.

### **Phytochemicals as Anticancer Agents**

Biologically active substances present in plants are known as phytochemicals.

Studies demonstrate a consistent dose-response relationship between fruit and vegetable intake and the prevention of cancers; higher intake is associated with lower risk.

The association appears to be most marked for epithelial cancers, particularly those of the alimentary and respiratory tracts, a weaker to nonexistent relationship has been noted for hormone related cancers.

A large number of potentially anticarcinogenic agents are found in fruits and vegetables including antioxidant vitamins, fibre and other agents such as dithiolthiones, glucosinolates and indoles, isothiocyanates, flavonoids, phenols, protease inhibitors, plant sterols, allium compounds, and limonene.

These agents have both complementary and overlapping mechanisms of action, including the induction of detoxification enzymes, inhibition of nitrosamine formation, provision of substrate for formation of antineoplastic agents, dilution and binding of carcinogens in the digestive tract, alteration of hormone metabolism and antioxidant effects.

It is unlikely that any one substance is responsible for the anticarcinogenic effect, it is the range of agents in fruits and vegetables that together may offer the observed protection. Thus, fruits and vegetables act through their micronutrients.

### **Resistant Starch**

A strong correlation exists between high intakes of resistant starch present in substances like beans and lower risk of colorectal cancer.

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Resistant starch increases the production of short chain fatty acids. Butyrate may slow the growth of colon tumour cells.

### **Dietary Fibre**

Several mechanisms have been proposed to explain the protective action of dietary fibre against colon cancer.

The fibre dilutes bile acids or binds to it thereby preventing its role in mutation or cell proliferation.

Fermentation of dietary fibre results in production of short chain fatty acids lowering the intestinal pH. This inhibits conversion of primary bile acids to secondary bile acids. This secondary bile acids are believed to promote mutation in intestine.

At low pH the solubility of free bile acids is reduced, diminishing their availability for carcinogenic activity.

Fermentation of dietary fibre results in production of butyrate which has been shown to be antineoplastic.

Dietary fibre exerts its beneficial effect by speeding the passage of faeces through the large intestine so that carcinogens are in contact with the intestinal wall for much shorter period of time.

Additionally the bulk and water of faeces may dilute the carcinogen to a nontoxic level.

Dietary fibre also influences the colonic microbial metabolism, influences fermentation in the colon and the production and distribution of short chain fatty acids in the colon.

It modifies pH, increases the faecal nitrogen and influences mutagens, and faecal enzymes in the colon.

Many human studies find that the consumption of Fructo Oligosaccharides (FOS) increases beneficial bifidobacteria in the gut, while decreasing concentrations of potentially harmful E.coli, clostridia, and bacteroides.

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## **13.7 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS**

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Chemotherapy is the use of chemical agents or medications to treat cancer. The objective of this chemotherapy is to destroy cancer cells throughout the body without causing serious permanent damage to the host.

Chemotherapy is done with the help of some chemotherapeutic agents, and the target of action of these agents is not limited to malignant cells. It affects the normal cell as well. Cells of the body with a rapid turnover such as bone marrow, hair follicles, and the mucosa of the alimentary tract are typically the most affected.

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## **13.8 SUMMARY**

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Cancer can be treated by surgery, chemotherapy, radiation therapy, hormonal therapy, targeted therapy (including immunotherapy such as monoclonal antibody therapy), and synthetic lethality. The choice of therapy depends upon the location and grade of the tumor and the stage of the disease, as well as the general state of the patient (performance status). Cancer genome sequencing helps in determining

which cancer the patient exactly has for determining the best therapy for the cancer. A number of experimental cancer treatments are also under development. Under current estimates, two in five people will have cancer at some point in their lifetime

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### 13.9 KEY WORDS

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- **Malabsorption:** Malabsorption is a disorder that occurs when people are unable to absorb nutrients from their diets, such as carbohydrates, fats, minerals, proteins, or vitamins. Some commonly known disorders related to malabsorption are lactose intolerance and celiac disease.
- **Immunotherapy:** Immunotherapy is the treatment of disease by activating or suppressing the immune system. Immunotherapies designed to elicit or amplify an immune response are classified as activation immunotherapies, while immunotherapies that reduce or suppress are classified as suppression immunotherapies.

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### 13.10 SELF-ASSESSMENT QUESTIONS

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#### Short Answer Questions

1. Explain the effects of cancer therapy.
2. Discuss the Hematopoietic Stem Cell Transplantations
3. Discuss the dietary management of cancer therapy.

#### Long Answer Questions

1. Elaborate on the process involved in chemotherapy.
2. Explain the role of food in the prevention of cancer

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### 13.11 FURTHER READINGS

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# UNIT – 14 NUTRITIONAL MANAGEMENT OF NERVOUS SYSTEM AND MUSCULO SKELETAL SYSTEM

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## Structure

- 14.0 Introduction
- 14.1 Objectives
- 14.2 dysphagia
  - 14.2.1. Nutritional goals for the treatment of a dysphagic patient
  - 14.2.2. Dietary regimens for patients with dysphagia
- 14.3 Epilepsy
  - 14.3.1. pathophysiology
  - 14.3.2. Medical treatment
  - 14.3.3. dietary intervention
- 14.4 Hyperkinetic Behaviour syndrome
  - 14.4.1. Dietary management of nervous system
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- 14.5. Answers to Check Your Progress Questions
- 14.5 Summary
- 14.6 Key Words
- 14.7 Self-Assessment Questions
- 14.8 Further Readings

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## 14.0 INTRODUCTION

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The human musculoskeletal system (also known as the locomotor system, and previously the activity system) is an organ system that gives humans the ability to move using their muscular and skeletal systems. The musculoskeletal system provides form, support, stability, and movement to the body. It is made up of the bones of the skeleton, muscles, cartilage, tendons, ligaments, joints, and other connective tissue that supports and binds tissues and organs together. The musculoskeletal system's primary functions include supporting the body, allowing motion, and protecting vital organs. The skeletal portion of the system serves as the main storage system for calcium and phosphorus and contains critical components of the hematopoietic system.

This system describes how bones are connected to other bones and muscle fibers via connective tissue such as tendons and ligaments. The bones provide stability to the body. Muscles keep bones in place and also play a role in the movement of bones. To allow motion, different bones are connected by joints. Cartilage prevents the bone ends from rubbing directly onto each other. Muscles contract to move the bone attached at the joint. There are, however, diseases and disorders that may adversely affect the function and overall effectiveness of the system. These diseases can be

difficult to diagnose due to the close relation of the musculoskeletal system to other internal systems. The musculoskeletal system refers to the system having its muscles attached to an internal skeletal system and is necessary for humans to move to a more favorable position. Complex issues and injuries involving the musculoskeletal system are usually handled by a physiatrist (specialist in physical medicine and rehabilitation) or an orthopaedic surgeon.

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## **14.1 OBJECTIVES**

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After studying this unit you will be able to

- Define and explain the nervous system and musculo system.
- Brief the causes of nervous system and musculo system.
- Suggest measures to prevent system.

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## **14.2 DYSPHAGIA**

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Dysphagia, or difficulty in swallowing, is a common problem in patients with neurological diseases, often resulting in aspiration pneumonia, compromised nutrient intake, dehydration and malnutrition. The main signs and symptoms of dysphagia include:

- Excessive saliva excretion, choking and coughing during or after meals
- Poor control of tongue, excessive tongue movement and spitting food out of the mouth
- Inability to drink liquids through a straw
- Pocketing of food in cheek or under tongue
- Wet ‘gurly’ voice after eating or frequent throat clearing
- Delayed or absent laryngeal elevation
- Prolonged chewing or eating time
- Chronic or recurrent upper respiratory problems.

### **14.2.1. Nutritional goals for the treatment of a dysphagic patient**

The main goals for the nutritional management of a dysphagic patient are:

- The determination of the safest route for the provision of food, to prevent aspiration and choking.
- The evaluation of the problem and the assessment of the texture of the foods that the patient can tolerate.
- The provision of sufficient energy and nutrient intake, to ensure the best possible nutritional status of the patient.
- The intake of sufficient liquid to prevent dehydration.

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*Self-Instructional Material*

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Consistency	Description
<i>Foods</i>	
Puréed	pudding-like consistency, thick, homogenous textures
Ground or minced	Easily mashed foods, no coarse textures, raw fruits or vegetables (except for raw banana)
Soft or easy to chew	Soft foods prepared without use of blender; meats minced or cut in small pieces, no tough skins, nuts or raw crispy or stinky foods
Modified, general	Soft textures prepared without grinding or chopping; no nuts or crispy foods
<i>Fluids</i>	
Thin	Regular fluids
Nectar-like	Fluids thin enough to be sipped through a straw or a cup, but thick enough to fall off a tipped spoon slowly (e.g. buttermilk, milkshake)
Honey-like	Thick fluids eaten with a spoon, too thick for a straw and unable to hold their shape (e.g. thick yogurt, honey)
Spoon-thick	Pudding-like fluids that must be eaten with a spoon, that can hold their shape on a spoon (e.g. milk pudding, jelly)

### 14.2.3. Dietary regimens for patients with dysphagia

Dysphagia diets must be highly individualized, depending on the patient's chewing and swallowing ability. Foods' texture and viscosity may be altered in order to be tolerated. Fluids and liquids are categorised in four groups, progressing from the easiest to most difficult to swallow. The description of food and fluid consistencies is included in Table 16.1. What are the main problems caused by the texture and density manipulation of foods for dysphagic patients? Food items used in pureed preparations should be thoroughly cooked. This causes the loss of a significant amount of their vitamin and mineral content and often the prescription of a multivitamin is necessary in order to ensure that the patient receives sufficient micronutrient intake. Moreover, constipation is very common among these patients, since texture manipulation through stirring and diluting the food items results in feeds relatively low in dietary fibre. Another problem that can also compromise the nutritional intake of dysphagic patients is the alteration in food appearance and smell following texture manipulation. The use of special equipment that can give special forms to the food and taste enhancers can be used in order to ensure food palatability and better patient compliance.

## 14.3 EPILEPSY

Epilepsy is an intermittent derangement of the nervous system presumably caused by a sudden, excessive, disordered discharge of cerebral neurons. It is estimated that 2.3 million individuals in the United States have epilepsy (200,000 new cases each year); 45,000 children under the age of 15 develop epilepsy each year according to the Epilepsy Foundation in 2006. Direct medical costs for persons with continued seizure activity are reported to be 55% higher than the average costs for all persons with epilepsy (Mandel et al., 2002).

### 14.3.1. Pathophysiology

Most seizures begin in early life, but a resurgence of epileptic events occurs after age 60. The first occurrence of a seizure in adults should prompt investigation into a cause. A clinical workup usually reveals no

anatomic abnormalities, and the cause of the seizure may remain unknown (idiopathic). Seizures before age 2 are usually caused by developmental defects, birth injuries, or a metabolic disease (see Chapters 44 and 45). The medical history is the key component for suggesting further avenues of diagnostic investigation and potential treatments, especially in children. An electroencephalogram can help to delineate seizure activity. It is most helpful in localizing partial complex seizures.

### **14.3.2. Medical Treatment**

The dramatic tonic-clonic (grand mal) seizure is the most common type of a seizure (lasting 1 to 2 minutes), yet numerous classifications of seizures, each with a different and often less dramatic clinical presentation, exist. A generalized seizure is one that involves or appears to involve the entire brain cortex from its beginning phases. The tonic-clonic seizure comes under this heading. After such a seizure the patient wakes up slowly after a time; he or she will be groggy and disoriented for minutes to hours after the event. This is termed the postictal phase and is characterized by deep sleep, headache, confusion, and muscle soreness.

The absence seizure (petit mal) is also generalized in nature. A patient with absence seizures may appear to be daydreaming during an episode, but he or she recovers consciousness within a few seconds and has no postictal fatigue or disorientation. Partial seizures occur when there is a discrete focus of epileptogenic brain tissue. A simple partial seizure involves no loss of consciousness, whereas a complex partial seizure is characterized by a change in consciousness. Failure of partial seizure control may prompt consideration of seizure surgery. A localized focus resected from nonessential brain renders a patient seizure free in 75% of cases. Determining the seizure type is key to implementing effective therapy. Generalized seizures are ordinarily managed with valproate or phenytoin. Phenytoin metabolism has unusual kinetics; thus toxic levels may be attained with very small dosage adjustments. These drugs interact with other drugs metabolized in the liver and may cause liver damage. Liver enzymes and serum drug levels must be monitored periodically. Gabapentin has been introduced recently, and it is rapidly gaining popularity because of its safety and ease of use. Carbamazepine or phenytoin can usually control partial seizures.

Medications used in anticonvulsant therapy may alter the nutrition status of the patient. Phenobarbital has been associated with decreased intelligence quotient (IQ) when used in children. It is occasionally considered for use after failure of other antiepileptic drugs. Phenobarbital, phenytoin, and primidone interfere with intestinal absorption of calcium by increasing vitamin D metabolism in the liver. Long-term therapy with these drugs may lead to osteomalacia in adults or rickets in children. Vitamin D supplementation is recommended. Folic acid supplementation interferes with phenytoin metabolism; thus it contributes to difficulties in achieving therapeutic levels. For this reason sporadic folic acid supplementation should be avoided. Phenytoin and phenobarbital are bound primarily to albumin in the bloodstream. Decreased serum albumin levels in malnutrition or with reduced albumin synthesis secondary to advanced cirrhosis limit the amount of drug that can be bound. This results in an increased free drug concentration and possible drug toxicity with a

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*Self-Instructional Material*

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standard dose. New treatment guidelines for medication use and for preventing photic- and pattern-induced seizures have been released by the Epilepsy Foundation. These guidelines emphasize the public health nature of seizure management and the special needs of women and older Americans in optimizing strategies. Use of just one antiseizure medication is recommended initially, resorting to combination therapies only when needed. Continuous enteral feeding slows the absorption of phenytoin, thus necessitating an increase in the dose to achieve a therapeutic level. Decreased serum phenytoin concentrations associated with enteral feeding may increase the risk of seizures; a patient-specific care plan that includes consideration of the enteral feeding formulation and method of administration, as well as the phenytoin dosage form, schedule of administration, and monitoring, is needed (Au Yeung and Ensom, 2000). Recommendations to separate phenytoin suspension from tube-feeding formulas are common. Stopping the tube feeding before and after the phenytoin dose is generally suggested. The most common recommendation is a 2-hour feeding-free interval before and after the dose of phenytoin is administered (Au Yeung and Ensom, 2000). Whenever tube feedings are stopped, the dose of phenytoin needs to be adjusted to avoid toxicity. Absorption of phenobarbital is delayed by the consumption of food; therefore administration of the drug must be staggered around mealtimes if it is used. Medication therapy A ketogenic diet has been used for treatment of all types of seizures in children in whom drug therapies have failed. This diet is also used in the management of several inborn errors of metabolism (Roman, 2006). The ketogenic diet is financially beneficial, particularly in comparison to total costs for care (Mandel et al., 2002). A report that evaluated medical costs for children (2 to 28 years of age) with drug refractory epilepsies demonstrated cost advantage, reduction in seizures, and a reduced need for drugs with the use of a ketogenic diet (Mandel et al., 2002). The ketogenic diet has minimum side effects. However, data on the long-term impact on growth or cardiovascular risk are lacking. One recent report on growth change in children did find that subjects on the ketogenic diet showed a growth delay; additional research is needed (Peterson et al., 2005). Although the diet is initially demanding, it completely controls epilepsy in one third of the children whose seizures are otherwise uncontrollable. Practice guidelines released by the American Academy of Neurology and the American Epilepsy Society can be retrieved from the Epilepsy Foundation website (Epilepsy Foundation, 2006). The diet is designed to create and maintain a state of ketosis (Bough and Rho, 2007). Although its mechanism of action is not clearly understood, the beneficial effect in epilepsy may be caused by a change in neuronal metabolism, whereby a ketone body behaves as an inhibitory neurotransmitter thus producing an anticonvulsant effect on the body. Two forms of the ketogenic diet are in use: the "traditional" approach, developed in the 1920s, and the medium-chain triglyceride (MCT)-based approach (Liu et al., 2003). With either approach the child fasts in the hospital for 24 to 72 hours until a 4+ ketonuria is produced. For the majority of patients, if the diet is going to work, it usually works during the initial fasting period. It should also be noted that antiepileptic drugs need to be stopped when administering the

ketogenic diet. In the traditional approach, once ketosis is established, caloric intake is resumed in a ratio of 4:1 for fat kcal:protein/ carbohydrate kcal in the diet. For a child the diet is calculated so that 75% of the kilocalories are from fat. Protein is calculated to provide appropriate intake for growth (about 1 Skg/ day). Carbohydrates are added to make up the remaining portion of protein and carbohydrate calories, which is usually a minimum to negligible amount. The Exchange Lists (see Appendix 34) can be used to adjust the carbohydrate amount. A multiple vitamin/mineral and extra calcium supplement is recommended to ensure that the diet is nutritionally complete; this should be provided in a sugar-free form. Mild dehydration is used to prevent dilution of the level of ketones circulating at any time (Berryman, 1997). Fluids are carefully controlled not to exceed 2 L/day (Kinsman et al., 1992). The MCT-based ketogenic diet replaces the long-chain fats of the traditional diet with MCT oil. MCT oil is an odorless, colorless, tasteless oil and was originally used as a means of improving the palatability of the diet. A greater amount of nonketogenic foods such as fruits and vegetables and small amounts of bread and other starches can be allowed because ketosis from MCT can be more readily achieved (Table 41-8). Fluids are not limited in the MCT ketogenic diet. Initiating the ketogenic diet is intense. Further, the diet may seem unpalatable as well as complex, thus making compliance difficult to achieve. To be successful, children may benefit from behavioral techniques, whereas parents most often require substantial psychosocial support. Attention required during the follow-up phase varies and is affected by the patient's health status, growth, and development and the caregiver's level of anticipation. For the child whose epilepsy is controlled on the diet, complying with the diet is much easier than dealing with devastating seizures and associated injuries. Fortunately the duration of the diet is limited; it can often be discontinued after 2 to 3 years.

Hyperkinetic disorder is the generic ICD-10 (WHO, 1992) term used to describe one of the most common childhood psychiatric disorders. It is a severe form of a syndrome which is referred to in DSM-IV (APA, 1994) and the American literature as attention deficit hyperactivity disorder (ADHD). Hyperactivity or hyperkinesis can be defined as "an enduring disposition to behave in a restless, inattentive, distractible and disorganised fashion" (Taylor, 1994). It is thus more than motor overactivity. Diagnostically there are three main groups of symptomatology: overactivity, inattentiveness and impulsiveness. Overactivity is either a general increase in the tempo and therefore amount of purposeful activity; an increase in the number of purposeless minor movements which are irrelevant to the task at hand (fidgeting); or an increase in the number of purposeless whole body movements (restlessness). It includes excessive talkativeness and noisiness. Inattentiveness refers to difficulties in focusing and sustaining attention. This results in careless mistakes, failure to sustain or follow through on set tasks, particularly if these contain cognitive demands. Typically tasks are left unfinished. In addition, the affected individual may be demonstrably distractible and often poorly organised. Impulsiveness is common but not necessary for the clinical diagnosis (although it is included in the research criteria). It is characterised by interrupting others, blurting out answers, failure to wait for one's turn and

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excessive talk beyond normal social constraints. It commonly has a quality of impatient social disinhibition. In some individuals it is mainly evident in reckless behaviour; things are done suddenly without heed for danger or adverse consequences. To make the clinical diagnosis, there must be abnormalities in both attention and overactivity which (a) are excessive compared with the norm for a child of that age or developmental ability; (b) have been present from an early age (< 6 years), and (c) are pervasive, i.e. present in more than one type of social situation. They are most obvious in situations which demand a measure of self-control and /or persistence with cognitive tasks. It is a difficult diagnosis to make with confidence in pre-school children.

In order to satisfy ICD-10 research diagnostic criteria, hyperactive must have abnormalities on three axes: with at least six inattention symptoms, three of the hyperactivity symptoms and one of the impulsivity symptoms. The age before which signs must be evident is a little higher (<7 years) than that used for the clinical diagnosis.

#### **14.3.3. Dietary Intervention:**

Research into the effect of food on ADHD started forty years ago when pediatric allergist Benjamin Feingold hypothesized that both artificial food additives (colorings and flavors) and foods rich in salicylates (chemicals occurring naturally in some foods ) might be important etiologic agents' of the hyperkinetic syndrome . The Feingold studies were followed by other elimination diet studies , investigating the effects of either artificial food color (AFC) elimination or of a diet eliminating many foods and additives, i.e. the few-foods diet (FFD), and by supplement studies investigating the effects of vitamins, minerals and polyunsaturated fatty acids (PUFA) on ADHD. Recent reviews on ADHD and diet interventions. The efficacy of diet treatments in ADHD was recently evaluated in three reviews . The main aim of reviews is to summarize the evidence on a specific topic, of which both researchers and clinicians may benefit . However, the three reviews show divergent conclusions, i.e.: 1) there is evidence for a small effect of PUFA on ADHD, while the potential effect of AFC elimination remains unclear and more research is needed for a FFD ; 2) there is emerging consensus for the effect of food additives elimination (concurrently providing a food additive list to be given to a patient), while a one-week FFD is indicated in case of comorbid food allergy symptoms and 3) none of the diet interventions are recommendable as ADHD treatment .

Numerous studies have demonstrated that the behavior of some children improves when they avoid certain foods. Those children may react to any of a variety of different foods and ingredients, and some may be affected by more than one. Your goal is to identify the specific foods or additives, if any, that affect your child. What makes that task especially challenging is that children's behavior ordinarily is so variable. Needless to say, controlling the diet of young children can be difficult, especially once children go to school. Foods containing dyes and other potentially provoking ingredients are advertised aggressively and available everywhere: at supermarkets, restaurants, schools, vending machines, parties, theaters, and the homes of friends and relatives. Many young children are already "hooked" on the very foods that may cause problems, though it is

getting easier to find acceptable alternatives. And children who do not eat what all their friends eat may feel left out or stigmatized. Some parents who have put their children on special diets, though, say that their children willingly cooperate in making dietary changes, especially after they discover that those changes make them feel better. Some older children avidly read labels to avoid certain ingredients. Some studies suggest that the children who respond best to dietary therapy are young (preschool) and those who suffer from asthma, eczema, hives, hay fever, or similar symptoms. Children who still have significant problems after taking stimulant medications might also be good candidates. But, no matter the age of your child or the exact nature of his or her behavioral problem, it could be worth trying diet. It is certainly safer and cheaper than using stimulant drugs, and, if your child has been eating a lot of artificially colored foods, it may also be more nutritious. At worst, a modified diet won't help and you've delayed for several weeks trying another option. Trying a modified diet. Finding a diet that will help child will require time, patience, and experimentation. We discuss diets that involve varying degrees of change, starting with eliminating only dyes. The most restricted diets begin by eliminating numerous common foods and then add them back one by one to identify any that cause problems. Numerous studies have demonstrated that some children are sensitive to dyes. Thus, you might start by eliminating only foods (and vitamins, drugs, and toothpastes) that contain artificial colorings.

The Feingold diet, which is based mostly on unconfirmed reports from parents and doctors, eliminates additional additives, as well as "salicylate-containing" foods. That diet eliminates:

- artificial colorings (look for names like Red 40 and Yellow 5 on labels)
- artificial flavorings (including vanillin, used in synthetic vanilla)
- artificial sweeteners (acesulfame-K, aspartame, saccharin, sucralose)
- BHA, BHT, and TBHQ preservatives.

One study suggests that sodium benzoate and benzoic acid should also be on that list. The Feingold diet also excludes certain fruits and vegetables, though, again, studies have not demonstrated that they cause problems. While that diet excludes many common foods, later you can add back any to which child is not sensitive. Once you have decided which foods and additives you will eliminate, check all the foods in your refrigerator, pantry, and cupboards. Remove or discard any foods that contain banned ingredients. Learn about the ingredients used by the restaurants you frequent, though during the test period it may be best to stick mostly to foods prepared at home. Major fast-food chains offer lists of the ingredients in their products; ask servers or call their consumer-affairs offices. The Feingold Association publishes lists of selected packaged and chain-restaurant foods that fit into the diet, but you'll have to become a careful label reader and inquisitive restaurant-goer to learn the ingredients in children's favorite foods.

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## 14.4 HYPERKINETIC BEHAVIOUR SYNDROME

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Hyperkinetic disorder is an outdated term for a psychiatric neurodevelopmental condition emerging in early childhood. Its features included an enduring pattern of severe, developmentally inappropriate symptoms of inattention, hyperactivity, and impulsivity across different settings (e.g., home and school) that significantly impair academic, social and work performance. It was classified in the World Health Organization's ICD-10 and was roughly similar to the "combined presentation" of attention deficit hyperactivity disorder in the American Psychiatric Association's DSM-5. However, in the ICD-11 the entry for hyperkinetic disorder no longer exists and is replaced by *attention deficit hyperactivity disorder*.

Hyperkinetic people display disorganized, poorly controlled and excessive activity; they lack perseverance in tasks involving thought and attention, and tend to move from one activity to the next without completing any. They are frequently accident-prone, reckless and impulsive, and may thoughtlessly (rather than defiantly) break rules. Cognitive impairment and delayed language and motor development are more common in this group than in the general population; and they may experience low self-esteem and engage in dissocial behavior as a consequence of the disorder.

While hyperkinetic children are commonly incautious and unreserved with adults, they might be isolated and unpopular with other children.

Hyperkinesis is one of the more prevalent psychological disorders related to learning problems in school-aged children. While the estimates of prevalence of hyperkinesis vary from three percent to 15 percent, depending on the studies reviewed, the more frequently cited rates range between five percent and ten percent. As school psychologists and educators have become more aware of the concept and treatment of hyperactivity, more children have been screened for this disorder. The diagnosis and management of hyperkinesis have become a , three-ring arena involving the medical profession, the schools, and the home, with critics watching and commenting on the show. Hyperkinesis is difficult to define and diagnose, consequently there is no universally accepted definition of this entity. However, if one focuses on the hyperactive behaviours of children who are of normal intelligence and free of underlying physical disorders, the most commonly mentioned symptoms are overactivity, distractibility, restlessness and short attention span.

### 14.4.1. Dietary management of nervous system

We all know eating healthy is important for our mental and physical well being. But sometimes our bodies require some specific nutrients to be more active and healthy. Brain being a part of our body also requires some important nutrients to function properly. Here we bring to you a list of foods that will help you improve the functioning of your brain and nervous system, especially the gray matter.

#### Green leafy vegetables

Green leafy vegetables are rich in Vitamin B complex, Vitamin C, Vitamin E and Magnesium all of which are important for proper functioning of our nervous system. Vitamin B is essential in the process of synthesizing and

circulating neurotransmitters, which are brain chemicals that regulate heartbeat, respiration and digestion. Magnesium helps in calming the nerves. Vitamin E and C acts as anti-ageing for the nervous system.

### **Fish**

Nerves are protected by myelin sheaths, which contain very high level of fatty acid. So, people who are deficient in fatty acids may suffer from damage of the nerves. Fish has Omega 3 fatty acid and thus helps in healing of the nerves and nervous system.

### **Dark chocolate**

Not all chocolates are manufactured equally. In fact, 70 per cent of chocolates available in the market are highly processed and barely have any benefits. Dark chocolates are full of flavonols which have anti-inflammatory and anti-oxidant property. These properties help to lower the blood pressure and improve the blood flow, to both brain and heart. Skip milk and white chocolates and go for a minimally processed dark chocolate with at least 70 percent of cocoa. This will ensure that you get its brain benefits!

### **Broccoli**

Broccoli is rich in Vitamin K which is known to improve brain power and cognitive skills. Many studies have reported that because broccoli is rich in a compound called glucosinolates which can slow the breakdown of the neurotransmitter, acetylcholine, which the central nervous system needs to perform its function properly, keeps our brain and memory sharp. Low levels of acetylcholine are linked with Alzheimer's.



### **Eggs**

A study conducted at Boston University tracked 1400 healthy adults for 10 years who consumed eggs daily, and the results found that regular egg intake resulted in better performance on some memory tests. Eggs are rich in choline and B vitamin. When you consume eggs, the choline in them is used by the brain to make acetylcholine, a neurotransmitter that is important for memory and communication among brain cells.

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### **Salmon**

As we have already mentioned, omega 3 fatty acid has an important role to play in cognitive functioning. According to New York Times, Journal neurology, low level of omega 3 fatty acid is linked with smaller brain volume and poor mental performance.

Salmon is a rich source of omega 3 which can strengthen the brainpower. According to a research conducted at the University of Pittsburgh, adults under the age of 25 who increased their omega-3 intake over six months improved their test scores, measuring the working memory.

### **Avocados**

Rich in both Vitamin K and folate, avocados help prevent blood clot in the brain and thus protects you from stroke. Apart from that, avocados also help to improve memory and concentration. The best thing about avocados is they have the highest protein and lowest sugar content as compared to any other fruit.

### **Almonds**

Almonds have high levels of brain-healthy omega-3 fatty acids and lots of brain-protecting vitamin E just like the salmon fish.

### **Pumpkin seeds**

Pumpkin seeds are an excellent source of magnesium, copper, iron, and zinc. Apart from all this, pumpkin seeds contain powerful antioxidants that protect the body and brain from free radical damage. Magnesium is essential for learning and memory and low magnesium levels are linked to many neurological diseases, including migraines, depression, and epilepsy. Your brain uses copper to help control nerve signals. And when copper levels are out of whack, there's a higher risk of neurological disorders, such as Alzheimer's. Zinc is crucial for nerve signaling and its deficiency has been associated with many neurological conditions, including Alzheimer's disease, Parkinson's disease and depression.

### **Nuts**

A study conducted in 2014 showed that nuts can improve cognition skills and even help prevent neurological disorder. Also, another study found that women who ate nuts regularly over the course of several years had a sharper memory, compared to those who didn't eat nuts at all. There are several nutrients in nuts, such as healthy fats, antioxidants, and vitamin E, all of which are good for brain health. Vitamin E shields cell membranes from free radical damage thus helps to slow the mental decline. While all nuts are good for your brain, walnuts are considered a better choice, since they also contain omega-3 fatty acids.

#### **14.4.2. Foods for healthy bones and muscles**

We'll be looking at the typical nutrient content of a range of different foods and have provided this information in tables. This is a lot to take in at once so we've also provided this information as a PDF which you can download from the bottom of the page for later reference.

### **Calcium**

Good sources of calcium include:

- Milk, cheese and other dairy foods including cheese, yoghurt and ice cream
- Green leafy vegetables such as broccoli, cabbage and okra (but not spinach)
- Soya beans, tofu and soya drinks with added calcium
- Nuts
- Bread and anything made with fortified flour
- Fish where you eat the bones – such as sardines and pilchards.

Many cereal products in the UK are fortified with calcium carbonate. The Bread and Flour Regulations require that, subject to certain exceptions, calcium carbonate must be added to all wheat flour, whether or not mixed with other flour, for fortification. Calcium carbonate does not need to be added to self-raising flour which has a calcium content of not less than 0.2%, to wholemeal flour or to wheat malt flour.

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### **Vitamin D**

Good food sources of vitamin D are:

- Oily fish – such as salmon, sardines, herring and mackerel
- Red meat
- Liver
- Egg yolks
- Fortified foods such as most fat spreads and some breakfast cereals

In the UK, cows' milk is generally not a good source of vitamin D because it isn't fortified, as it is in some other countries. A number of yoghurt products are available fortified with vitamin D.

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### **Protein**

In the UK, about one-third of dietary protein comes from plant sources and the other two-thirds comes from animal sources. The protein content of nuts, dried peas, and beans is high and compares favourably with the protein content of meats. After soaking in water, the proportion of protein in dried peas and beans is reduced, but they still are an excellent source of protein..

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## **14.5 ANSWERS TO CHECK YOUR PROGRESS QUESTIONS**

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Dysphagia, or difficulty in swallowing, is a common problem in patients with neurological diseases, often resulting in aspiration pneumonia, compromised nutrient intake, dehydration, and malnutrition.

The main goals for the nutritional management of a dysphagic patient are:

- \_ the determination of the safest route for the provision of food, to prevent aspiration and choking.
- \_ the evaluation of the problem and the assessment of the texture of the foods that the patient can tolerate.

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\_ the provision of sufficient energy and nutrient intake, to ensure the best possible nutritional status of the patient.

\_ the intake of sufficient liquid to prevent dehydration.

Epilepsy is a central nervous system (neurological) disorder in which brain activity becomes abnormal, causing seizures or periods of unusual behavior, sensations, and sometimes loss of awareness. Anyone can develop epilepsy.

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## 14.6 SUMMARY

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Receptors in muscles provide the brain with information about body position and movement. The brain controls the contraction of skeletal muscle. The nervous system regulates the speed at which food moves through the digestive tract. Functions of the Nervous System -The nervous system has 3 main functions: sensory, integration, and motor. Sensory. The sensory function of the nervous system involves collecting information from sensory receptors that monitor the body's internal and external conditions

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## 14.7 KEY WORDS

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- **Epilepsy:** a neurological disorder marked by sudden recurrent episodes of sensory disturbance, loss of consciousness, or convulsions, associated with abnormal electrical activity in the brain.
- **Dysphagia:** difficulty or discomfort in swallowing, as a symptom of disease.

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## 14.8 SELF ASSESSMENT QUESTIONS

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### Short Answer Questions

1. Define and explain the impact of dysphagia.
2. Describe epilepsy.
3. How to prevent dysphagia and epilepsy?

### Long Answer Questions

1. Explain the dietary management of dysphagia.

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## 14.9 FURTHER READINGS

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