UNIT 3 CARBOHYDRATES

Structure

3.1 INTRODUCTION

In the previous unit we have studied about the energy requirements. You now know that energy must be supplied regularly to individuals through the diet for their survival and maintenance of life and to carry out all activities jointly termed as work. Our body derives energy from the catabolism of energy-yielding nutrients: carbohydrates, lipids and proteins. Ainong these, carbohydrates are the single most abundant source of dietary energy comprising 50-70% of the total energy intake in different populations. In this unit we will learn about the different types of carbohydrates, their utilization by the body and how glucose homeostasis is maintained. In addition the health benefits of dietary fibre and carbohydrates not absorbed in the small intestines, will also be covered in this unit.

Before going through this unit it is important for you to revise the sections on chemistry of carbohydrates, their structures and different metabolic pathways covered under Nutritional Biochemistry Course (MFN-002) in Unit 1 and 6, respectively.

Objectives

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

- classify carbohydrates,
- describe their importance,
- critically evaluate their role in nutrition,
- explain the digestion, absorption and utilization of carbohydrates in the body,
- elaborate on the recent advances in the area of non-digestible carbohydrates, and
- discuss the role of carbohydrates in health and certain disorders.

3.2 CLASSIFICATION OF CARBOHYDRATES

Carbohydrates are defined as polyhydroxy aldehydes or ketones or substances that produce such compounds when hydrolyzed. Carbohydrates are veiy diverse organic molecules and can be classified based on their:

a) Molecular size / degree of polymerization (DP)

b) Digestive fate

You may recall studying about the classification of carbohydrates in the Nutritional Biochemistry Course. The following brief discussion will help you to brush up / recapitulate the information presented in the Nutritional Biochemistry Course.

3.2.1 Classification on the Basis of Degree of Polymerization (DP)

Carbohydrates are classified by their degree of polymerization as follows:

1) *Sugar* (DP: 1-2): *Monosaccharide* (consisting of *a* single unit of sugar. Also known as simple sugar) and *Disaccharides* (consisting of *2* monosaccharide)

2) *Oligosaccharides (DP:* 3-9): Each molecule containing 3-9 monosaccharide units.

3) *Polysaccharides* (DP: > *9):* Each molecule containing more than 9, but usually several monosaccharide units.

A brief review follows: Examples of different classes of carbohydrates and their sources are also given in Table 3.1.

1) *Sugars:* This group includes monosaccharide and disaccharides. List down the names of sugars under both these groups and check it out with those given here,

Monosaccharides (DP: 1) are structurally the simplest form of carbohydrates and cannot be reduced in size to smaller units by hydrolysis. They are grouped according to the number of carbon atoms per molecule i.e. *trioses, tetroses, pentoses, hexoses*. Nutritionally the most important carbohydrates are *hexoses,* Metabolically, the most important hexose is *glucose.* The *disaccharides (DP:* 2) are sources of energy in the diet. *Sucrose* and *lactose* are more abundant than inaltose or malt sugar.

2) *Oligosaccharides* (DP: 3-9) Oligosaccharides consists of short chains of monosaccharide units joined by covalent bonds. The glycosidic bond may be α or β in orientation. The number of units is designated by the prefixes - tri, tetrn followed by 'saccharides'.

Table 3.1: Different types **of** carbohydrates

S.No.	Class	Examples	Component Monosaccharide	Sources
1.	Monosaccharides	Glucose Fructose Galactose		Fruits, honey etc. Fruits, honey etc. Milk and products
2.	Disaccharides	Maltose Lactose Sucrose	Glucose (2 molecules) Glucose + Galactose Glucose + Fructose	Glucose syrup Milk and its products Sugarcane, sugar used as additive
3.	Trisaccharides	Raffinose Maltotriose	Glucose, Fructose, Galactose Glucose (3 molecules)	Chick peas, legumes, pulses Glucose syrup
4.	Tetrasaccharides	Maltotetrose Stachyose	Glucose (4 molecules) Galactose (2 molecules), Glucose, Fructose	Glucose syrup Beans, Legumes
5.	Pentasaccharides	Verbascose		Beans, Legumes
6.	Sugar alcohols	Sorbitol Xylitol Mannitol		Exclusively present if used as food ingredients

3) Polysaccharides (DP: > 9) Polysaccharides are high molecular weight polymers of monosaccharide units formed by glycosidic bonding. They may be long unbranched inolecules or branched molecules. If the structure is composed of a single type of monosaccharide unit, it is referred to as a 'homopolysaccharide'. If two or more different types of monosaccharides make up its structure, it is called 'heteropolysaccharides.'

Let us now have a look at another way of classifying carbohydrates.

3.2.2 Classification Based on Digestive Fate of Carbohydrates

The digestive fate of carbohydrates depends on their inherent chemical nature (monosaccharide composition and type of linkage between sugars) and on the supramolecular structures within foods of which they are a part.

Mc *Cance* and Lawrence in 1929 were first to classify carbohydrates as 'available' and 'unavailable'. According to them, carbohydrates that are digested to constituent monosaccharides and absorbed fell under the category of 'available' carbohydrates. Carbohydrates that are not digested by the endogenous enzymes of the human inteslinal tract and therefore not absorbed were classified as 'unavailable' carbohydrates.

However, these undigested carbohydrates enter the colon and are fermented by microflora. It is realized now that it is misleading to use the term 'unavailable' carbohydrates because some indigestible carbohydrates can provide body with energy through fermentation in the colon. Therefore, 'unavailable' carbohydrates are not really 'unavailable' and the term '*Non Glycemic* Carbohydrates' is suggested for these by FAO (Food and Agriculture Organization) and WHO (World Health Organization). Non glycemic carbohydrates include the raffinose series of oligosaccharides, non-starch polysaccharides (NSP), some disaccharides such as lactulose and resistant starch (RS). You may recall reading about RS in the Principles of Food Science Course (MFN-008) in Unit 2. Here, we will get to know more about RS and the other non-glycemic carbohydrates.

Similarly, carbohydrates that are digested to monosaccharides and absorbed as such in the small bowel are termed as 'Glycemic' carbohydrates. They include disaccharides, starch, maltodextrin and glycogen.

Let us now study the functions of carbohydrates in the body in the following section.

3.3 FUNCTIONS

Carbohydrates in the body function primarily in the form of glucose, although a few have structural roles. Important functions of carbohydrates are listed below:

- Source of energy: Glucose is a major source of energy for all the body cells. One gram of carbohydrate provides 4 Kcal. RBCs are particularly dependent on glucose. It is also indispensable for the maintenance of functional integrity of the nerve tissue and under normal circumstances; it is the sole source of energy for the brain.

  Similarly, glucose is important for heart muscles. Although fatty acids are the preferred regular fuel of heart muscle, glycogen in cardiac muscle is an important emergency source of contractile energy. In a damaged heart, poor glycogen stores or low carbohydrate intake may cause cardiac symptoms of angina.

- Protein sparing effect: Carbohydrates help in regulating the protein metabolism. Presence of sufficient carbohydrates to meet energy demands prevents the channeling of too much protein for this purpose. This protein sparing action allows the major portions of protein to be used for its basic structural purpose of tissue building. Therefore, patients who are unable to eat are temporarily administered 5% glucose solution intravenously.

- *Antiketogenic effect*: Presence of carbohydrates is necessary for normal fat metabolism. In the absence of sufficient carbohydrates, larger amounts of fat are used for energy than the body is equipped to handle. This results in incomplete oxidation and accumulation of ketone bodies. This may in turn lead to acidosis, sodium imbalance and dehydration. In extreme conditions such as starvation (carbohydrates are inadequate) and uncontrolled diabetes (carbohydrates are unavailable for energy needs), ketoacidosis is a common coinplication.
- *Excretion of toxins:* Glucuronic acid, a metabolite of glucose, combines with chemical and bacterial toxins and some normal metabolites in the liver and thereby helps in their excretion.
- *Act as precursors:* Carbohydrates and their derivatives serve as precursors to compounds such as nucleic acid, connective tissue matrix and galactoside of nerve tissue.
- *Overall positive health:* Non-glyceinic carbohydrates including non-starch polysaccharides are beneficial for the functions and physiology of gastrointestinal tract and thus have a positive effect on.the overall health.

Now that you have gone through the functions, let us move on to the mechanism of the digestion in the following section.

3.4 DIGESTION AND ABSORPTION

You are aware that 60-70% of energy is supplied by the dietary carbohydrates which are primarily present as polysaccharides (starch) followed by disaccharide and free monosaccharide. But the monosaccharides are present in very small amounts in our diet. To be absorbed from the gut, these carbohydrates must be broken down to their constituent monosaccharide units. Let us now briefly review how these carbohydrates are digested in the gut.

The hydrolytic enzymes involved in the digestion of carbohydrates are collectively called 'glycosidases' or 'carbohydrases'. The major carbohydratase enzyme secreted by the salivary glands and the acinar cells of the pancreas is the endo-glycosidase - *amylase*. This enzyme hydrolyzes a-1, 4-linkages in amylose and amylopectin to yield maltose, maltotriose and dextrins. The further hydrolysis of these and dietary sucrose and lactose are brought about by 'oligosaccharidases', which are expressed on the apical membrane of the epithelial cells on the small intestinal villi. Figure 3.1 clearly illustrates the digestion of carbohydrates.

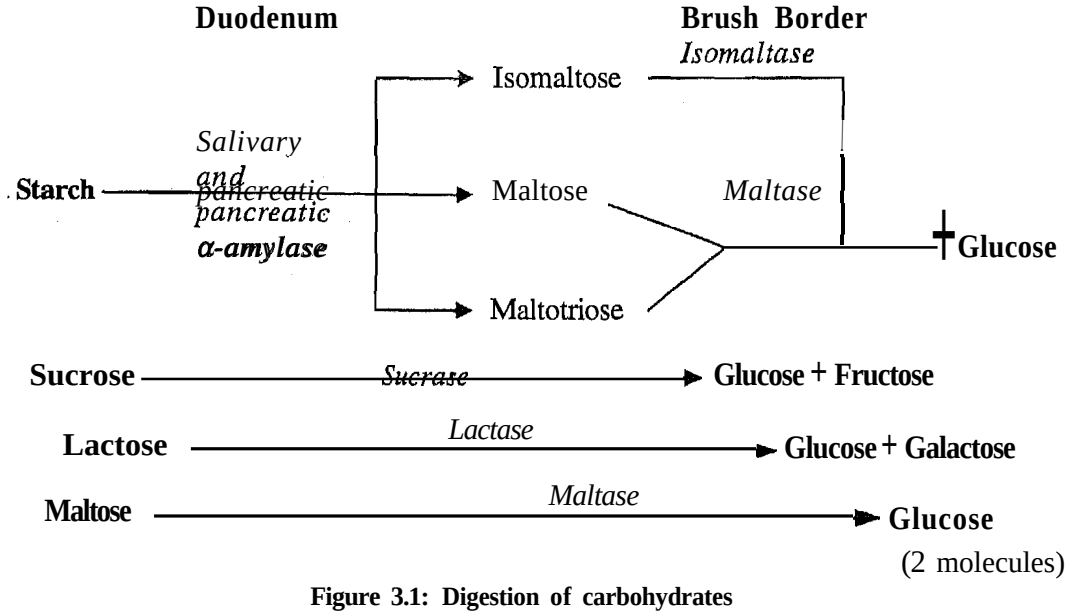

Figure 3.1: Digestion of carbohydrates

Let us now learn how these monosaccharides are absorbed from the gastrointestinal tract.

Glucose and galactose are absorbed into the mucosal cells by *active transport* which requires energy. The carrier of glucose and galactose is a specific protein complex known as 'sodium-glucose transport protein-1' (SGLT-1), which is dependent on Na^+/K^+ ATPase pump. Glucose and galactose cannot attach to the carrier until it has been preloaded with sodium as illustrated in Figure 3.2. Hence, you would realize that oral rehydration syrup (ORS) always contains sodium chloride and glucose / sugar. *Fructose* is absorbed by a *facilitated transport,* involving a specific transporter– GLUT-5.

Another transport protein called GLUT-2, present on the basolateral membrane shuttles all three monosaccharides from enterocytes towards the blood vessel. Study Figure **3.2** for a clearer understanding of the process of absorption.

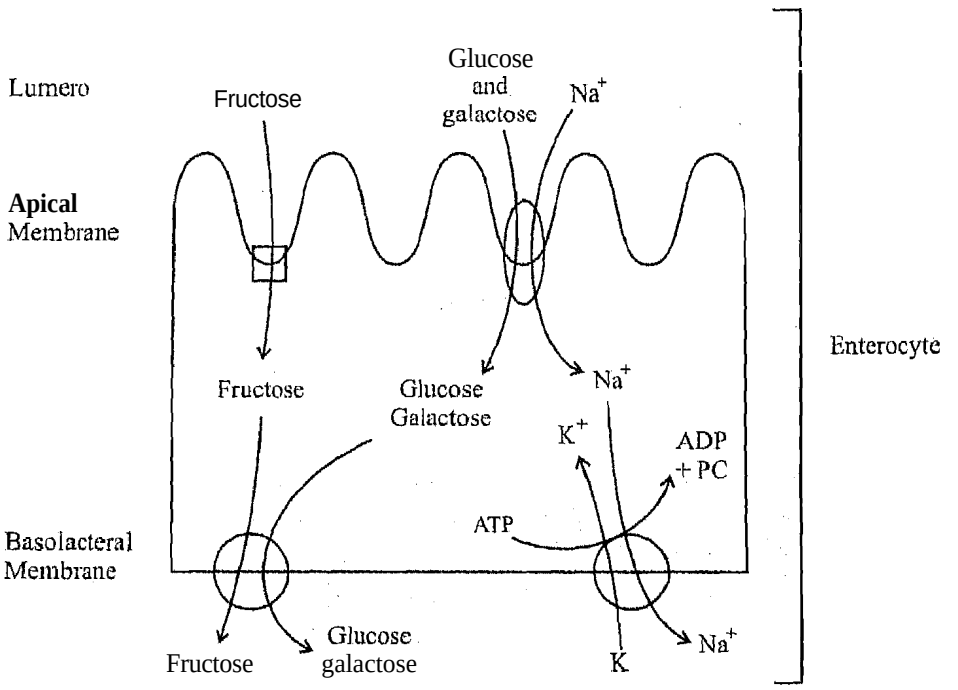

Figure 3.2: Absorption of carbohydrates across apical membrane

Before moving on to the utilization of carbohydrates, let us have a look at the disorders of carbohydrate absorption.

Carbohydrate Malabsorption

Carbohydrates malabsorption is usually caused by *an* inherited or acquired (in intestinal infection, celiac disease, PEM) defect in the brush border *oligosaccharidases,* the most common being 'lactose intolerance'. In such individuals, ingestion of lactose leads to passage of the sugar to the large bowel, where it is fermented to produce short chain fatty acids (SCFA) and gases. In humans, lactase activity declines as individuals grow.

3.5 METABOLIC UTILIZATION.OF CARBOHYDRATES

Following absorption, the monosaccharides enter the portal circulation and are carried to the liver. Both galactose and fructose are converted to glucose in the hepatocytes. A part of glucose is converted into glycogen while some is catabolized for energy in the liver. The remainder of glucose passes on into the systemic blood supply and is distributed among other tissues such as skeletal muscle, adipose tissue and kidney. Nearly all the cells in the body admit glucose passively by a carrier-mediated transport mechanism that does not require energy. The protein carriers involved in the process are called glucose transporters and are abbreviated GLUT. A brief description of human GLUT is given in Box 3.1.

BOX 3.1 Human Glucose Transporters (GLUT)

All GLUTs are the integral proteins, which penetrate and span the lipid bilayer of plasma membrane. Six isomers of GLUT have been described, the tissue distribution of which is given below:

Type	Major Sites of Expression
GLUT 1	Erythrocytes, blood, brain barrier, placenta
GLUT 2	Liver, β cells of pancreas, kidney, small intestine
GLUT 3	Brain
GLUT 4	Adipocytes, heart, skeletal muscle
GLUT 5	Small intestine
GLUT 7	Endoplasmic reticulum of hepatocytes

Among these, GLUT 4 is sensitive to insulin and its concentration increases in response to the hormone. Insulin resistance observed in NIDDM patients is believed to arise from abnormalities in the synthesis and activity of GLUT-4.

After going through the contents in Box 3.1, it must be clear to you that cells of brain, erython, liver and pancreas do not require insulin in order to permit entry of glucose. On the other hand, entry of glucose in the cells of adipose tissue and skeletal muscle is insulin dependent.

The metabolic fate of glucose in different tissues depends to a great extent on the body's energy demands. The major regulatory mechanisms are hormonal, involving the action of hormones and allosteric enzyme activation and suppression.

Further, the nature of diet especially with respect lo carbohydrates modifies the activity of lipogenic pathway. A diet rich in carbohydrates stimulates lipogenic pathway, whereas starvation or a diet rich in lipids and poor in carbohydrates decreases the function of lipogenic enzyme. You have already studied metabolic pathways of carbohydrates in detail in Nutritional Biochemistry Course in Unit 6. For recapitulation, an integrated overview of these pathways is shown in Figure 3.3. For details we suggest you get back to the Nutritional Biochemistry Course.

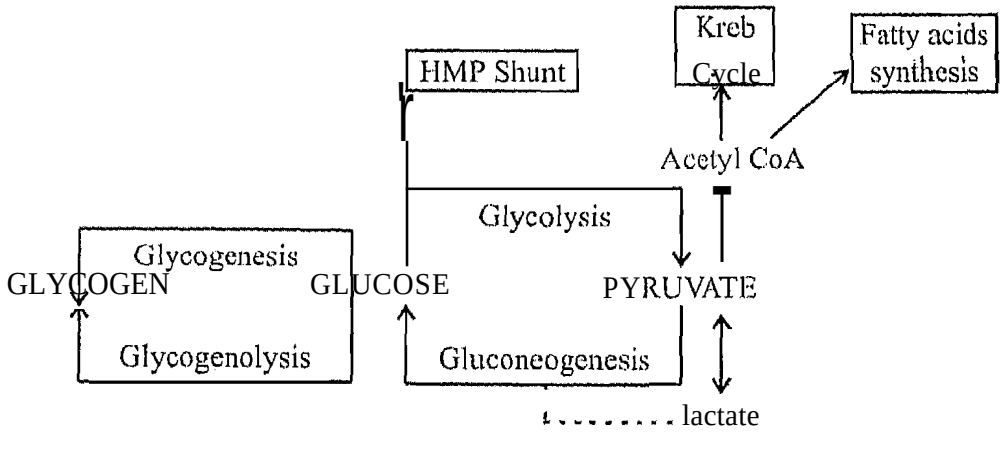

Figure 3.3: Integrated overview of metabolic pathways of carbohydrates

Next, let us get to know how glucose levels are regulated in the body.

3.6 REGULATION OF BLOOD GLUCOSE CONCENTRATION

A number of mechanisms function to maintain blood glucose at remarkably constant level of 70-100 mg/dl under fasting conditions. Regulation is the net effect of the organ's metabolic processes that remove glucose from the blood for either glycogen synthesis or for energy release and of processes that return glucose to the blood, such as glycogenolysis and gluconeogenesis.

Let us understand both of these mechanisms of blood glucose regulation.

After a meal, when blood glucose levels increase, the peptide hormones (such as cholecystokinin) secreted from enteroendocrine cells within the mucosa of the small bowel amplify the response of the β-cells of pancreas resulting in the secretion of insulin. Insulin facilitates the transport of glucose by glucose transporter - GLUT 4 into the adipocytes and muscle cells and stimulates glycogenesis (synthesis of glycogen) and fatty acid biosynthesis, thus returning the blood glucose to homeostatic level.

This was the case when there are high glucose levels in blood. But what happens, when the level of glucose falls? How, then the energy demands of cells are met? Well, in case of post absorptive state, the fall in blood glucose level signals the reversal of the pancreatic hormonal secretion i.e. decreased insulin and increased glucagon release. Blood glucose levels are maintained by the breakdown of glycogen and in this way, the glucose demands of brain, RBCs and testis are met.

In long periods of fasting' or starvation, glucose is supplied from non-carbohydrate sources by gluconeogenesis. Glucose is synthesized from a range of substrates including pyruvate, lactate, glycerol and amino acids. Body proteins are catabolized to release amino acids while triacylglycerol yields glycerol.

These gluconeogenic processes are triggered by a fall in blood glucose concentration below 5.0 mmol (90 mg/dl) and are signaled to the tissues by the secretion of glucagon and glucocorticoid hormones. Figure 3.4 illustrates the regulatory process.

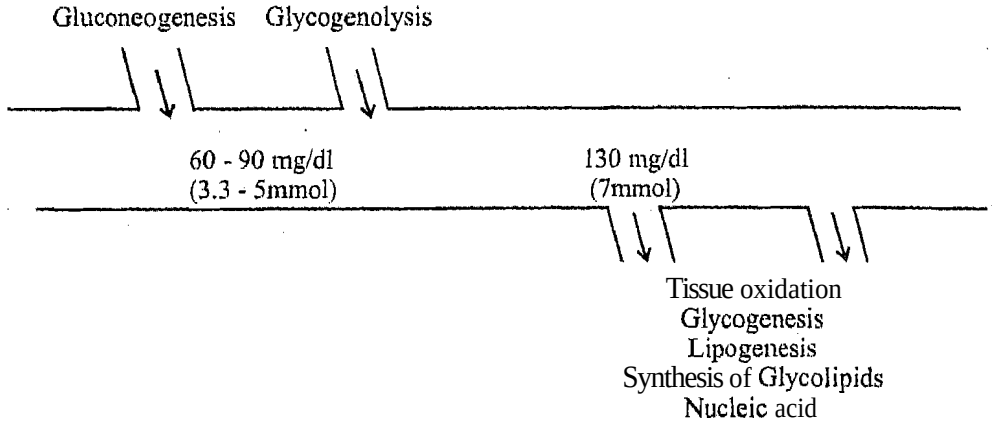

Figure 3.4: Regulation of blood glucose

With a brief review of the carbohydrate metabolism, let us now recapitulate what we have learnt so far by answering the check your progress exercise 1.

Check Your Progress Exercise 1

1) Fill in the blanks:

a) A group of organic molecules whose carbon compounds is extensively hydrated is ..... .

b) Carbohydrates can be classified on the basis of degree of ....and ..... fate.

c) The hydrolytic enzymes involved in digestion of carbohydrates are ....and ..... .

d) The most common manifestation of carbohydrate malabsorption is ....

e) The integral proteins which penetrate and span the lipid bilayer of plasma membrane are called as glucose ..... .

2) List some important functions of carbohydrates.

.....

.....

.....

.....

.....

3) Explain the mechanism of absorption of monosaccharide from the gastrointestinal tract.

.....

.....

.....

.....

.....

4) Explain the following:

a) Starvation or uncontrolled diabetes can lead to ketosis.

.....

.....

.....

.....

b) ORS always contains sodium chloride and glucose/sugar.

.....

.....

.....

.....

After studying about the carbohydrates which are digested and absorbed (i.e. glycemic carbohydrates), we will now discuss the fate and importance of those carbohydrates which are not digested in the gut. The next section will focus on dietary fibre, resistant starch (RS) and fructooligosaccharides.

3.7 DIETARY FIBRE

You are all aware that fibre is an important component in the structure of plants. However, fibre as a dietaiy constituent was considered important in the early 1970's when *Burkitt* and *Trowell* proposed that many western diseases were due to a lack of fibre in the diet. These included metabolic diseases such as diabetes, cardiovascular diseases, as well as, the diseases which were a result of straining at stool such as diverticular disease, hiatus hernia and haemorrhoids. Protective effects of fibre against colon cancer were also suggested by *Burkitt.*

Since then, extensive research has implicated dietary fibre as important in various aspects of gastrointestinal function and in prevention and management of a variety of disease states. The varying effects of dietary fibre as observed by researchers are obvious because it is made up of different components, each with its own distinctive characteristics. Methodologies have been developed to isolate these components, however, the definitions and methods of measuring fibre have changed over time. Let us see how definition of fibre has been modified over time.

Originally, *Burkitt* and *Trowell* defined fibre as *'the components of plant cell walls that are iitdigestible in the human small intestine'*. Later the definition was expanded to include storage polysaccharides within plant cells also.

Recently, the *American Association of Cereal Chemists (AACC)* developed an updated definition of dietary fibre to ensure tliat the term enconipassed the complete characterization of the components, as well as, their function. The AACC along with the *Carbohydrate Technical Committee of the North American branch of International Life Sciences Institute* developed the definition as:

"Dietary fibre is the edible part of plants or analogous carbohydrates that are resistant to digestion and absorption in the huinan small intestine with complete or partial fermentation in the large intestine. Dietary fibre includes polysaccharides, oligosaccharides, lignin and associated plant substances. Dietary fibre promotes beneficial physiological effects including laxation and/or blood cholesterol attenuation and / or blood glucose attenuation."

Now that we know the definition of fibre, let us learn about components of dietary fibre.

3.7.1 Components of Dietary Fibre

Dietary fibre (DF) includes many components which can be categorized on the basis of solubility or 'their location in the plant. Let us find out what are these.

Components classified on the basis of solubility are:

Insoluble DF	Soluble DF
Cellulose, Some hemi-celluloses, and Lignin.	Pectin, Gums, Mucilages, **and** Some hemi-celluloses.

Based on the *location on the plant,* DF components can be categorized as *plant cell wall constituents* and *non-plant cell wall constituents.*

Plant cell wall constituents include:	Non-Plant cell wall constituents include:
Lignin Cellulose, Hemicelluloses, and Pectins	Gums, Mucilages, Algal polysaccharides, Suberin, and Cutin

Table 3.2 presents the structure, properties, functions and food sources of different components. Kindly study it carefully.

Table **3.2: Important** components of dietary **fibre**

Component	Structure	Properties	Foods **High** in Content
Cellulose	Long, linear polymer of 1, 4 β-linked glucose. Hydrogen bonding between sugar residues in adjacent parallel running cellulose chain imparts the microfibril a three-dimensional structure.	Water-insoluble but can he modified chemically (sodium carboxy methyl cellulose) to be more soluble. Poorly fermented by colonic bacteria.	Bran, legumes, peas, vegetables of cabbage family, outer covering of seeds, apples.
Hemicellulose	Consists of heterogenous group of polysaccharide substances containing number of sugars in its backbone and side chains. Sugars which form backbone include xylose, mannose, galactose. Sugars present in side chain are arabinose, glucuronic acid and galactose. Hemicelluloses are categorized on the basis of predominant sugar in their backbone e.g. xylan, mannan, galactan.	Hemicelluloses that contain acids in their side chains are slightly charged and water soluble. Others are insoluble. Fermentability by intestinal flora is influenced by the sugars and positions e.g. hexose and uronic acids are more accessible to bacterial enzymes.	Bran and whole grains.
Pectin	They are polymers of D-galacturonic acid with a-1,4 glucosidic bonds. Rhamnose is also part of this backbone. Side chain consists of galactose, glucose, rhamnose, arabinose. Galacturonic acid monomers of backbone can also be in methyl ester forms.	They are water-soluble and gel forming. They have ion-binding potential. They are completely metabolized by colonic bacteria.	Apples, guavas, strawberries, citrus fruits.
Lignin	Main non-carbohydrate component of fibre. It is a three-dimensional polymer composed of phenol units - trans-coniferyl, trans-sinapyl and trans-p-coumaryl.	It is highly insoluble in water and responsible for the structural adhesion of plant cell wall components. It has hydrophobic binding capacity. It is not fermented by colonic microflora.	Mature root vegetables such as carrots. Wheat and fruits with edible seeds such as strawberries.
β Glucan	It is a polymer of glucose with mixed glucosidic bonds of both the β (1 − 3) and β(1 − 4) types.	It is soluble and hydrate, well forming viscous solutions and are often referred to as food gums or mucilage.	Grains, especially barley and oats.
Gums	Gums are secreted at the site of plant injury. They are comprised of a variety of sugar and sugar derivatives, the important ones being galactose and glucuronic acids. Gums used as food additives: i) Guar gum: It is a linear non-ionic galacto mannan. ii) Gum arabic: Has β (1 − 3) galactose backbone with side chains of arabinose, rhamnose, glucuronic acid, methyl glucuronic acid. iii) Guin karaya: It is a cylindrical complex polysaccharide, partially acetylated and highly branched with galacturonorhamnose chains to which galactose and rhamnose are attached.	They are water-soluble. They are highly fermented by colonic bacteria. It is highly soluble and possess gelling characteristic.	Oat meal, barley and legumes.

These components are present in varying proportions in different plant foods and their content is dependent upon the part of the plant (leaf, root, stem, seed) and maturity. What are the properties of fibre? This is elaborated in the next section.

3.7.2 Properties of Fibre

The structural make up of fibre influences its properties which in turn affects the physiologic and metabolic roles. This is well-depicted in the Figure 3.5 given herewith.

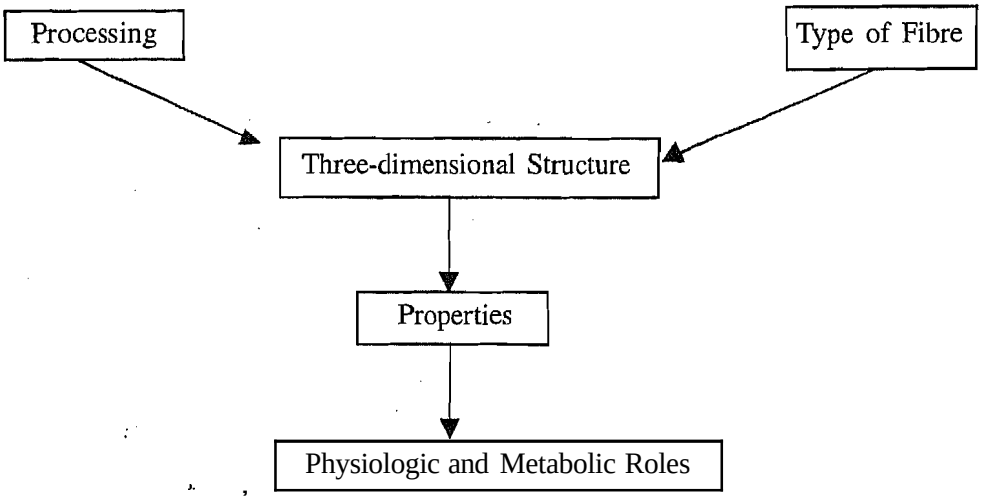

Figure 3.5: Relationship between structure and fibre properties

The polymeric backbones and / or side chain units determines the fibre's 2-dimensional structure, which influences the 3-dimensional structure i.e. how the polymer interacts with itself and other polymers e.g. because of their two-dimensional linearity, cellulose molecules interact with theinselves via hydrogen bonding to form crystalline regions.

Besides the type of fibre, the type and degree of processing will influence its structure and hence its profiles.

Significant properties / characteristics of dietary fibre that affects its role are:

1) Solubility in water
2) Hydrationlwater holding capacity and viscosity
3) Adsorptive attraction (ability to bind organic and inorganic molecules)
4) Degradability or fermentability by intestinal microflora.

Some of these properties you may have studied in the Principles of Food science Course (MFN-008) in Unit 2. Let us briefly review these properties.

1) *Solubility* in Water: Fibres that dissolve in hot water are soluble and those that do not, are insoluble. Several structural features affect solubility. We shall not go into the details of these features since it is not within the purview of this course, we will however, study the functions of soluble and insoluble DF.

   Generally soluble fibre:

   - delays gastric emptying;
   - increases the transit time (slower movement) through the intestine; and decreases nutrient absorption (glucose).

   Insoluble fibre:

   - decreases (speed up) intestinal transit time, and
   - increases faecal bulk.

We will now move on to the next characteristic of DF i.e. water holding capacity and viscosity.

Water Holding /Hydration Capacity (WHC) and Viscosity: WHC refers to the 'ability of fibre to bind water', just as dry sponge does when soaked in water. WHC of the fibre is influenced by a variety of factors. These are listed as:

solubility

- pH of the gastrointestinal tract,
- size of the fibre particles.

Many water-soluble fibres such as pectin, gums and some hemicelluloses have a high WHC. Further, pectin, gums and mucilage form viscous,solutions with the gastrointestinal tract. Cellulose and lignin have a low WHC.

These fibres:

- delay emptying of food from stomach;
- reduce mixing of gastrointestinal contents with digestive enzymes;
- reduce enzyme function;
- decrease nutrient diffusion rate and delayed nutrient absorption; and
- alter small intestine transit time

3) *Adsorption or Binding ability:* Some fibre coinponents have the ability to bind (adsorb) substances in the gastrointestinal tract. Wheat bran, guar gum, mannan and isolated lignin have been shown to bind bile acids in small intestinal contents. In humans, pectin, guar gum, oat bran and wheat bran have been shown to increase faecal bile acid excretion. Among the fibre components, pectin and lignin seem to have the greatest ability to adsorb bile acids.

Mechanisms suggested for bile acid adsorption are:

- hydrophobic interactions between lignin and bile acids,

  hydrogen binding between bile acids and pectins, and
- fibre (phenolic and uronic residues) may sequester or even chemically bind bile acids particularly when pH in lumen is low.

Physiological effects of ingestion of fibres with adsorption properties are:

- diminished absorption of lipids. Within the small intestine, bile acids and phospholipids are required for micelle formation and subsequent digestion and absorption. Hence, interaction between bile acid and fibre reduces lipid absorption.
- increased faecal excretion of bile acids absorbed to fibre cannot be reabsorbed and recirculated.
- hypocholesterolemic properties, and
- altered mineral balance.

4) *Fermentability or. Degradability:* As you are aware that colon contains over 400 known species of bacteria that exist in a symbiotic relationship with the host. All fibres are broken down to some extent by these microorganisms. Fermentation depends on the accessibility of the molecules to the microorganisms, which in turn depends on physical properties particularly solubility. Soluble fractions especially pectin, gums, mucilages and algal polysaccharides are very accessible and ferment rapidly. Insoluble fibre fraction ferment much more slowly.

The first step in fermentation is the breakdown of polysaccharides, oligosaccharides and disaccharides to their monosaccharide subunits by hydrolytic enzymes of bacteria. Monosaccharide are further converted to various end products as seen in Figure 3.6.

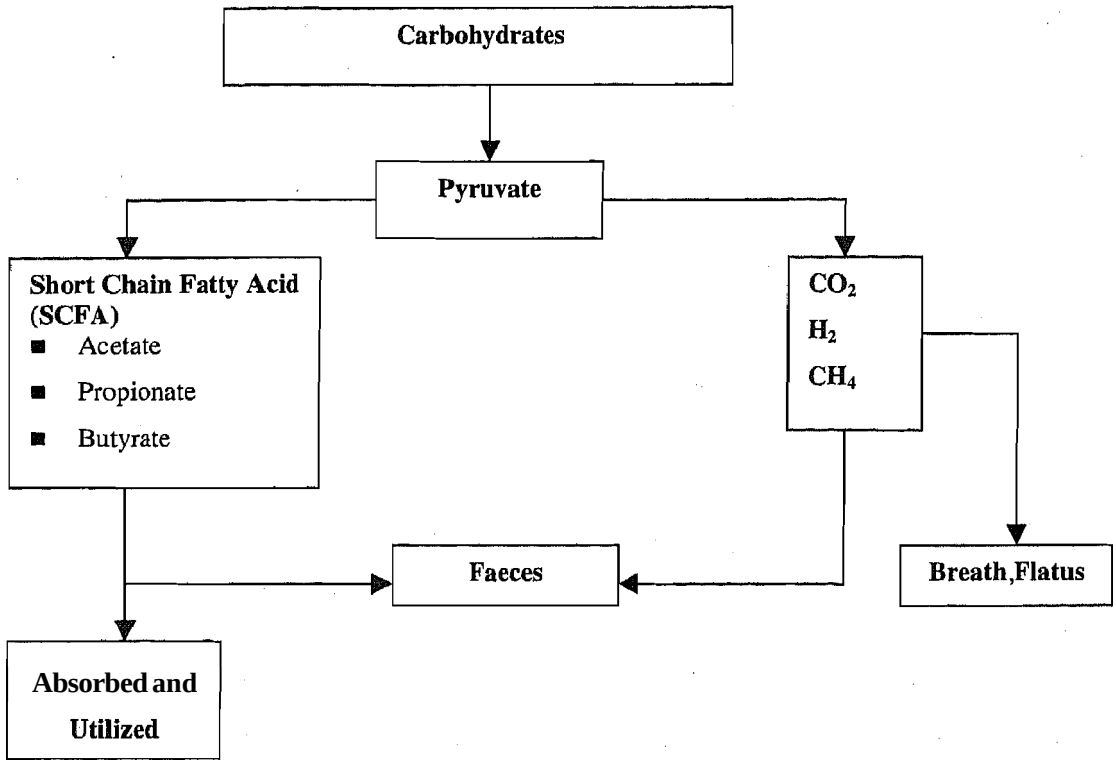

Figure 3.6:' Overview of carbohydrate fermentation on colon.

According to the calculations by Cummings and *Macfarlane*, if approximately 20 g of fibre is fermented in the colon each day, 200 mM SCFA will be produced, of which 62% will be acetate, 25% propionate and 16% butyrate. Of these, butyrate is almost completely consumed by the colonic mucosa, while acetate and propionate enter the portal circulation. The mechanism by which SCFAs cross the colonic mucosa is thought to be a saturable process-passive diffusion of unionized acid into mucosal cells.

In addition to these acids, other products of fibre fermentation are hydrogen, carbon dioxide and methane gases (refer to Figure ***3.6)*** that are excreted as flatus or expired by the lungs.

We will see in the next section that fermentability of dietary fibre is an important property linked to physiological effects.

3.7.3 Effects of Dietary Fibre

After reviewing the properties of fibre, we will now study various physiological effects of fibre, as it passes through the gut. The effect of dietary fibre in the intestinal tract depends on the type of fibre ingested (physicochemical / physical properties), the physical state of subjects and their previous diet, as well as, other components of diet.

Dietary fibre has major effects on:

a) Satiety: Several investigators have speculated that ingestion of a high fibre food induces a feeling of satiety, reduces meal size and food intake. Fibres forming viscous gels slow the rate of gastric emptying and create a feeling of postprandial satiety.

b) Nutrient Absorption: Inclusion of fibre has been shown to retard the absorption of some nutrients. The inclusion of viscous polysaccharides reduces the postprandial glucose level concentration. Guar gum and pectin have been shown

to be beneficial in controlling hyperglycemia. Increasing the viscosity of gastrointestinal contents delay absorption in a number of ways. These include:

- inadequate mixing of luminal contents due to increased viscosity may slow the movement of digestive enzymes to their substrate thus delaying digestion; and
- viscous properties inhibit the access of nutrients to the epithelium. Nutrients have to diffuse across the thin, relatively unstirred layer of fluid lying adjacent to epithelium. This is achieved by intestinal contractions, which create convection currents, thus bringing the material from the centre of the lumen close to the epithelium. Increasing the viscosity of the lurninal content impairs convective effects and thus delays absorption.

The absorption of nutrients is also reduced by mechanisms other than increasing the viscosity of gastrointestinal content. These mechanisms are:

- Soluble fibres (pectin, guar gum, oat bran), as well as, insoluble fibrelignin, may affect lipid absorption by their ability to adsorb fatty acids and cholesterol, thereby inhibiting their incorporation in miscelle. Further adsorption of bile acids to these fibres reduces the availability of bile acids for micelle formation.
- In case of unrefined whole plant material, the nutrients are sequestered within.the cellular matrix and hence unavailable for absorption. Grinding of food material and thorough chewing can influence absorption.
- The altered mineral availability associated with diets high in fibre appear to be due to binding of metal ions. Fibres which possess uronic acid (pectins, hernicellulose gum) can form cationic bridges with minerals. Lignin, with both carboxyl and hydroxyl groups, can adsorb minerals. Certain constituents of plants e.g. phytates, silicates and oxalates also chelate divalent cations. However, the overall effect that a fibre will have on mineral balance will depend on the composition of diet and degree of fermentability of fibre. Rapidly fermentable fibres release the bound minerals as fermentation occurs and these minerals may possibly be absorbed in the colon.

SCFAs (short chain fatty acids) have a stimulatory effect on sodium absorption from colonic lumen. The unionized SCFA crosses the epithelial cell where it disassociates. The hydrogen ion is then moved back into the lumen in exchange for sodium. Thus, SCFAs provide a powerful stimulant to sodium and water absorption.

c) ***Integrity of gut/colon***: Dietary fibre especially fermentable fibres, play an important role in maintaining the integrity of gut. SCFAs generated during fermentation stimulate the proliferation of mucosal cells in the gut and thus maintains its integrity.

Butyrate serves as a preferred energy source for colonic cells and regulates colonic cell proliferation.

Also, SCFAs acidify the colon thus reducing the solubility of bile acids. Further, the activity of 7 α-*dehydroxylase* diminishes which decreases the conversion of primary bile acids to secondary bile acids. These changes may protect against colon cancer.

d) ***Stool weight and laxation:*** The amount of stool excreted varies markedly from individual to individual and in an individual over a period of time.

Faeces are complex and consist of water, unfermented fibre, excreted compounds and bacterial mass. Of the dietary constituents, dietary fibre has been shown to influence the stool weight to a great extent,

The ability of different types of fibres to increase faecal bulk depends on a complex relationship between the chemical and physical properties of the fibre and the bacterial population in the colon. In general, faecal bulk increases as fibre fermentability decreases.

The mechanism by which a fibre increases stool weight is through the water-holding capacity of unfermented fibre. Animal and human studies have indicated that cereal fibres have the greatest faecal bulking power. Wheat bran added to the diet increases stool weight in a predictable linear manner and decreases intestinal transit time. Besides quantity, the particle size is also important. Coarsely ground wheat bran has little or no effect and may even be constipating.

Fibre may influence faecal output by another mechanism. Colonic microbial growth may be stimulated by ingestion of fermentable fibre sources. Bacteria are an important component of faecal mass. However, increase in weight does not always occur from eating these fibres. Some laxative effects may be due to volatile SCFAs produced during fermentation. Osmotic effects of these fermentation products may also be important but this mechanism is not yet well defined.

e) *Other effects:* In addition to all the physiological effects mentioned above, dietary fibre may exert other effects. Acetate and propionate enter the portal circulation, thus sxtending the effects of dietary fibre beyond the intestinal tract. Initial in-vitro experiments have indicated that cholesterol synthesis by isolated hepatocytes is inhibited by propionic acid. However, a wide variety of data from human and animal studies do not consistently support this finding. Thus, whether inhibition of cholesterol synthesis by propionate occur in-vivo at physiological concentration is not clear.

After knowing the effects of dietary fibre, we will now very briefly review some of the potential health benefits of fibre.

3.7.4 Potential Health Benefits of Dietary Fibre

A number of experimental studies in animal models, as well as, epidemiological studies have established protective role of dietary fibre against chronic degenerative diseases. Its relation with colon cancer and cardiovascular diseases will be discussed in this section.

Dietary fibre and colon cancer

The relationship between colorectal cancer and dietary fibre remains complex. Although a cause-and-effect relationship between fibre and colon cancer has not been established, the majority of epidemiological studies (descriptive, case-conk01 and cohort) support an inverse relationship between consumption of vegetables and fruits and colorectal cancer risk. Fruits, vegetables and grains, in addition to fibre, also contain **a** variety of anticarcinogenic compounds, which may contribute to this protective effect. Epidemiological evidence that whole grains protect against colorectal cancer is also strong. However, evidence from prospective, large epidemiological studies for protective effect of dietary fibre on colorectal cancer is not strong.

Several plausable mechanisms have been formulated by which fibre may provide protection against colon cancer. These include:

- Fibre that increases stool bulk results in the dilution of carcinogens. Fibre also decreases transit time thereby reducing the interactions of carcinogens with colonic mucosal cells.
- Fibre binds potential carcinogens.
- High bile acid concentrations are associated with increased risk of colon cancer. Fibres adsorb bile acids, thereby reducing the risk.
- Fibre by providing fermentable substrate to colonic rnicroflora, alters species and number of microorganisms which may inhibit proliferation of tumor cells or conversion of procarcinogen to carcinogens.
- Fermentation to SCFAs, reduces the pH, which in turn reduces synthesis of

secondary bile acids. These bile acids have been shown to promote tumors.

- Butyrate has been shown to have an effect on chromatin structure and thus could slow the proliferation and differentiation of cancer cells. Butyrate induces apoptosis (disintegration of cells).
- Lignin may act as a free radical scavenger, thus reducing the risk of cancer.

Further, fiber has been shown to lower serum oestrogen concentrations, and therefore may have a protective effect against hormone-related cancers. Recent studies have shown a decreased risk of endometrial cancer, ovarian cancer, and prostate cancer with high fiber intakes. More research is needed before conclusions can be drawn on these relationships.

From our discussion above it is evident that dietary fibre by different mechanisms mentioned above can play a role in reducing the risk of cancer. Let us now get to know the relationship between fibre and cardiovascular disease.

Fibre and Cardiovascular Disease (CVD)

The role of dietary fibre in modulation of blood lipids was demonstrated by Keys and his co-workers in a series of experiments conducted during 1960's. Later *Trowell* supported the protective effect of dietary fibre against hyperlipidemia and ischemic heart disease.

An inverse relationship between CVD and dietary fibre has been shown in many prospective and epidemiological studies and cross-sectional population survey. However, uniform results have not been yielded across studies.

Studies in which diets were modified to reduce fat and increase carbohydrate and fibre level have shown favourable impact on the incidence and regression of CVD.

Evidence supports a protective effect of dietary fiber for CHD, particularly viscous fibers that occur naturally in foods, which reduce total cholesterol and LDL cholesterol concentrations. Reduced rates of CHD were observed in individuals consuming high fiber diets. These studies used fiber-containing foods; fiber supplements may not have the same effects. The type of fiber is important, oat bran (viscous fiber) significantly reduces total cholesterol, but wheat bran (primarily non-viscous fiber) may not. Viscous fibers are thought to lower serum cholesterol concentrations by interfering with absorption and recirculation of bile acids and cholesterol in the intestine and thus decreasing the concentration of circulating cholesterol. These fibers may also work by delaying absorption of fat and carbohydrate, which could result in increased insulin sensitivity and lower tiiacylglycerol concentrations, Dietary fiber intake has also been shown to be negatively associated with hypertension in men but not women. Fiber intake was shown to have an inverse relationship with systolic and diastolic pressures.

Thus, it is important to note that with respect to CVD, only soluble fibres which are also viscous have been shown to reduce serum cholesterol. This effect is not simple but could be due to multiple factors operating simultaneously. Possibly dietary fibre displaces fat from the diet. Also polyunsaturated fatty acids consumed in conjuction with fibre play a role.

Some fibres reduce the reabsorption of bile acids in the ileum, thus affecting the enterohepatic circulation. Enterohepatic pool is renewed by increased synthesis of bile acids from cholesterol, which in-turn reduces body cholesterol.

Fibres such as oat bran and pectin may decrease absorption of dietary cholesterol by altering the composition of bile acid pool. Since exogenous cholesterol represents only a small proportion of the body's cholesterol, this mechanism may contribute partially to the fibre-induced hypocholestermia.

Data is available from some animal studies, which indicate that endogenous cholesterol synthesis is affected by feeding dietary fibre. HMG *CoA reductase*, the rate limiting enzyme in cholesterol biosynthetic pathway is inhibited by deoxy cholic acid (DCA) as compared to cholic acid or chenodeoxycholic acid. Administration of certain fibres increases the proportion of DCA in bile acid pool. The importance of this mechanism needs to be studied in humans.

All these factors may contribute to the hypocholesterolernic effect of fibre, but the relative importance of each is not well known. Further, many natural plant constituents have been shown to affect lipid metabolism. These components are frequently present in dietary fibre sources and may confound effects of dietary fibre.

A diet that prevents CVD or slows its progression is the one which is low in fat and high in complex carbohydrates. Such diets, which are minimally processed, are high in dietary fibre and may contain other hypocholestermic components like phytoesterogens.

So, we have seen the benefits of fibres, as well as, its role in preventing the disease like cancer and CVD. But is there a minimum amount of daily fibre intake or we can consume as much as we like? Let's find out in the next section, what is the desirable level of fibre intake as recommended by the Nutritional Institutes/ Associations.

3.7.5 Recommended Intake of Fibre

A minimum of fibre intake of 20 g/day is recommended by the American Dietetic Association (ADA), the National Cancer Institute, US and the Federation of American Societies for Experimental Biology (FASEB).

Alternately, 10-13 g dietary fibre intake per 1000 Kcal also has been suggested by ADA. FASEB, the National Cancer Institute and ADA suggest an upper limit of 35 g/day.

Dietary fibre is now a niandatory component of US food labels.

Thus, dietary fibre is now .recognized as important component of diet and plays an important role in gastrointestinal physiology and has a number of potential health benefits.

Answer the questions given in check your progress exercise 2 and recapitulate what you have learnt so far.

Check Your Progress Exercise 2

1) What is a Dietary Fibre? What are its components?

.....

.....

.....

.....

.....

2) What are the factors affecting WHC of a fibre?

.....

.....

.....

.....

3) Explain the physiological effects of ingestion of fibres with respect to:

a) WHC

.....

.....

.....

b) Absorption property

.....

.....

.....

4) Discuss the effect of dietary fibre on the absorption of nutrients.

.....

.....

.....

.....

.....

5) List the mechanisms by which fibres provide protection against colon cancer.

.....

.....

.....

.....

.....

Next, we shall learn about resistant starch. You may recall studying about this earlier in the course Principles of Food Science (MFN-008) in Unit 2. We suggest you look up this unit in the MFN-008 course now.

3.8 RESISTANT STARCH

Until 1980's starch was thought to be completely hydrolyzed and absorbed from the small intestine of man, independent of its source, type and preparation. However, about two decades ago, it became apparent that appreciable amounts of starch are not digested and enter the colon. The term 'Resistant Starch' (RS) was coined for this fraction. Recently, RS has been an active area of research because of its potential health benefits especially its effect on the large bowel functions. Therefore, in this section we will talk about what RS is, how RS content of food is' influenced and its beneficial effects.

What is RS?

Well, *RS is defined as the starch, which escapes enzymatic hydrolysis in the small intestine and passes to the colon where it is fermented by colonic microflora which results in the formation of short chain fatty acids (SCFAs).*

From the physiological perspective:

RS is defined as the sum of starch and products of starch degradation not absorbed in the small intestine of healthy individuals,

Thus, starches can be subdivided into *'digestible'* and *'indigestible'* starch. The former may be hydrolyzed at a particular rate but nevertheless has an ideal digestibility of ~ 100%. On the other hand, indigestible starches (i.e. RS) have an ideal digestibility of less than 100%.

RS can be further sub-classified into 4 classes. The four types of RS and the foods in which they are present are given in Table 3.3.

Table 3.3: Classification of resistant starch

Type	What it Constitutes	Examples of Occurrence
RS1	Comprises of physically inaccessible starches	Partially milled grains and seeds
RS2	Consists of starch in certain granular form, which is inherently resistant to enzymatic digestion	Raw potatoes, green bananas, some pulses
RS3	Includes retrograded starch formed by cooking and then cooling starchy food	Cooked and cooled potato, rice and other high starch products
RS4	Chemically modified starches used in food industry for their technological attributes. Modifications include esterification and etherisation.	Modified starches

In the subsequent sub-sections we shall look at the factors influencing RS content of foods and the potential benefits of RS. We begin by understanding the factors influencing the RS content of foods.

3.8.1 Factors Influencing RS Content of Foods

Different fractions of RS mentioned in Table 3.3 may be naturally present in some foods or may be generated as a result of industrial or home level processing. The amount of RS produced is influenced by several factors.

As we will see in this section that inherent properties of food, ingredients added during processing, as well as, different processing techniques influence the RS content of the food. Some of these factors are discussed here in brief.

i) *Amylose/Amylopectin ratio: In* general, foods containing high amylose/amylopectin ratio lead to higher yield of RS. You may recall studying about the amylose and amylopectin in the Principles of Food Science Course, in Unit 2. Arnylose retrogrades very rapidly and results in a material highly resistant to arnylolysis. In contrast, amylopectin undergoes retrogradation more slowly and is almost completely degraded by amylase.

ii) *Water content:* The yield of RS formed in heat-moisture treatment is closely related to water content, which may be an inherent component of food or added during cooking. Generally, optimum water content leads to proper and complete gelatinization followed by retrogradation, thus contributing to the RS.

iii) *Presence of sugar and lipids:* Studies so far show that presence or addition of lipids and sugar reduces RS content.

iv) *Calcium and potassium ions:* Investigations have indicated that the yield of RS in starch gels decreases in the presence of calcium and potassium ions. These ions are adsorbed on starch and prevent the formation of hydrogen bonds between arnylose and amylopectin .chains.

v) *Polyphenols and phytic acid* Polyphenols (catechin, tannic acid) and phytic acid may affect starch digestibility through their interaction with amylose activity and contribute to RS formation.

vi) *Antinutritional factors:* Presence of certain anti-nutrients, such as α-amylase inhibitors, greatly determines the extent of starch hydrolysis and hence the RS content.

vii) *Processing techniques:* Freezing, storage, heating-cooling cycle, baking, flaking, popping increase the RS content while reheating of stored food, germination, fermentation results in reduction.

Thus, you have seen that a number of factors influence the RS content. It is important to note that at any given time, more than one factor may operate simultaneously thereby influencing the RS in a complex manner.

In addition to what is termed as 'chemical' RS i.e. the value obtained by standard analytical procedures, certain factors which may vary from one individual to another such as extent of chewing, mastication and intestinal transit time may also influence the digestibility. Therefore, RS determined analytically (in-vitro) need not represent the total RS, occurring 'in-vivo'. The latter quantity is termed as 'physiological RS'. Therefore there is a need to develop analytical procedures that take into account the physiological influences on starch digestibility.

Next, let us learn about the potential health benefits of RS.

3.8.2 Potential Health Benefits

Like dietary fibre, RS can also play a potential role in helping to maintain or improve health of an individual. As you have already seen that dietary fibres exert their effects through their physical presence, as well as, through their metabolism by microflora. But in case of RS majority of effects are related to fermentation by colonic bacteria. In fact, unlike some fibres (bran) very little starch appears in the faeces under normal circumstances.

Thus, like soluble fibre, RS is fermented in the colon producing SCFAs (acelate, butyrate, propionate) gases and bacterial biomass. However, fermentation of RS produces higher amounts of butyric acid compared to soluble dietary fibre.

The physiological effects of SCFA's can be summarized as follows. They:

- lower the pH in the gastrointestinal tract, which in turn reduces the colonization of pathogenic bacteria (acid-sensitive). Lowering of pH also increases ionization of toxic compound, thereby reducing their absorption,
- stimulate colonic blood flow and motor activity,
- enhance water and electrolyte absorption,
- stimulate colonocyte proliferation,
- butyrate is a preferred substrate for colonocyte and contributes to energy needs of the colonic epithelial cell. Acetate and to a lesser extent propionate are absorbed into the system and contribute to energy needs of the host.
- butyrate enhances DNA stabilization and repair, induces apoptosis in potential cancer cells and thus promote a normal cell phenotype,

All these effects have a positive influence on intestinal function and health and subsequently whole body.

The sum of evidence suggests that RS has beneficial effects on large bowel physiology. Numerous studies in animals have shown that feeding of RS improved indices of bowel health as evident by low pH, high SCFA concentration. Many of these studies

were invasive and not practical for humans. Therefore, human studies have relied on indirect measures such as greater hydrogen breath or changes in faecal variables. In humans direct benefit has been observed in cholera patients. Addition of RS to oral rehydration solution (ORS) reduced faecal fluid loss and shortened the duration of diarrhoea. These benefits probably stem from the increased absorption of sodium and water.

It has been proposed that RS may have protective effect against colon cancer. The anticarcinogenic effect of RS may be attributed to the following:

- protective effects of butyrate,
- reduced concentrations of total and secondary bile acids. Lowering of pH by SCFAs reduces activity of 7 a dehydroxylase,
- reduced bacterial β-glucosidase activity. β-glucosidases are related to the risk of colon cancer because they release potential chemical carcinogens. A high RS diet has been shown to reduce bacterial β-glucosidase activity, and
- reduced concentration of ammonia. Ammonia has a range of toxic effects. It may damage colonic epithelium, enhance cell proliferation, favour growth of malignant cells in preference to normal cells. The higher amount of carbohydrate available for bacterial fermentation may reduce protein fermentation and in turn ammonia production.

Many of the above observations are made in animals and their relevance to humans needs to be explored.

It has been shown that generally foods high in RS yield low glycemic index values in humans. Also postprandial plasma glucose values have been shown to decrease proportionately with increasing RS content. Thus, a higher RS content could be important in the management of diabetes.

Experimental studies in animals and a few human studies indicate that RS may play a regulatory role in post prandial triglyceridemia and in a limited number of cases, in plasma cholesterol concentration. However, more work is warranted in this aspect.

Thus, in view of potential health benefits, inclusion of RS in the diet would be beneficial. Ingestion of high starch foods should lead to a substantial appearance of the RS in the large intestine. The amount of RS can also be increased during processing by modifying the processing parameters without affecting the sensory qualities of food. Alternately, various methods are now available to produce starches high in RS, with desirable properties as food ingredient. Commercial RS has a better appearance and is devoid of certain drawbacks of commercial fibres such as high water holding capacity, viscosity, gritty mouthfeel and characteristic fibre taste.

From our discussion above surely you may have got a deep insight into the functions and the role of RS in our diet. Another non-digestible carbohydrate about which we need to know are the fructo oligosaccharides. What are fructo oligosaccharides? What are its sources, role and properties? Let us read and find it out.

3.9 FRUCTO OLIGOSACCHARIDES (FOS)

FOS are polymers of fructose, usually attached to an initial glucose molecule. The total number of fructose units range from 2 to 8 and are linked by (2 → 1) linkages. They are naturally present in certain foods like onion, banana, leak, wheat, garlic, chicory root and artichoke. FOS can also be obtained commercially by an enzymatic action on sucrose.

Like other non-digestible oligosaccharides, they resist hydrolysis by human digestive enzymes. They enter the cecum without significant changes, where they are fermented by the resident microflora into short chain fatty acids, lactic acid, carbon dioxide and

hydrogen. They enhance the growth of intestinal flora especially growth of bifidobacteria. Thus, like dietary fibre and resistant starch they exert many health benefits.

FOS are low-energy bulk ingredients that have a taste profile similar to that of sucrose. Also their physical and chemical properties match precisely those of sucrose. Thus, they can replace sucrose and have been successfully used in food industry, especially bakery industry.

We will get to know more about FOS later in Unit 11 on 'functional foods'

While talking about carbohydrates, you may have realized that some carbohydrates are rapidly digested and absorbed, some are digested slowly while some are not digested at all. Different carbohydrates have different glycemic index. What is glycemic index? What is its significance? We will get a detailed insight into this aspect in the Clinical and Therapeutic Nutrition Course (MFN-005), however, a brief review on this aspect is presented next.

3.10 GLYCEMIC INDEX (GI)

In the previous sections, we have studied that some carbohydrates are rapidly digested and absorbed, some are digested slowly while some are not digested at all. Thus, it is obvious that different carbohydrates will raise blood glucose levels to a different extent. *The ability of carbohydrates to raise blood glucose is ,referred to as "Glycemic Index" (GI).*

GI is a quantitative assessment of foods based on post prandial blood glucose response (i.e. blood glucose level after a meal) expressed as a *'percentage of the response to an equivalent carbohydrate portion of a reference food'*. The reference food is white bread with a GI set at 100. Glycemic indices of some foods are given in Table 3.4. Here you can see 2 columns indicating GI. In the first one, the reference food is white bread (Wb), while the second column has glucose (g) as its reference food with a GI of 100.

Table 3.4: Glycemic indices of some foods

Food	G/ wb	G/ g
Sucrose	92	67
Glucose	138	100
Fructose	32	23
Honey	104	75
Milk	39	28
Beans	40 - 60	30 - 43
Lentils	30 - 40	22 - 30
Pasta	50 - 70	36 - 51
Potatoes	120	87
Banana (ripe)	85	62
Banana (under-ripe)	43	31
Oranges	62	45
Grape fruit	36	26
Tomatoes	13	9

G/ wb - Standard food White bread

G/ g - Standard food: Glucose $G/g = \frac{G/wb}{1.38}$

Source: Augustin *et* al **(2002).**

What is the relevance of knowing about GI of foods? Let us see how this index is helpful in prescribing therapeutic diets. You would realize that the dietary GI provides an indication of the rate at which, carbohydrate foods are digested. High and low GI diets may be a better measure for assessing the physiological effects of dietary carbohydrates than the traditional 'simple' and 'complex' carbohydrate definition. It allows ranking of foods from those which give rise to the highest blood glucose and insulin responses (high glycernic food) to those associated with the lowest blood glucose and insulin responses (low GI foods).

This index integrates multiple influences on glucose availability and is proposed as a means for prescribing diabetic and energy controlled diets. We will learn the practical application of this concept in the Clinical and Therapeutic Nutrition Course in Unit 10 as mentioned above. Let us get to learn about the factors which influence GI of foods next.

3.10.1 Factors Affecting GI of Foods

A variety of factors affect GI of foods. The factors which affect the rate of glucose absorption from starchy foods and therefore the GI value are:

Nature ofstarch: High levels of amylase decreases GI while low levels increase the GI.

- *Nature of monosaccharide components:* High levels of fructose and galactose decrease the GI whereas high levels of glucose increase the GI.
- *Viscous Fibre:* Presence of guar gum and β-glucan reduce the GI.
- *Cooking/processing*: Parboiling, cold extrusion decrease the GI while flaking, popping increase it.
- *Particle size:* Consumption of large particles of starchy foods reduces the GI. On the contrary, grinding of starchy foods results in an increase in the GI.
- *Ripeness and food storage:* Cooling of starchy food before consumption decreases the GI. Similarly, unripe or under ripped fruits have a lower GI.
- *a-amylase inhibitors:* Presence of α-amylase inhibitors like lecithin, phytates lowers the GI.,
- *Nutrients-starch interactions:* High levels of protein and fat decreases the GI. Fat and protein appear to modify the glycemic response to a carbohydrate food by slowing gastric emptying and increasing insulin secretion, respectively. However, neither protein nor fat in amounts found in most foods significantly affect the GI. Protein levels of 30 g and fat levels of 50 g per 50 g of available carbohydrate may decrease the GI.

Now we shall understand the role of GI in chronic disease such as diabetes, coronary heart disease and cancer.

3.10.2 GI in Chronic Diseases

In addition to serve as an aid in planning diets for diabetics, GI of diets has been linked to a number of chronic diseases. Several health benefits appear to exist by reducing the rate of carbohydrate absorption by means of a low GI diet.

The health benefits include reduced insulin demand, improved blood glucose control and reduced blood lipid levels. All these factors play an important role in the prevention and management of chronic diseases including diabetes, coronary heart disease and certain cancers. Epidemiological evidence suggests a direct association between GI and risk of diabetes, coronary heart disease (CI-ID), obesity and certain cancers.

Let us first deal with diabetes, the most widespread disease. In some studies, a positive association between GI, Glycemic load (GL i.e. product of the average GI and total carbohydrate intake) and risk of type II diabetes has been observed. This could be because high GI foods lead to a rapid rise in blood glucose and insulin levels. Hyper insulinemia in turn may down regulate insulin receptors and therefore reduce insulin efficiency resulting in insulin resistance. On the contrary, low GI foods result in reduced peak insulin concentrations and reduced insulin demand thus decreasing the risk of type II diabetes.

Next, we move on to CHD.

As similar to studies conducted on diabetes certain epideiniological evidence suggests that low GI diet may reduce the risk of CHD independently. The possible positive effects of a low GI diet in the prevention of CHD may be due to improvements in blood lipid profile, insulin levels, thrombolitic factors and endothelial function.

Further, it has been suggested that generally low GI foods are associated with greater satiety as compared to high GI foods or meals. High GI foods result in fast carbohydrate absorption, large blood glucose and hormonal (insulin/glucagon) fluctuations and reduced satiety. All these factors may favour over eating and weight gain. Thus, low GI food appears to play a role in prevention and management of obesity, which is a risk factor for many diseases.

Available evidence thus suggest that low-fat, high-carbohydrate diets advocated by the heath organizations can be further improved by switching from a high GI to a low GI diet.

Finally, we shall end our discussion on carbohydrates with a brief insight into the kind of modifications recommended for carbohydrate intake for specific disorders.

3.11 MODIFICATION OF CARBOHYDRATE INTAKE FOR SPECIFIC DISORDER

In our daily diet almost 60-70% of energy is contributed by carbohydrates. Majority of them are comprised of starch (from cereals, millets, pulses and root vegetables), while a small amount is also contributed by sucrose (sugar), glucose and fructose (fruits), as well as, lactose (milk and milk products). However, there is a need to modify carbohydrate intakes in certain disorders such as:

1) *Lactose intolerance:* This has been covered under the section on digestion and absorption earlier. We learnt that in case of lactose intolerance, the ingestion of lactose leads to passage of the sugar to the large bowel, where it is fermented to produce SCFA and gases, which causes discomfort. Lactose is present in dairy products such as milk, cheese, yoghurt, ice cream etc. Hence, these foods need to be avoided.

2) *Diabetes mellitus:* You are aware that diabetes may be diagnosed as an exaggerated response in blood glucose concentration following ingestion of a fixed amount of glucose (glucose tolerance test). The most common forms of diabetes are *insulin-dependent diabetes mellitus (IDDM or type I diabetes) and non-insulin-dependent diabetes mellitus (NIDDM or type 2 diabetes).* IDDM results from the autoimmune destruction of the α-cells the endocrine pancreas, the consequence of which is insulin insufficiency. In this, the patient requires exogenous supply of insulin. The amount of carbohydrate and frequency of feeding is modified and depends on the insulin dose, type of insulin and the weight of the person.

In contrast, expression of NIDDM is due to lifestyles (excessive energy intakes and low physical activity) resulting in obesity, Early stages are characterized by insulin insensitivity/ resistance. Management of these patients involves maintenance of ideal body weight.

In both types of diabetes, the diet should be high in fibre, and in complex carbohydrates and low in simple sugars. Foods with low glycemic index should be encouraged. For detail regarding principles of planning diet for diabetes we suggest you look up the Unit/ Practical in the Clinical and Therapeutic Theory and Practical Courses (MFN-005, MFNL-005), respectively.

Now try to answer the questions given in the check your progress exercise 3 to ascertain your knowledge regarding the issues discussed so far.

Check Your Progress Exercise 3

1) What do you mean by the term 'resistant starch'? List a few foods in which they occur.

.....

.....

.....

.....

.....

2) Mention the factors affecting the RS content of food.

.....

.....

.....

.....

.....

3) What are the factors which contribute to the anti-carcinogenic effect of RS?

.....

.....

.....

.....

.....

4) Discuss the significance of GI of foods.

.....

.....

.....

.....

.....

.....

5) List the factors that affect GI value of foods.

.....

.....

.....

.....

.....

6) How do the foods with a high GI content favour the risk of obesity?

.....

.....

.....

.....

.....

7) Explain the following terms:

a) Glycemic Index

.....

.....

.....

b) FOS

.....

.....

.....

3.12 LET US SUM UP

In this unit, we studied about carbohydrates, its functions and types. We learnt that carbohydrates provide energy (4 cal/g). They are formed by plants in bewildering array of possible single unit and polymer structures.

Humans have the ability to digest only a few of the many possible bonds linking carbohydrate units with each other and with other types of organic molecules. About 80% of edible carbohydrates are absorbed as single glucose unit and metabolized. For optimum function of nervous system and cells, blood glucose concentrations are tightly controlled by hormones (insulin in the absorptive phase; glucagons, adrenaline and cortisol in the post absorptive phase) utilizing several possible metabolic pathways for glucose anabolism and catabolism.

Depending on the structure, non-digestible carbohydrates pass into the colon. They are fermented in varying degrees to short-chain fatty acids, carbon dioxide, hydrogen and methane in the large bowel, Absorbed short chain fatty acids are metabolized in colonic epithelial, hepatic and muscle cells. Thus, these perform a number of beneficial functions.

Intakes of optimum amounts of different types of carbohydrates are associated with good health through effects on energy balance, digestive function, blood glucose control and other risk factors for several chronic diseases.

3.13 GLOSSARY

Apoptosis : disintegration of cells into membrane-bound particles that are then eliminated by phagocytosis or by shedding.

Degree of polymerization : the length in monomeric or base units of the average linear polymer chain at any time in a polymerization reaction.

Diverticular disease : a condition in which small pouches called diverticulae, develop at the weak spots in the wall of the colon, that eventually bulge out to form pouches.

Glucose transporters : the integral proteins which penetrate and span the lipid bilayer of plasma membrane.

Glycemic index : ability of carbohydrates to raise blood glucose.

Glycemic load : a product of average glycemic index total carbohydrate intake.

Glycosidases : hydrolytic enzymes involved in the digestion of carbohydratres. Also termed as carbohydrases.

Haemorrhoids : also called as piles; these are the small troublesome tumors or swellings with a painful mass of dilate veins in anal tissue.

Hiatus hernia : the protrusion of part of the stomach through the diaphragm.

Homopolysaccharide : a polysaccharide composed of single monosaccharide unit.

Resistant starch : starch which escapes enzymatic hydrolysis in the small intestine and passes to the colon where it is fermented by colonic bacteria and forms SCFA.

Water holding capacity : the ability of fibre to bind water.

3.14 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress' Exercise 1

1) a) carbohydrates
   b) polymerization, digestive
   c) endo-glycosidase-α-amylase, oligosaccharidases
   d) lactose intolerance
   e) transporters

2) Carbohydrates are a major source of energy, have protein-sparing effect, have anti-ketogenic effect, help in the excretion of toxins, serve as precursors to compounds of nerve tissue and are beneficial for the functions and physiology of the GI tract.

3) Glucose and galactose are absorbed into the mucosal cells by active transport which requires energy. The carrier of glucose and galactose is a specific protein attach to the carrier until preloaded with sodium. Fructose is absorbed by a facilitated transport involving GLUT-5.

4) a) In case of starvation or uncontrolled diabetes, there is an absence of sufficient carbohydrates and hence, larger amounts of fat are used for energy. This results in incomplete oxidation and accumulation of ketone bodies.

b) ORS always contains sodium chloride and glucose /sugar because glucose is absorbed into the mucosal cells by active transport which requires energy. The carrier of glucose is a specific protein complex known as sodium-glucose transport protein-1 (SGLT-1) which is dependent on Na^+/K^+ ATPase pump. Glucose cannot attach to the carrier until it has been preloaded with sodium.

Check Your Progress Exercise 2

1) Dietary fibre is the edible part of plants or analogous carbohydrates that is resistant to digestion and absorption in the human small intestine with complete or partial fermentation in the large intestine. Its components include cellulose, hemicelluloses, lignin, pectin, gum, mucilages, algal polysaccharides, suberin, cutin.

2) Solubility, pH of the GI tract and size of fibre particles.

3) a) The physiological effect with respect to water holding capacity include delay emptying of food from stomach, reduces enzyme function, decreases nutrient diffusion rate and delayed nutrient absorption, and small intestine transit time.

b) Absorption property effect include diminished absorption of lipids, increased faecal excretion of bile acids absorbed to fibre cannot be reabsorbed and recirculated, hypocholesterolenic properties, and altered mineral balance.

4) Inclusion of fibre retards the absorption of some nutrients, as well as, post-prandial glucose level concentration. A few mechanisms by which nutrient absorption is delayed are inadequate mixing of luminal contents due to increased viscosity may slow the movement of digestive enzymes to their substrate thus delaying digestion; and viscous properties inhibit the access of nutrients to the epithelium.

5) There are numerous hypotheses as to how fiber might protect against the development of colon cancer. These include the dilution of carcinogens, procarcinogens, and tumor promoters in a bulky stool, a more rapid rate of transit through the colon with high-fiber diets, a reduction in the ratio of secondary bile acids to primary bile acids by acidifying colonic contents, the production of butyrate from the fermentation of dietary fiber by the colonic microflora, and the reduction of ammonia, which is known to be toxic to cells.

Check Your Progress Exercise 3

1) The starch which escapes enzymatic hydrolysis in the small intestine and passes to the colon where it is fermented by colonic rnicroflora is referred to as RS. It is found in partially milled grains and seeds, raw potatoes, rice and modified starches.

2) The factors affecting the RS content of foods include amylose / amylopectin ratio, water content, presence of sugar and lipids, calcium and potassium ions, polyphenols and phytic acid, antinutritional factors, and processing techniques. Look up sub-section 3.8.1 for details related to these factors.

3) The factors which contribute to the anti-carcinogenic effect of RS include the protective effects of butyrate, reduced concentrations of total and secondary bile acids, lowering of pH by SCFAs reduces activity of 7 a-dehydroxylase, reduced bacterial β-glucosidase activity, and reduced concentration of ammonia.

4) The dietary GI provides an indication of the rate at which carbohydrate foods are digested. It allows ranking of foods from those which give rise to the highest

blood glucose and insulin responses to those associated with lowest blood glucose and insulin responses. It integrates multiple influences as glucose availability and is proposed as a means for prescribing diabetic and energy controlled diets.

5) The factors which affect the GI value of foods include nature of starch, nature of monosaccharide components, presence of viscous fibre such as guar gum and β-glucan, cooking/processing method, particle size, ripeness and food storage, presence of α-amylase inhibitors like lecithin, phytates etc., and nutrients-starch interactions. High levels of protein and fat decreases the GI.

6) High GI foods result in fast carbohydrate absorption, large blood glucose and hormonal fluctuations and reduced satiety resulting in weight gain and overeating, leading to obesity.

7) a) Glycemic index is a quantitative assessment of foods based on postprandial blood glucose response expressed as a percentage of the response to an equivalent carbohydrate portion of a reference food.

b) FOS are polymers of fructose Units (2-8) usually attached to an initial glucose molecules.