Nutrition & Biochemistry for Nurses

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CHAPTER

2 Carbohydrates

Carbohydrates or saccharides are the most abundant of the four major classes of biomolecules. Carbohydrates are simple organic compounds that are aldehydes or ketones with many hydroxyl groups added, usually one on each carbon atom that is not part of the aldehyde or ketone functional group. The general stoichiometric formula of an unmodified monosaccharide is $(CH_2O)_n$, where 'n' is any number of three or greater; however, not all carbohydrates conform to this precise stoichiometric definition (e.g., uronic acids, deoxy-sugars such as fucose), nor are all chemicals that do conform to this definition automatically classified as carbohydrates.

CLASSIFICATION

There are a variety of interrelated classification schemes. The most useful classification scheme divides the carbohydrates into groups according to the number of individual simple sugar units (Table 2.1):

- *Monosaccharides* contain a single unit
- *Disaccharides* contain two sugar units, and
- *Polysaccharides* contain many sugar units as in polymers - most contain glucose as the monosaccharide unit.

Monosaccharides can be further classified by the number of carbons present (Table 2.2). Hexoses (6-carbons) are by far the most prevalent.

Classification Based on Functional Groups

Aldoses contain the aldehyde group - Monosaccharides in this group are glucose, galactose, ribose, and glyceraldehyde.

Table 2.1: Classification of carbohydrates based on number of monomeric units

Monosaccharides	*Disaccharides*	*Polysaccharides*
Glucose	Sucrose	Starch
Galactose	Maltose	Glycogen
Fructose	Lactose	Cellulose
Ribose		
Glyceraldehyde		

Table 2.2: Classification of monosaccharides based on number of carbons present

Hexoses (6C)	*Pentoses (5C)*	*Trioses (3C)*
Glucose	Ribose	Glyceraldehyde
Galactose		
Fructose		

Ketoses contain the ketone group - The major sugar in this group is fructose.

Reducing: Contain a hemiacetal or hemiketal group – The major sugars in this group include, glucose, galactose, fructose, maltose, and lactose.

Non-reducing: Contain no hemiacetal groups. The major sugars in this group include, sucrose and all polysaccharides.

CALORIE VALUE

1 gram of carbohydrate yields 4 Calories of energy.

RECOMMENDED DAILY ALLOWANCES

At least 313 grams of carbohydrate per day should be consumed, if daily calorie requirement is 2,500 calories.

At least 250 grams of carbohydrate per day should be consumed, if daily calorie requirement is 2,000 calories

At least 188 grams of carbohydrate per day should be consumed, if daily calorie requirement is 1,500 calories.

Most people should get between 40% and 60% of total calories from carbohydrates, preferably from complex carbohydrates (starches) and natural sugars. Complex carbohydrates provide calories, vitamins, minerals, and fiber.

Foods that are high in processed, refined simple sugars provide calories, but very little nutrition. It is wise to limit these sugars.

To increase complex carbohydrates and healthy nutrients:

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Table 2.3: Common carbohydrates and their dietary source

Name	*Derivation of name and source*
	Monosaccharides(contain single monosaccharide)
Glucose	From Greek word for sweet wine; grape sugar, blood sugar, dextrose.
Galactose	Greek word for milk—"galact", found as a component of lactose in milk.
Fructose	Latin word for fruit—"fructus", also known as levulose, found in fruits and honey; sweetest sugar.
Ribose	Ribose and deoxyribose are found in the backbone structure of RNA and DNA, respectively.
	Disaccharides (contain two monosaccharides)
Sucrose	French word for sugar—"sucre", a disaccharide containing **glucose and fructose**; table sugar, cane sugar, beet sugar.
Lactose	Latin word for milk—"lact"; a disaccharide found in milk containing **glucose and galactose**.
Maltose	French word for "malt"; a disaccharide containing **two units of glucose**; found in germinating grains, used to make beer.
	Common Polysaccharides
Starch	Plants store glucose as the polysaccharide starch. The cereal grains (wheat, rice, corn, oats, barley) as well as tubers such as potatoes are rich in starch.
Cellulose	The major component in the rigid cell walls in plants is cellulose and is a linear polysaccharide polymer with many glucose monosaccharide units.
Glycogen	This is the storage form of glucose in animals and humans which is analogous to the starch in plants. Glycogen is synthesized and stored mainly in the liver and the muscles.

- Eat more fruits and vegetables.
- Eat more whole-grain rice, breads, and cereals.
- Eat more legumes (beans, lentils, and dried peas).

Here are recommended serving sizes for foods high in carbohydrates:

- Vegetables: 1 cup of raw vegetables, or 1/2 cup cooked vegetables, or 3/4 cup of vegetable juice
- Fruits: 1 medium-size fruit (such as 1 medium apple or 1 medium orange), 1/2 cup of a canned or chopped fruit, or 3/4 cup of fruit juice
- Breads and cereals: 1 slice of bread; 1 ounce or 2/3 cup of ready-to-eat cereal; 1/2 cup of cooked rice, pasta, or cereal; 1/2 cup of cooked dry beans, lentils, or dried peas
- Dairy: 1 cup of skim or low-fat milk

For information about how many servings are recommended, see the food guide pyramid.

Here is a sample 2,000 calorie menu, of which 50% - 60% of the total calories are from carbohydrates.

- Breakfast
  - Cold cereal
    - 1 cup shredded wheat cereal
    - 1 tbsp raisins
    - 1 cup fat-free milk
  - 1 small banana
  - 1 slice whole-wheat toast
    - 1 tsp soft margarine
    - 1 tsp jelly
- Lunch
  - Smoked turkey sandwich
    - 2 ounces whole-wheat pita bread
    - 1/4 cup romaine lettuce
    - 2 slices tomato
    - 3 ounces sliced smoked turkey breast
    - 1 tbsp mayo-type salad dressing
    - 1 tsp yellow mustard
  - 1/2 cup apple slices
  - 1 cup tomato juice
- Dinner
  - Grilled top loin steak
    - 5 ounces grilled top loin steak
  - 3/4 cup mashed potatoes
    - 2 tsp soft margarine
  - 1/2 cup steamed carrots
    - 1 tbsp honey
  - 2 ounces whole-wheat dinner roll
    - 1 tsp soft margarine
  - 1 cup fat-free milk
- Snacks
  - 1 cup low-fat fruited yogurt

DIETARY SOURCES

There are innumerable sources of carbohydrates (Table 2.3), but not all of them are nutritious. It is

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recommended to opt for healthy natural carbohydrate food sources that have not undergone any sort of processing, since many nutrients are lost during processing.

Tables 2.4 and 2.5 show the high and low carbohydrate sources respectively.

FUNCTIONS

Carbohydrates have six major functions within the body:

- Providing energy and regulation of blood glucose
- Sparing the use of proteins for energy
- Preventing Breakdown of fatty acids and ketosis
- Biological recognition processes
- Flavor and sweeteners
- Dietary fiber

Providing Energy and Regulating Blood Glucose

Glucose is the only sugar used by the body to provide energy for its tissues. Therefore, all digestible polysaccharides, disaccharides, and monosaccharides must eventually be converted into glucose or a metabolite of glucose by various liver enzymes. Because of its significant importance to proper cellular function, blood glucose levels must be kept relatively constant.

Among the enormous metabolic activities the liver performs, it also includes regulating the level of blood glucose. During periods of food consumption, pancreatic beta cells sense the rise in blood glucose and begin to secrete the hormone insulin. Insulin binds to many cells in the body having appropriate receptors for the peptide hormone and causes a general uptake in cellular glucose. In the liver, insulin causes the uptake of glucose as well as the synthesis of glycogen, a glucose storage polymer. In this way, the liver is able to remove excessive levels of blood glucose through the action of insulin.

In contrast, the hormone glucagons is secreted into the bloodstream by pancreatic alpha cells upon sensing falling levels of blood glucose. Upon binding to targeted cells such as skeletal muscle and brain cells, glucagon acts to decrease the amount of glucose in the bloodstream. This hormone inhibits the uptake of glucose by muscle and other cells and promotes the breakdown of glycogen in the liver in order to release glucose into the blood. Glucagon also promotes gluconeogenesis, a process involving the synthesis of glucose from amino acid precursors. Through the effects of both glucagon and insulin, blood glucose can usually be regulated in concentrations between 70 and 115 mg/100 ml of blood.

Other hormones of importance in glucose regulation are epinephrine and cortisol. Both hormones are secreted from the adrenal glands, however, epinephrine mimics the effects of glucagon while cortisol mobilizes glucose during periods of emotional stress or exercise.

Despite the liver's unique ability to maintain homeostatic levels of blood glucose, it only stores enough for a twenty-four hour period of fasting. After twenty four hours, the tissues in the body that preferentially rely on glucose, particularly the brain and skeletal muscle, must seek an alternative energy source. During fasting periods, when the insulin to glucagons ratio is low, adipose tissue begins to release fatty acids into the bloodstream. Fatty acids are long hydrocarbon chains consisting of single carboxylic acid group and are not very soluble in water. Skeletal muscle begins to use fatty acids for energy during resting conditions; however, the brain cannot afford the

Table 2.4: High carbohydrate sources

Foodstuff	*Carbohydrate content per 100 gms of edible portion*
Jowar	72.6 gms
Rice (raw)	78.2 gms
Ragi	72.0 gms
Whole wheat flour	69.4 gms
Bengal gram whole	60.9 gms
Green gram dhal	59.9 gms
Rajmah	60.6 gms
Arrow root flour	83.1 gms
Apricot dry	73.4 gms
Dry dates	75.8 gms
Cane Sugar	99.4 gms
Honey	79.5 gms
Jaggery	95 gms

Table 2.5: Low carbohydrate sources

Foodstuff	*Carbohydrate content per 100 gms of edible portion*
Spinach	2.9 gms
Bottle gourd	2.5 gms
Cucumber	2.5 gms
Snake gourd	3.3 gms
Figs	7.6 gms
Musambi	9.3 gms
Straw berry	9.8 gms
Cow's milk	4.4 gms
Butter milk	0.5 gms
Cauliflower	4 gms
Ladies finger	6.4 gms
Tomato green	3.6 gms
Melon	3.5 gms
Fish	2 gms
Curd	3.0 gms
Beet root	8.8 gms

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same luxury. Fatty acids are too long and bulky to cross the blood-brain barrier. Therefore, proteins from various body tissues are broken down into amino acids and used by the liver to produce glucose for the brain and muscle. This process is known as gluconeogenesis or "the production of new glucose." If fasting is prolonged for more than a day, the body enters a state called ketosis. Ketosis comes from the root word ketones and indicates a carbon atom with two side groups bonded to an oxygen atom. Ketones are produced when there is no longer enough oxaloacetate in the mitochondria of cells to condense with acetyl CoA formed from fatty acids. Oxaloacetate is a four-carbon compound that begins the first reaction of the Krebs Cycle, a cycle containing a series of reactions that produces high-energy species to eventually be used to produce energy for the cell. Since oxaloacetate is formed from pyruvate (a metabolite of glucose), a certain level of carbohydrate is required in order to burn fats. Otherwise, fatty acids cannot be completely broken down and ketones will be produced.

Sparing Protein and Preventing Ketosis

So why are carbohydrates important if the body can use other carbon compounds such as fatty acids and ketones as energy? First of all, maintaining a regular intake of carbohydrates will prevent protein from being used as an energy source. Gluconeogenesis will slow down and amino acids will be freed for the biosyntheses of enzymes, antibodies, receptors and other important proteins. Furthermore, an adequate amount of carbohydrates will prevent the degradation of skeletal muscle and other tissues such as the heart, liver, and kidneys. Most importantly, ketosis will be prevented. Although the brain will adapt to using ketones as a fuel, it preferentially uses carbohydrates and requires a minimum level of glucose circulating in the blood in order to function properly. Before the adaptation process occurs, lower blood glucose levels may cause headaches in some individuals. To prevent these ketotic symptoms, it is recommended that the average person consume at least 50 to 100 g of carbohydrates per day.

Although the processes of protein degradation and ketosis can create problems of their own during prolonged fasting, they are adaptive mechanisms during glucose shortages. In summary, the first priority of metabolism during a prolonged fast is to provide enough glucose for the brain and other organs that dependent upon it for energy in order to spare proteins for other cellular functions. The next priority of the body is to shift the use of fuel from glucose to fatty acids and ketone bodies. From then on, ketones become more and more important as a source of fuel while glucose become less important.

Flavor and Sweeteners

A less important function of carbohydrates is to provide sweetness to foods. Receptors located at the tip of the tongue bind to tiny bits of carbohydrates and send what humans perceive as a "sweet" signal to the brain. However, different sugars vary in sweetness. For example, fructose is almost twice as sweet as sucrose and sucrose is approximately 30% sweeter than glucose.

Sweeteners can be classified as either nutritive or alternative. Nutritive sweeteners have all been mentioned before and include sucrose, glucose, fructose, high fructose corn syrup, and lactose. These types of sweeteners not only impart flavor to the food, but can also be metabolized for energy. In contrast, alternative sweeteners provide no food energy and include saccharin, cyclamate, aspartame, and acesulfame. Controversy over saccharin and cyclamate as artificial sweeteners still exists but aspartame and acesulfame are used extensively in many foods in the United States. Aspartame and acesulfame are both hundreds of times sweeter than sucrose but only acesulfame is able to be used in baked goods since it is much more stable than aspartame when heated.

Dietary Fiber

Dietary fibers such as cellulose, hemicellulose, pectin, gum and mucilage are important carbohydrates for several reasons. Soluble dietary fibers like pectin, gum and mucilage pass undigested through the small intestine and are degraded into fatty acids and gases by the large intestine. The fatty acids produced in this way can either be used as a fuel for the large intestine or be absorbed into the bloodstream. Therefore, dietary fiber is essential for proper intestinal health.

In general, the consumption of soluble and insoluble fiber makes the elimination of waste much easier. Since dietary fiber is both indigestible and an attractant of water, stools become large and soft. As a result, feces can be expelled with less pressure. However, not enough fiber consumption will change the constitution of the stool and increase the amount of force required during defecation. Excessive pressure during the elimination of waste can force places in the large intestine wall out from between bands of smooth muscle to produce small pouches called diverticula. Hemorrhoids may also result from unnecessary strain during defecation.

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The disease of having many diverticula in the large intestine is known as diverticulosis. Although diverticula is often asymptomatic, food particles become trapped in their folds and bacteria begin to metabolize the particles into acids and gases. Eventually, the diverticula may become inflamed, a condition known as diverticulitis. To combat the disease, antibiotics are administered to the patient to destroy the bacteria while the intake of fiber in the diet is decreased until the inflammation has subsided. Once the inflammation has been reduced, a high fiber diet is begun to prevent a relapse.

Besides the prevention of intestinal disease, diets high in fiber have other health benefits. High fiber intake reduces the risk of developing obesity by increasing the bulk of a meal without yielding much energy. An expanded stomach leads to satisfaction despite the fact that the caloric intake has decreased.

Beyond dieters, diabetics can also benefit from consuming a regular amount of dietary fiber. Once in the intestine, it slows the absorption of glucose to prevent a sudden increase in blood glucose levels. A relatively high intake of fiber will also decrease the absorption of cholesterol, a compound that is thought to contribute to atherosclerosis or scarring of the arteries. Serum cholesterol may be further reduced by a reduction in the release of insulin after meals. Since insulin is known to promote cholesterol synthesis in the liver, a reduction in the absorption of glucose after meals through the consumption of fiber can help to control serum cholesterol levels. Furthermore, dietary fiber intake may help prevent colon cancer by diluting potential carcinogens through increased water retention, binding carcinogens to the fiber itself and speeding the passage of food through the intestinal tract so that cancer-causing agents have less time to act.

Biological Recognition Processes

Carbohydrates not only serve nutritional functions, but are also thought to play important roles in cellular recognition processes. For example, many immunoglobulins (antibodies) and peptide hormones contain glycoprotein sequences. These sequences are composed of amino acids linked to carbohydrates. During the course of many hours or days, the carbohydrate polymer linked to the rest of the protein may be cleaved by circulating enzymes or be degraded spontaneously. The liver can recognize differences in length and may internalize the protein in order to begin its own degradation. In this way, carbohydrates may mark the passage of time for proteins.

DIGESTION, ABSORPTION AND STORAGE

Digestion

Carbohydrates are formed in growing plants and are found in grains, leafy vegetables, and other edible plant foods. The molecular structure of these plants is complex, or a polysaccharide; poly is a prefix meaning many. Plants form carbohydrate chains during growth by trapping carbon from the atmosphere, initially carbon dioxide (CO_2). Carbon is stored within the plant along with water (H_2O) to form a complex starch containing a combination of carbon-hydrogen-oxygen in a fixed ratio of 1:2:1 respectively.

Plants with a high sugar content and table sugar represent a less complex structure and are called disaccharides, or two sugar molecules bonded. Once digestion of either of these forms of carbohydrates are complete, the result is a single sugar structure, a monosaccharide. These monosaccharides can be absorbed into the blood and used by individual cells to produce the energy compound adenosine triphosphate (ATP).

The digestive system starts the process of breaking down polysaccharides in the mouth through the introduction of amylase, a digestive enzyme in saliva. The high acid content of the stomach inhibits the enzyme activity, so carbohydrate digestion is suspended in the stomach. Upon emptying into the small intestines, potential hydrogen (pH) changes dramatically from a strong acid to an alkaline content. The pancreas secretes bicarbonate to neutralize the acid from the stomach, and the mucus secreted in the tissue lining the intestines is alkaline which promotes digestive enzyme activity. Amylase is secreted by the pancreas into the small intestines and works with other enzymes to complete the breakdown of carbohydrate into monosaccharides which is absorbed into the surrounding capillaries of the villi.

Nutrients in the blood are transported to the liver via the hepatic portal circuit, or loop, where final carbohydrate digestion is accomplished in the liver. The liver accomplishes carbohydrate digestion in response to the hormones insulin and glucagon. As blood glucose levels increase following digestion of a meal, the pancreas secretes insulin causing the liver to transform glucose to glycogen, which is stored in the liver, adipose tissue, and in muscle cells, preventing hyperglycemia. A few hours following a meal, blood glucose will drop due to muscle activity, and the pancreas will now secrete glucagon which causes glycogen to be converted into glucose to prevent hypoglycemia.

Note: In the discussion of digestion of carbohydrates; nouns ending in the suffix -ose usually indicate a sugar, such as lactose. Nouns ending in the suffix -ase indicates the enzyme that will break down the sugar, such as lactase. Enzymes usually begin with the substrate (substance) they

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are breaking down. For example, malt**ose**, a disaccharide, is broken down by the enzyme malt**ase** (by the process of hydrolysis), resulting in a two glucose molecules, a monosaccharide.

Simple sugars are the predominant carbohydrate absorbed in the digestive tract, and in many animals the most important source of energy. Monosaccharides, however, are only rarely found in normal diets. Rather, they are derived by enzymatic digestion of more complex carbohydrates within the digestive tube.

Particularly important dietary carbohydrates include starch and disaccharides such as lactose and sucrose. None of these molecules can be absorbed for the simple reason that they cannot cross cell membranes unaided and, unlike the situation for monosaccharides, there are no transporters to carry them across.

This section will focus on understanding the processes involved in assimilation of three important carbohydrates: starch, lactose and sucrose. The key concepts involved in all three cases are that:

- The final enzymatic digestion that liberates monosaccharides is conducted by enzymes that are tethered in the lumenal plasma membrane of absorptive enterocytes (so-called *"brush border hydrolyases"*).
- Glucose generated by digestion of starch or lactose is absorbed in the small intestine only by *cotransport with sodium*, a fact that has exceptionally important implications in medicine.

Brush Border Hydrolases Generate Monosaccharides

Polysaccharides and disaccharides must be digested to monosaccharides prior to absorption and the key players in these processes are the brush border hydrolases, which include maltase, lactase and sucrase. Dietary lactose and sucrose are "ready" for digestion by their respective brush border enzymes. Starch, as discussed previously, is first digested to maltose by amylase in pancreatic secretions and, in some species, saliva.

Dietary lactose and sucrose, and maltose derived from digestion of starch, diffuse in the small intestinal lumen and come in contact with the surface of absorptive epithelial cells covering the villi where they engage with brush border hydrolases:

- Maltase cleaves maltose into two molecules of glucose
- Lactase cleaves lactose into a glucose and a galactose
- Sucrase cleaves sucrose into a glucose and a fructose

At long last, we're ready to actually absorb these monosaccharides. Glucose and galactose are taken into the enterocyte by cotransport with sodium using the same transporter. Fructose enters the cell from the intestinal lumen via facilitated diffusion through another transporter.

Absorption

Absorption of glucose entails transport from the intestinal lumen, across the epithelium and into blood. The transporter that carries glucose and galactose into the enterocyte is the sodium-dependent hexose transporter, known more formally as SGLUT-1. As the name indicates, this molecule transports both glucose and sodium ion into the cell and in fact, will not transport either alone.

The essence of transport by the sodium-dependent hexose transporter involves a series of conformational changes induced by binding and release of sodium and glucose, and can be summarized as follows (Fig. 2.1):

α-amylase	α-*amylase* hydrolyzes α-1, 4 glycosidic bonds of complex carbohydrates.
	Salivary amylase: degraded in acidic environment (pH 3 - 3.5) of the stomach.
	Pancreatic amylase: responsible for intraluminal starch hydrolysis.
Oligosaccharide products of α-amylase digestion are hydrolyzed by enzymes on the brush border membrane surface. Only free glucose is transported into the enterocytes.	

Enzymes in the brush border responsible for hydrolysis of oligosaccharides to glucose	Glucoamylase: removes single glucose residues sequentially from the non-reducing end of the chain.
	Sucrase: efficiently hydrolyzes short α-1,4-linked oligosaccharides such as maltose and maltotriose.
	Isomaltase: cleaves the non-reducing, terminal α-1, 6 bond.
	Lactase: hydrolyzes lactose to glucose and galactose.

Enzymes levels (expression) are modified by dietary alterations

- A carbohydrate free diet decreases sucrase level
- A high sucrose diet increases level of sucrase and its m-RNA

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1. The transporter is initially oriented facing into the lumen - at this point it is capable of binding sodium, but not glucose
2. Sodium binds, inducing a conformational change that opens the glucose-binding pocket
3. Glucose binds and the transporter reorients in the membrane such that the pockets holding sodium and glucose are moved inside the cell
4. Sodium dissociates into the cytoplasm, causing glucose binding to destabilize
5. Glucose dissociates into the cytoplasm and the unloaded transporter reorients back to its original, outward-facing position

The Fig. 2.1 depicts entry of glucose, along with sodium, into the enterocyte (actually, two sodium ions are transported for each glucose). Despite the simplicity of the diagram, you should easily be able to identify the sodium-dependent hexose transporter and its conformational changes. Also, imagine the corresponding process involving lactose and sucrose assimilation.

Fructose is not co-transported with sodium. Rather it enters the enterocyte by another hexose transporter (GLUT5).

Once inside the enterocyte, glucose and sodium must be exported from the cell into blood. We've seen previously how sodium is rapidly shuttled out in exchange for potassium by the battery of sodium pumps on the basolateral membrane, and how that process maintains the electrochemical gradient across the epithelium. The energy stored in this gradient is actually what is driving glucose entry through the sodium-dependent hexose transporter described above. Recall also how the massive transport of sodium out of the cell establishes the osmotic gradient responsible for absorption of water.

Glucose, galactose and fructose are tranported out of the enterocyte through another hexose transporter (called GLUT-2) in the basolateral membrane. These monosaccharides then diffuse "down" a concentration gradient into capillary blood within the villus.

Glucose uptake is Na^+ dependent (co-transport), and fructose is absorbed via facilitated diffusion.

Galactose and Glucose are Actively Transported

1. SGLT1 is the transport protein responsible for Na^+ dependent glucose transport.
2. Glut-2 transports glucose out of the cell into the portal circulation.

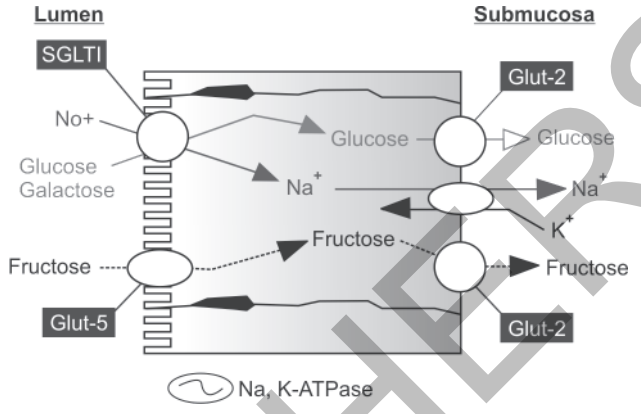

Fig. 2.1: Transport of monosaccharides in entrocytes

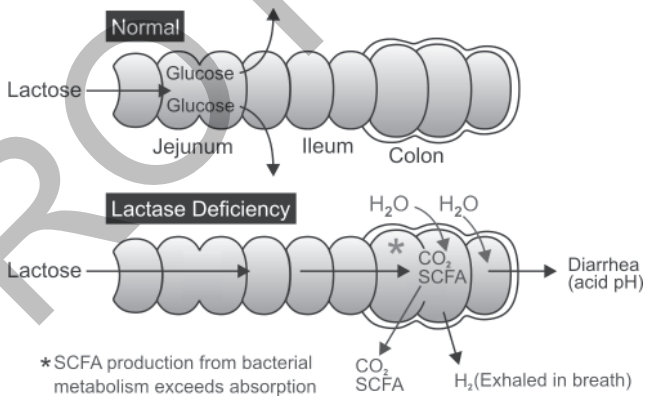

Fig. 2.2: Pathophysiology of lactose intolerance, SFCA=Short-chain fatty acid

3. Also transports galactose and fructose.
4. A high carbohydrate diet increases SGLT1 and Glut-2 levels.
5. SGLT1 mRNA and protein are increased in diabetes mellitus

Galactosemia (Fig. 2.2)

An inborn error of galactose metabolism (autosomal recessive trait) manifested as failure to thrive in newborn children due to anorexia, vomiting and diarrhea when milk or sugar water is ingested.

Lactose Intolerance

Lactase activity diminishes to 10% of neonatal level by age 5-10 yrs.

Symptoms: abdominal pain, cramping, distention, flatulence, diarrhea, possible nausea and vomiting.

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Mechanism: Lactose draws water into the intestinal lumen and produces osmotic diarrhea. Gut flora metabolizes the lactose to form gases such as hydrogen, methane, and carbon dioxide.

Storage

There are only 1200 calories stored as carbohydrate in liver and muscle glycogen. There are clear adaptive advantages to storing calories as fat, since fat can provide more energy per gram than carbohydrate or protein. However, since carbohydrate stores are so small, they are depleted in three days of uncomplicated starvation or sooner under conditions of increased energy expenditure. This dependence on fat and protein stores in starvation requires metabolic adaptations to minimize the loss of protein stores, and a shift to metabolic pathways predominantly utilizing the large fat stores available.

The most abundant forms of edible carbohydrates are the starches provided by grains and tubers. They provide the bulk of human energy needs, except in affluent Western societies where fat is a major source of energy and where sucrose supplies almost as much energy as starch. It is helpful to understand the particular problem that the organism has to solve when processing carbohydrates. For carbohydrates to be absorbed, they must first be broken down into monosaccharides. Small molecules exert a powerful osmotic effect and it is therefore important that intestinal absorption be able to keep up with the rate of starch hydrolysis. Furthermore, absorption must be completed before carbohydrates are subjected to bacterial fermentation; otherwise, there are unpleasant side effects, such as those experienced after lactose ingestion by individuals with low intestinal lactase activity. Meals commonly supply 50-150 g carbohydrate. This is much more than the 15-25 g free glucose present in the body, of which only *5* g is in the blood itself. To avoid hyperglycemia and spillage of glucose into the urine, glucose transferred into the circulation must be rapidly transferred into cells. Because glucose oxidation in the postprandial state consumes only 10 g/h, most of the glucose taken up must be stored by conversion to glycogen, primarily in liver and muscle. Glucose uptake and glycogen synthesis are greatly stimulated by insulin, the secretion of which increases when blood glucose concentrations rise. Two moles ATP are expended to incorporate 1 mol glucose into glycogen. Because 36 ATP are gained during the complete oxidation of one molecule of glucose, or 5% of the energy content of glucose must be expended to store it as glycogen. Glycogen molecules are very large; they are thus effectively trapped in the cells in which they were made without creating much of an osmotic effect. In view of the hydrophilic nature of carbohydrates, glycogen is associated with 3 g water/g glycogen. The energy density of glycogen stores is thus only 4.2 kJ/g (1 kcal/g), imposing definite limits on the amount of energy that can conveniently be carried in the form of glycogen. Glycogen concentrations are highest in the liver, i.e. typically 4% after an overnight fast, and up to 8% after meals. Because an adult's liver weighs 1.5 kg, hepatic glycogen storage capacity is limited to 120 g. Glycogen amounts in muscle are much lower and deliberate carbohydrate loading is necessary to raise them much above 2%. However, because muscle accounts for 20-30% of total body weight, the amount of glycogen stored in muscle is generally three to four times that in the liver. Total glycogen stones in adults can thus be estimated to be "200-500 g, depending on body size and on the amount of carbohydrate consumed, and vary substantially during the day as a function of food intake and exertion. The body's glycogen reserve is in effect not much greater than the amount of carbohydrate usually consumed in 1 day, and maintenance of glycogen amounts within a desirable range requires effective adjustment of carbohydrate oxidation to carbohydrate intake.

Marked glycogen depletion causes a threat to the ability of the liver to ensure adequate circulating blood glucose concentrations, which requires the release of *5* g glucose/h to replace the glucose removed by the nervous system. Because the influx of nutrients from the intestine occurs through the portal system, the liver has a prime opportunity to replenish its glycogen reserve after consumption of carbohydrates. Usually, this demands only a fraction of the carbohydrates provided by a meal and substantial amounts of glucose reach the periphery and are stored in muscle. The relative roles of liver and muscle in the disposition of a carbohydrate load vary depending on the degree of depletion of the glycogen stores, the size of the meal, and the type of carbohydrate consumed. It seems reasonable to envision that after a typical meal one-quarter to one-third of the carbohydrate is converted to glycogen in the liver and one-third to one-half to glycogen in muscle, whereas the remainder is oxidized during the postpnandial hours of tissue, making it possible for animals to accumulate and carry a reserve of fuel substantial enough to allow survival during extended periods of food deprivation.

Dietary fats are emulsified in the intestine with the help of bile and then degraded to fatty acids and glycerol by pancreatic and intestinal lipases. These are absorbed from the lumen, re-esterified into triglycerides by the intestinal cells, and secreted into the lymphatic vessels, draining the mesentery in the form of chylomicrons. These

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tiny lipid droplets are solubilized by a thin coating of apolipoproteins, allowing them to remain in the circulation. They give the plasma its milky appearance a few hours after a fatty meal has been consumed. To be transferred into cells, the triglycerides of the chylomicrons must be hydrolyzed, a function carried out by lipoprotein lipase (LPL), an enzyme produced most abundantly by the endothelial cells of adipose tissue capillaries. The fatty acids produced by LPL are promptly taken up by the fat cells and re-esterified, only a small fraction escaping to reach the pool of albumin-bound fatty acids, the form in which fatty acids are made available as a metabolic fuel. Dietary fatty acids are thus targeted for deposition in adipose tissue.

Conversion of Carbohydrate into Fat

Conversion of carbohydrate into fat allows animals to build up fat reserves even when their feed contains very little fat. In adult humans such de novo lipogenesis is primarily a hepatic process. Quantitatively, this conversion plays an insignificant role in adults consuming mixed diets. Even after ingestion of 500 g carbohydrate, the nonprotein respiratory quotient (RQ) (i.e. the ratio of CO_2 produced to O_2 consumed by carbohydrate and fat metabolism) does not rise above 1.0, which indicates that fatty acids are being synthesized at a rate exceeding the concomitant rate of fat oxidation.

Thus, unusually large, occasional carbohydrate loads are handled primarily by converting the absorbed glucose into glycogen. The massive expansion of the glycogen stores then leads to the nearly exclusive use of glucose as a fuel, in time reducing such temporary accumulations of glycogen. To induce substantial rates of carbohydrate conversion into fat, the body's total glycogen stores must be considerably raised, from their usual 4-6 g/kg body wt to > 8-10 g/kg body wt. This requires deliberate and sustained over consumption of large amounts of carbohydrates for 2-3 days.

Two important conclusions can be deduced from these observations: *(1)* under usual conditions of unrestricted access to food, glycogen stores are spontaneously maintained far below their maximal capacity, and *(2)* the common belief that carbohydrates are readily turned into fat can be dismissed as well as the frequently made argument that the high metabolic cost of lipogenesis is a cause for greater energy dissipation on high-carbohydrate diets. It should not be over looked, however, that consumption of carbohydrates reduces the need to use fat as a fuel, so that carbohydrate intake is an important factor in determining how much of the fat consumed will be retained.

METABOLISM

The major source of dietary carbohydrate for humans is starch from consumed plant material. This is supplemented with a small amount of glycogen from animal tissue, disaccharides such as sucrose from products containing refined sugar and lactose in milk.

Digestion in the gut converts all carbohydrate to monosaccharides which are transported to the liver and converted to glucose. The liver has a central role in the storage and distribution within the body of all fuels, including glucose.

Glucose in the body undergoes one of three metabolic fates:

- It is catabolized to produce ATP
  - This occurs in all peripheral tissues, particularly in brain, muscle and kidney.
- It is stored as glycogen
  - This storage occurs in liver and muscle.
- It is converted to fatty acids

Once converted to fatty acids, these are stored in adipose tissue as triglycerides.

Glucose Catabolism

Glucose will be oxidised by all tissues to synthesise ATP. The first pathway which begins the oxidation of glucose is called **glycolysis.**

Glycolysis

This pathway cleaves the six carbon glucose molecule ($C_6H_{12}O_6$) into two molecules of the three carbon compound pyruvate ($C_3H_3O_3^-$). This oxidation is coupled to the net production of two molecules of ATP/glucose.

The outline of glycolysis is shown in Figure 2.3. The full set of reactions and structures can be found in any biochemistry textbook.

One oxidation reaction occurs in the latter part of the pathway. It uses NAD as the electron acceptor. This cofactor is present only in limited amounts and once reduced to NADH, as in this reaction, it must be re-oxidised to NAD to permit continuation of the pathway.

This re-oxidation occurs by one of two methods:

Anerobic glycolysis

- Pyruvate is reduced to a compound called lactate. This single reaction occurs in the absence of oxygen (anerobically) and is ideally suited to

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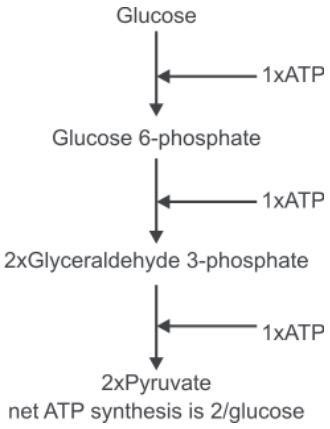

Fig. 2.3: Outline of glycolysis

utilization in heavily exercising muscle where oxygen supply is often insufficient to meet the demands of aerobic metabolism. The reduction of pyruvate to lactate is coupled to the oxidation of NADH to NAD^+.

Aerobic metabolism of glucose

- Pyruvate is transported inside **mitochondria** and oxidised to a compound called **acetyl coenzyme A** (abbreviated to "acetyl CoA"). This is an oxidation reaction and uses NAD^+ as an electron acceptor.

By a further series of reactions collectively called the ***citric acid cycle***, acetyl CoA is oxidised ultimately to CO_2. These reactions are coupled to a process known as the ***electron transport chain*** which has the role of harnessing chemical bond energy from a series of oxidation/reduction reactions to the synthesis of ATP and simultaneously re-oxidising NADH to NAD. (These pathways will be discussed in more detail later.)

Fast twitch muscle fibers utilize the first of the two mechanisms described above almost exclusively. Very heavily exercising muscle can use this pathway as the sole source of ATP synthesis for a short period of time. This probably evolved in humans as a defence mechanism, but is now used by athletes in sprint events.

The formation of lactate as an end product from glucose extracts only a relatively small amount of the bond energy contained in glucose. Accumulation of lactate (actually lactic acid) also causes a reduction in intracellular pH.

The lactate formed is removed to other tissues and dealt with by one of two mechanisms:

- It is converted back to pyruvate

  The pyruvate then proceeds to be further oxidized by the second mechanism described above, finally producing a large amount of ATP. It is converted back to glucose in the liver

Gluconeogenesis

The process of conversion of lactate to glucose is called ***gluconeogenesis***, uses some of the reactions of glycolysis (but in the reverse direction) and some reactions unique to this pathway to re-synthesise glucose. This pathway requires an energy input (as ATP) but has the role of maintaining a circulating glucose concentration in the bloodstream (even in the absence of dietary supply) and also maintaining a glucose supply to fast twitch muscle fibers.

Cori Cycle

It can be shown by a complex calculation of energy yields that this process of partially oxidising glucose to lactate in muscle, transporting it to the liver for conversion back to glucose and then re-supplying it to muscle, actually has a much higher energy yield than the 2 ATP/glucose produced by glycolysis alone. This co-operative cycle utilising both the muscle and liver tissue is called the Cori cycle. The process is shown in Figure 2.4.

Both of these mechanisms illustrate the interdependence of tissues on each other and the co-operative activities between organs which make up the total of the body's metabolic activities.

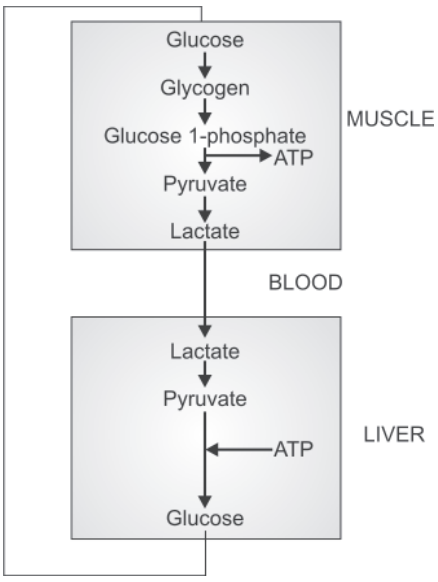

Fig. 2.4: Cori's cycle showing glycolysis and gluconeogenesis

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Glycogen and Glucose Interconversion

Glycogen is a highly branched polymer of glucose. The high degree of branching (about every twelve glucose residues) produces a molecule which is compact and thus can be efficiently stored in the limited space available in liver and muscle cells.

Even though the branching is designed to make the molecule compact, it is still a polar molecule and thus must be stored with associated water. It is stored as aggregates of glycogen molecules within cells (visible microscopically as glycogen granules) with up to 70% of the aggregate being water.

The amount of glycogen in muscle changes substantially between the fed state and following heavy exercise. The amount of glycogen stored in the liver is more constant and only falls substantially after prolonged starvation.

In both muscle and liver there is interconversion between the monomer glucose and the polymer glycogen (Table 2.6). This has the potential to be a futile cycle wasting energy if the interconversion occurred continuously; thus it is controlled to meet the body's glucose requirements at a particular time.

Hormonal Control of Glycogen Metabolism

The control which operates is via different enzymes catalysing the synthesis and breakdown (degradation) of glycogen. The activity of these enzymes is controlled such that only one is active at any one time and thus the pathway can proceed in only one direction - either towards glycogen synthesis OR towards glycogen breakdown and mobilisation of free glucose.

The control is exerted by hormones acting to control the activity of the key enzymes. There are some differences in the hormone action in liver and muscle (Table 2.7).

Utilization of Glucose in the Fed and Fasting States

Glucose Utilization after a Meal

A high circulating glucose concentration is present after a meal. Carbohydrate is digested and the glucose absorbed into the blood stream. **Insulin** is secreted in response to elevated blood glucose level.

Insulin:

- Stimulates uptake of glucose into both muscle and liver
- Stimulates increased glycogen synthesis in both muscle and liver

This is achieved by activation of key enzymes, involved in glycogen synthesis.

The amount of glycogen which can be stored in these two tissues is limited and once the stores are saturated, excess glucose will be diverted to the synthesis of fats.

Maintenance of Blood Glucose between Meals

When there is no dietary glucose intake (between meals), circulating glucose concentration must be maintained. The pancreas secretes more glucagon and less insulin.

The Glucagon

- Stops liver glycogen synthesis (by deactivating the synthesis enzymes)
- Increases liver glycogen breakdown (by activating the degradation enzymes)
- Stimulates gluconeogenesis in the liver to further increase the circulating blood glucose concentration

These mechanisms maintain an appropriate circulating blood glucose to supply tissues such as the brain which are major glucose consumers but do not store glycogen.

Table 2.6: Glycogen stores

	Organ mass	*Glycogen (g/kg tissue)*	*Total glucose*
Liver	1.6 kg	65	~100 g
Muscle	28 kg	14	~400 g

Table 2.7: Differences in hormones action in liver and muscle

Hormone	*Source*	*Target tissue*	*Action*
Glucagon	Pancreas	Liver	Stimulates glycogen breakdown
Adrenaline	Adrenals	Muscle	Stimulates glycogen breakdown
Insulin	Pancreas	Liver and Muscle	Stimulates glycogen synthesis

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Supply of Glucose to Exercising Muscle

Increasing muscle activity requires adequate fuel supply for ATP synthesis by muscle. When muscle activity is anticipated, the adrenal glands secrete adrenaline.

Adrenaline increases muscle glycogen degradation (by activating the breakdown enzymes and de-activating the synthesis enzymes).

When muscle activity ceases, adrenaline secretion is switched off. When glucose becomes available again after a meal glycogen stores in muscle are replenished. Glucose can only be supplied to muscle cells either by utilizing stored muscle glycogen or supply from the liver via the bloodstream. Muscle does not carry out gluconeogenesis.

Glycogen Metabolism in Liver and Muscle

The energy yield from the hydrolysis of stored glycogen and the subsequent oxidation of the released glucose is the same in muscle and liver.

When glycogen is hydrolysed, the product is glucose 1-phosphate. This is easily converted to glucose 6-phosphate (these are molecules with the phosphate group attached to different carbon atoms on the glucose). Glucose 6-phosphate is the first product in the glycolysis pathway and its formation from glucose requires the expenditure of 1 ATP molecule/glucose.

As glucose 6-phosphate is formed directly from glycogen hydrolysis, glucose that is derived from glycogen and enters the glycolysis pathway (rather than starting as monomeric glucose) yields a nett production of 3 ATP/glucose rather than just 2. This is a 50% increase in yield.

Role of Glucose 6-phosphatase

Muscle and liver have different metabolic needs. Liver supplies other organs with glucose so must be able to export glucose released from glycogen hydrolysis. Muscle is a major consumer of glucose and thus does not export glucose.

Glucose 6-phosphate formed as described in the previous section is highly polar and cannot cross the cell's cytoplasmic membrane. To leave the cell it must be converted to glucose. This reaction is catalyzed by an enzyme, **glucose 6-phosphatase.**

Glucose-6-phosphate ——*glucose 6-phosphatase*——→ glucose+phosphate

Liver possesses this enzyme, so glucose released from liver glycogen can be exported to other tissues.

It is very important to be aware that muscle does not possess glucose 6-phosphatase so it does not export glucose released from its glycogen stores, but rather uses it as a fuel to power muscle contraction.

Conversion of Excess Glucose to Fat

Sustained high glucose intake in the diet leads to increased fat synthesis.

If glucose intake continues after muscle and liver glycogen stores are saturated, the glucose is not excreted or wasted. It is converted to a fuel storage form which has an unlimited capacity i.e. triglycerides stored in adipose tissue.

Glucose is converted to pyruvate by glycolysis. The pyruvate is converted to acetyl CoA, which is the starting material for the synthesis of fatty acids. This synthesis occurs in the liver followed by conversion of the fatty acids to triglycerides (also in the liver) and then transport to adipose tissue for storage. Triglycerides (fat) form the major energy store in the body. The mechanism of fatty acid synthesis will be discussed under the heading of fat metabolism.

Summary of Carbohydrate Metabolism

The pathways used in carbohydrate metabolism are shown in Figure 2.5.

MALNUTRITION

Not getting enough **carbohydrates** can cause a lack of calories (**malnutrition**), or Getting too many carbohydrates can lead to an increase in total calories, causing obesity.

Deficiencies

Marasmus

Marasmus is the most common form of Protein Energy Malnutrition (PEM) you will observe among children below 1 year of age. Marasmus is caused when the mother suddenly stops breast feeding the child and instead feeds him on starchy foods like sago, potato, etc. This results in providing insufficient energy and protein to the child leading to marasmus. Lack of these important nutrients, results in marasmus. A marasmic child can be recognized by following clinical features:

- *Muscle wasting:* Due to the inadequacy of energy and protein in the diet, the muscles get loose and skin becomes wrinkled. The child looks very'thin and weak.
- *Irritability:* The child becomes extremely weak and becomes irritable. The child cries continuously. His cries can be barely heard due to extreme weakness.

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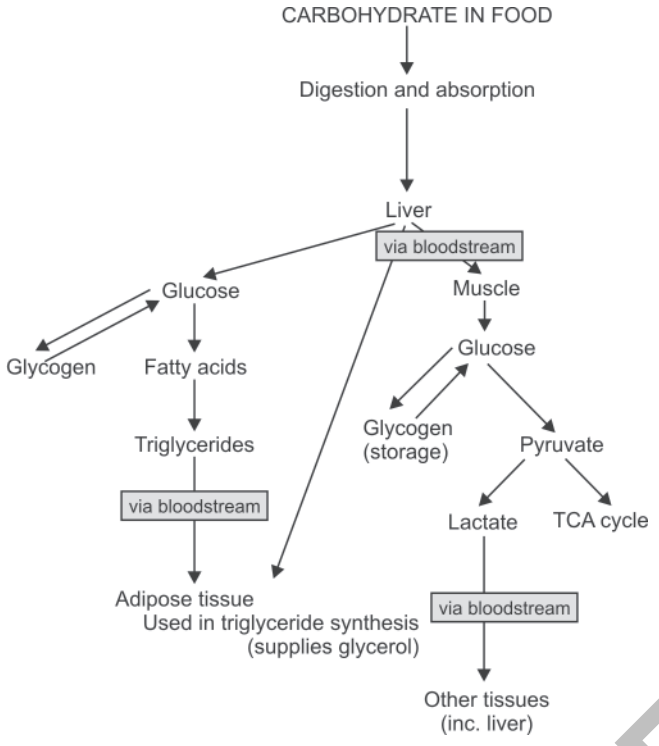

Fig. 2.5: Summary of carbohydrate metabolism

- *Growth failure:* Deficiency of energy and protein cannot meet the in creased requirements of the growing body the child. Hence, the child fails to grow and thus is smaller than the healthy children of his age.
- *Diarrhea:* A marasmic child has frequent watery stools (diarrhoea). Consequently, the child gets dehydrated.

Hypoglycemia and Ketosis

Testing for Hypoglycemia

"Many more people believe they have hypoglycemia than actually test positive when given blood-sugar tests...

Dr. Callaway suspects that people can experience pharmacological effects from sugar, even if blood-sugar levels remain within the normal range. People's responses to fluctuating blood-sugar levels vary greatly. Some people experience symptoms when blood-sugar levels are well within the normal range, while others report no symptoms even when blood-sugar levels have dropped substantially. It is likely that each person has a unique blood-sugar range, with fatigue or mood changes occurring when levels fluctuate above or below these levels.

Symptoms

Functional Hypoglycemia (FH)

"Symptoms of FH are episodic and have a direct relationship to the time and type of meal that was last eaten. Symptoms include fatigue, weakness in legs, swollen feet, tightness in chest, constant hunger, eyeache, migraine, pains in various parts of the body, nervous habits, mental disturbances, and insomnia. Rapid fluctuations in blood sugar level give rise to many bizarre symptoms that may suggest mental disorders."

Snacking

"A hypoglycemic child often needs to eat every hour or two during school hours, as well as at home, to avoid low blood sugar reactions, which cause recurrent fatigue, irritability, tension, hyperactivity and aggression. One clue in spotting hypoglycemia is the way young children ask for food. Do they request food, or demand it? The latter suggests low blood sugar. It can happen on and off all day, but it is most apt to occur between 10:30 and 11:30 [AM] and again between 15:00 [3:00 PM] and 16:00 [4:00 PM]. Some children are 'impossible' when they awaken in the morning until they eat. Their brains may need glucose (sugar)."

Other Symptoms

"Other symptoms of hypoglycemia include an inability to **concentrate, mood swings, anxiety, depression** and being more emotional than usual. **Asthma, fatigue, headache, nervousness, insomnia, irritability, restlessness, poor memory** and **indecisiveness** are also frequently reported."

Causes

The two most significant factors contributing to hypoglycemia are diet and emotional stress.

A Low Carbohydrate Diet May Cause Low Mood

Low-Carb Risks

In the 1980's and 1990's, dieters flocked to low-fat diets and products. But it became evident that eating fat did not necessarily cause body fat-in fact, instead of viewing fat as the enemy, awareness that there are "good fats"

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(such as omega-3 essential fatty acids) starting growing in the late 1990's. And during this time Americans were not losing weight. Diabetes has run rampant and it's estimated that two-thirds of the U.S. population is overweight.

Enter the low-carbohydrate craze. Support for the Atkins concept began to pick up steam in recent years as new research suggested that a carbohydrate-controlled approach to dieting was more effective at causing weight loss than low-fat diets. Now there are many low-carb diet programs to compete with Atkins, each with variations on caloric consumption and maintenance, such as South Beach, Sugar Busters, No-Grain, Protein Power and many more.

As with any diet, there are critics that point to potential problems that could result from favoring a particular nutritional source for caloric intake over another. In response to the low-carb diet craze, the American Dietetic Association and the American Heart Association (AHA) have warned of serious health risks, especially to those with heart disease, high cholesterol, high blood pressure or type 2 diabetes. The AHA has also expressed concern that people who stay on low-carb diets for extended periods of time may not be getting enough vitamins and minerals and be at risk for osteoporosis and disorders of the kidney and liver.

A low-carbohydrate diet starts to work when a reduced carb intake causes the body to begin burning glycogen (stored carbohydrates) for energy. When your body burns glycogen, water is released and you lose weight. However, a heavily carb-restricted diet can also force the body to burn fat. This is a metabolic state called ketosis, in which by-products called ketones build up in the bloodstream. These are removed by the kidney and eliminated. According to the Mayo Clinic, ketosis can cause the desirable effect of physically removing body fat, but muscle tissue can also be lost in the process. And an abundance of ketones can cause symptoms of nausea, fatigue and bad breath, or worse, cause kidney failure and gout.

Many controlled-carb dieters follow a basic, but flawed, philosophy that they can load up on fat and protein — an approach that worries some health experts. The AHA continues to endorse a low-fat component as part of its dietary recommendations because of the risks like heart disease and high blood pressure that are associated with over consumption of saturated fats. And excessive protein intake can put a strain on the kidneys, possibly leading to kidney disease in extreme cases, according to the Cleveland Clinic. High protein diets have also been shown to cause people to excrete more calcium and other minerals than normal, creating a potential susceptibility for osteoporosis and kidney stones.

There is also concern that the low intake of fruits, vegetables and whole grains typically associated with a low-carb diet could create a deficiency of important vitamins, minerals, fiber and antioxidants. The Atkins web site encourages everyone, not just dieters, to supplement their diet with vitamins and minerals, particularly B vitamins, plus antioxidants like vitamins A, C and E, selenium, glutathione, COQ10 and bio-flavonoids. New versions of Wyeth's Centrum® and Bayer's One-A-Day® Vitamins have recently been introduced to support low-carb dieters with added or increased levels of certain nutrients that can help break down fats and proteins into energy, or nutrients that may be lacking in a low-carb regimen.

Low-carb dieters are susceptible to *mood* swings, according to MIT researchers. The issue involves serotonin, the "feel good" brain chemical that elevates mood, suppresses appetite and acts as a natural tranquilizer.

In news released earlier this year, the Massachusetts Institute of Technology Clinical Research Center reported that a lack of dietary carbohydrates causes the brain to stop regulating serotonin. Researchers discovered that serotonin is naturally produced only after consumption of carbohydrates in the form of sweets and starches.

In the 1970's, MIT professor Richard Wurtman and colleagues first showed that eating carbohydrates raises brain serotonin levels. Since then, additional studies at MIT, including those by Dr. Judith J. Wurtman, have explored the relationship between carbohydrates in the brain and their connection to mood and weight loss. Dr. Wurtman states in a February MIT press release that "When serotonin is made and becomes active in your brain, its effect on your appetite is to make you feel full before your stomach is stuffed and stretched." The researchers explain that people may still feel hungry after eating a large steak-their stomachs may be full but their brains may not be producing enough serotonin to shut off their appetites.

It's not unusual for people who are changing their eating patterns or embarking on any kind of diet to experience episodes of irritability. However, according to *Psychology Today*, many who are trying testing low-carbohydrate regimens are reporting unusually high feelings of anger, tension and depression. Dr. Wurtman claims that it's a very well documented response. She calls it the "Atkins attitude". Dieters who have eliminated or reduced dietary carbohydrates may find that their low mood is combined with irritability if the diet is heavy on

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protein, and/or combined with a lack of energy if the diet is heavy on saturated fats.

Are certain people more susceptible to low mood? According to Wurtman, some people are "carbohydrate cravers" — they tend to experience a change in their mood, usually in the late afternoon or early evening — and they need to eat a certain amount of carbohydrates to keep their moods steady. And although both men and women can experience low mood when cutting carbs, women are more likely to feel the effects because they are known to have typically lower levels of serotonin in their brains than men.

Because of her research, Wurtman believes that low-carb diets may be dangerous for individuals who are already struggling with depression or bipolar disorder. In an Oxford study involving women, researchers did find that those who had a predisposition to mood disorders exhibited a measurable drop in mood when following a low-carb regimen, but reported that other subjects on the diet did not seem to be impacted.

Of course, the pharmaceutical solution to depression and mood disorders is the use of antidepressant drugs. These are designed to help regulate mood by keeping serotonin production active in the brain for extended periods, accomplished via an unnatural mechanism that prevents the cellular reuptake of the brain chemical (which is why most of these medications are known as "SSRIs", for "selective serotonin reuptake inhibitors"). St. Johns Wort is a popular herbal solution that also mimics the unnatural mechanism of SSRIs because the herb's active ingredient, hypericin, increases circulating levels of serotonin by selectively preventing its cellular reuptake.

There are natural alternatives that can assist in regulating mood by playing a similar role as carbohydrates in serotonin production. Low-carb dieters who are experiencing low mood might consider taking specific vitamins that are known to help boost serotonin naturally. Important nutrients like selenium, several B vitamins (thiamine, riboflavin, pyridoxine, folic acid, biotin, etc.), and vitamin D have been shown to help increase the enzyme supply that converts certain amino acids into serotonin.

Levity Mood Elevating Formula was designed to include the six most important vitamins and minerals that can play a role in boosting serotonin. It can be a very effective means of warding off the mood swings and irritability that can accompany a low carb diet — safely and inexpensively!

Over-consumption

Obesity is an excess proportion of total body fat. A person is considered obese when his or her weight is 20% or more above normal weight. The most common measure of obesity is the body mass index. Obesity is common in North America, Australia and Europe. Indians are genetically susceptible to weight accumulation especially around the waist and 5 percent of the population suffers due to obesity in India.

According to the study : The states which ranks the rate of obesity are :

Punjab	:	30.3% Males,	37.5% Females
Kerala	:	24.3% Males,	34 % Females
Goa	:	20.8% Males,	27 % Females

In 1997, it stated that the incidence of obesity in India is 7-9 % , though the number of obesity is less as compared to America and the other countries, but it is also significant due to the sheer size of the population in India. With such large numbers, India has been requested to join the International Congress on Obesity (ICO) for further study on the risk and management of the overweight in a developing economy. With this, India would be one of the first nations from the developing countries in Asia to be put on the obesity map.

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