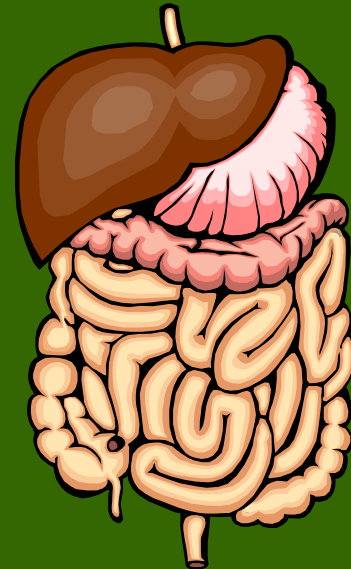


Irritable Bowel Syndrome

Mechanisms and Dietary Management

Janice M. Joneja, Ph.D., RD



Irritable Bowel Syndrome

- Irritable bowel syndrome (IBS) tends to be an umbrella term for a variety of minor bowel disturbances of unknown origin
- Sometimes called:
 - “irritable colon”
 - “spastic colon”

Symptoms of IBS

- Symptoms include:
 - Change in bowel habit
 - often alternating constipation and diarrhoea
 - Abdominal bloating and distension
 - Sometimes abdominal pain, frequently relieved by defaecation
 - Feeling of incomplete defaecation

IBS Characteristics

- There is usually no sign of structural damage to the wall of the intestine (frequently indicated by blood in the stool)
- Weight loss or nighttime fever are not experienced
- A diagnosis of irritable bowel syndrome is made when all organic disease has been ruled out by appropriate medical tests
- Some physicians use the Manning Criteria or the Rome II questionnaires for diagnosis

Initial Triggers of IBS

- Infection in the digestive tract:
 - Viruses
 - Bacteria
 - Parasites (amoeba; intestinal worms)
- Pathology in the digestive tract
 - Inflammatory bowel disease
 - Coeliac disease
- Surgical procedures in the digestive tract

Triggers of IBS (continued)

- Stress:
 - Stress hormones are released
 - Neuropeptides may trigger the release of inflammatory chemicals
- Hormone fluctuations:
 - Menstrual cycle
 - Pregnancy
 - Thyroid

Triggers of IBS (continued)

- Change in types of micro-organisms in the large intestine due to:
 - Oral antibiotics
 - Other oral medications
 - Change in substrate (ie type of food passing into the bowel)
- Alteration in microbial flora results in:
 - Different products resulting from the action of micro-organisms on undigested food material:
 - Gases
 - Organic acids
 - Others

Mechanisms Responsible for Symptoms

- Key factors in IBS resulting in symptoms include:
 - Inflammation
 - Resulting from release of inflammatory mediators
 - Increased sensitivity to pain
 - Neuropeptides (tachykinins) generated by the central nervous system interact with neurokinin receptors on the spinal cord
 - May also result from a response to inflammatory mediators (e.g. histamine)

Mechanisms Responsible for Symptoms

(continued)

– Motility dysfunction

- Resulting from changes in autonomic nervous system signals
- Resulting from products of microbial fermentation

– Fermentation

- Of undigested food in the large bowel
- As a consequence of abnormal motility
- As a consequence of altered microbial flora

Inflammation



- Inflammation is rarely visible in tissue viewed under the microscope in IBS
- However, there is research evidence of the presence of inflammatory activity in IBS, based on the presence of chemicals that indicate that inflammation is occurring

Causes of Inflammation in IBS

- Infection: bacterial; viral; parasitic
 - Infective microorganism may have been successfully eradicated, but inflammation of the intestinal tissues may persist, especially as food is continually passing through
- Autoimmune processes
- Food protein enteropathy
 - Cow's milk protein enteropathy
 - Soy protein enteropathy
 - Gluten-sensitive enteropathy (coeliac disease)
- Food allergy and food intolerance

Stress, Inflammation and IBS

- Neuropeptides are released into the digestive tract
- Vasoactive intestinal peptide (VIP), Substance P, somatostatin, and others can cause degranulation of mast cells
- Mast cells exist in large numbers in the digestive tract, and are intimately associated with nerves
- Mast cells store inflammatory mediators designed to protect the body from invasion
- Release of inflammatory mediators triggers inflammation

Altered Motility of the G.I. Tract

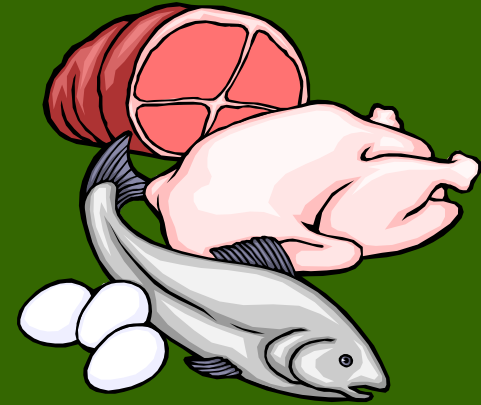
- Altered speed of food passing through the G.I. tract can result in disturbance of the normal process of digestion and absorption of nutrients:
 - Increased speed in the small intestine results in incomplete breakdown of food components in the small intestine
 - Food is not in contact with digestive enzymes long enough for molecules to be broken down to the size and state required for absorption

Increased Motility in the Small Intestine

- Incomplete breakdown of fats and oils by pancreatic lipases
 - Results in steatorrhoea (fatty stool)
- Incomplete breakdown of carbohydrates
 - Results in complex sugars passing into the large bowel
 - Damage to cells lining the digestive tract results in deficiency of enzymes that digest sugars (lactase; sucrase; isomaltase)
 - Undigested sugars pass into the large bowel



Increased Motility in the Small Intestine



- Incomplete protein digestion
 - Evidence of passage of fairly large polypeptides into circulation, especially if digestive tract lining is damaged by inflammatory processes
 - Undigested and unabsorbed proteins pass into large bowel
 - They are acted on by micro-organisms, resulting in production of organic acids, gases

Altered Motility in the Large Bowel

- Increased speed through the large bowel results in watery stool as fluid is not resorbed
 - Causes diarrhoea
- Results in net fluid loss, with accompanying loss of electrolytes
- Results in dehydration and electrolyte imbalance
- Requires rehydration and restoration of electrolytes



Altered Motility in the Large Bowel (continued)

- Decreased speed of movement through results in:
 - Increased microbial metabolism of food materials, resulting in production of:
 - Gases (hydrogen, carbon dioxide, methane, and others)
 - Organic acids
 - Other products of microbial activity
 - Increased resorption of fluid
 - Results in dry, hard stool
 - Constipation



Fermentation

- All food materials not absorbed through the lining of the small intestine pass into the large bowel
- Millions of bacteria colonise the organ
- Perform “end-stage digestion”
- Products of microbial activity can be important nutrients:
 - some B vitamins (pantothenic acid; biotin)
 - vitamin K

Colonic Fermentation

- Plant foods contain two broad classes of carbohydrates
 1. Free sugars (glucose, fructose, sucrose)
 2. Polysaccharides
- Free sugars are found mainly in fruit and vegetables and are rapidly absorbed from the small intestine in healthy humans
- Sugar is also present in milk, in the form of lactose

Causes of Intestinal Symptoms: Carbohydrates

- Non-digested carbohydrates pass into the large intestine causing:
 - Osmotic imbalance: causes excess fluid in the lumen of the large bowel resulting in loose stool
 - Increased bacterial fermentation resulting in production of:
 - organic acids (acetic, lactic, butyric, propionic)
 - increase osmotic imbalance
 - gases such as carbon dioxide and hydrogen
 - cause bloating and flatulence

Causes of Intestinal Symptoms:

Carbohydrates (continued)

- Increased bulk results in increased stool volume
- Increased fluid and acid environment stimulate intestinal motility and accelerate intestinal transit time.
- Increased speed of intestinal transit results in:
 - loose stool since fluid is not absorbed from food
 - Incomplete absorption of fat

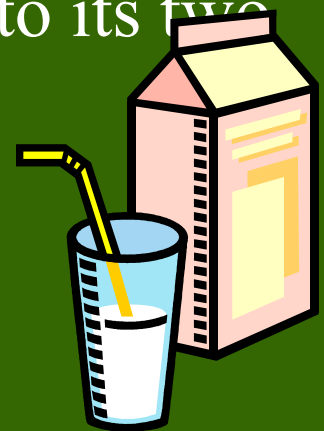
Symptoms of Excessive Fermentation of Carbohydrate

- Patients complain of abdominal fullness, bloating, and cramping pain, sometimes within 5-30 minutes, sometimes several hours after ingesting carbohydrate
- Watery diarrhoea occurs from 5 minutes to 5 hours after ingestion
- Excoriation of perianal skin and buttocks due to acid (pH less than 6) stool in children
 - Adults do not consistently experience such a low stool pH

Examples of Fermentation of Undigested Sugars:

Lactose Intolerance

- Milk sugar, lactose, is digested by lactase enzyme produced in the cells lining the digestive tract
- Lactose is a disaccharide (double sugar) which cannot be absorbed through the lining of the digestive tract until it is broken down into its two single sugars (monosaccharides):
 - Glucose
 - Galactose
- Lack of lactase reserves makes lactose particularly vulnerable to maldigestion
- There are three main types of lactose intolerance:



1. Congenital Alactasia

Primary lactase deficiency

- Very rare
- Due to inherited autosomal recessive gene
- Stools are loose from first days of life
- Condition is permanent



2. Idiopathic Lactase deficiency:

Natural attrition after infancy

- Wide variation in prevalence among different racial groups
- Most races except Northern Europeans have a 50-100% incidence
- 80% of the world's adult population have some degree of lactose intolerance
- Intolerance usually appears in adolescence
- There is normal lactase production in childhood

3. Secondary Lactase Deficiency

Temporary lactose intolerance

- Results from damage to the lactase-producing cells in the lining of the small intestine
- Common in childhood intestinal infections
- Often accompanies coeliac disease (*gluten-sensitive enteropathy; coeliac sprue*)
- Lactase is depressed earlier than the other disaccharides in intestinal injury
- Lactase returns to normal levels after cell injury resolves
- Lactase is the last disaccharidase to return to normal levels after cell damage

Secondary Lactase Deficiency: Characteristics

- Lactase deficiency is by far the most common form of carbohydrate intolerance in childhood and may result from:
 - viral or bacterial enteritis
 - gastrointestinal surgery
 - extensive small intestine resection
 - cow's milk protein allergy
 - *Giardiasis*
 - protein-calorie malnutrition

Sucrase-Isomaltase Deficiency

- Primary deficiency is rare: it is inherited as an autosomal recessive gene
- Greenland and Canadian Inuit have an unusual incidence of 10% of the population
- Appears when sucrose enters the child's diet, usually as fruit or fruit juice
- Severity of symptoms depends on the quantity of sucrose in the diet
- In practice sucrose must be avoided

Fermentation of Undigested Polysaccharides: Starch and Fibre

- Plant polysaccharides can be separated into two broad categories:
 1. Starch
 2. Non-starch

Starch is:

- a storage polysaccharide
- the major carbohydrate of cereal grains and potatoes

Non-starch polysaccharides are:

- structural components of the plant cell wall
- considered the dietary fiber of foods

Starch

- Starch is found in many of the world's staple foods such as cereals, legumes, potatoes, and bananas
- Usually starch in foods is readily digested in the small intestine by enzymes produced in the pancreas
- The products of digestion of starch are absorbed into the body
- The process can be speeded up by cooking: starch is gelatinized and rendered more available to the enzymes

Resistant and Non-resistant Starch

- In the 1980s, research showed that a significant portion of dietary starch may resist digestion and pass intact into the colon
- Food processing can render some starch partly resistant to enzymatic digestion
- This was classified as *resistant starch*
- Starch that is readily digested was called *non-resistant starch*

Resistant and Non-resistant Starch

(continued)

- All fibre and starch entering the large intestine is a suitable substrate for bacterial fermentation
- Gas, bloating, pain may result from excessive microbial fermentation
- Organic acids may be a source of irritation of mucosal tissues
- Microbial fermentation of resistant starch and fibre can produce volatile fatty acids which are absorbed into the body from the colon, and may help in protecting against disease such as colon cancer

Resistant and Non-resistant Starch

(continued)

Cooking and processing can affect the digestibility of starch:

- The quantity of some types of resistant starch in foods is critically dependent on processing conditions such as heating, cooling, freezing, or drying:
 - Starch from cereal products and freshly cooked potato is well digested
 - Cooled, cooked potato is less well digested than freshly cooked potato

Resistant and Non-resistant Starch

(continued)

- This may also occur with other starches such as rice and pasta
- Up to 89% of the starch from raw banana escapes digestion in the small intestine
- A high percentage of other raw fruits and vegetables may also be resistant to digestion in the small intestine and can provide a rich source of substrate for microbial fermentation

Comparison of Dietary Starch

a) Fed

b) Recovered after digestion in the small intestine

Food	Starch Fed (grams)	Starch Recovered (grams)	Percentage Starch Recovered (%)
White bread	62	1.6	3
Oats	58	1.2	2
Cornflakes	74	3.7	5
Banana (raw)	19	17.2	89
Potato			
freshly cooked	45	4.5	3
cooled	47	5.8	12
reheated	47	3.6	8

Englyst and Kingman 1994

Factors Affecting Amount of Starch in the Colon



- Cooking
 - Disrupts starch granules
 - Facilitates digestion by enzymes in saliva and the small intestine
 - When foods with a high level of resistant starch are eaten raw, more undigested starch passes into the colon
 - e.g. Banana
 - Retrograded starch increases on cooling: eat foods with high level of resistant starch when it is hot

Factors Affecting Amount of Starch in the Colon (continued)

- Chewing
 - Amylase (ptyalin) in saliva is first enzyme to start process of starch digestion
 - The more the food is chewed, the greater the exposure of the starch to enzymes in the mouth and the small intestine
- Speed of transit of food
 - The faster the food transits the small intestine, the less exposure to enzymes
 - High fat slows transit
 - High fluid (water with the meal) speeds the transit

Dietary Fiber

“The sum of the non-starch polysaccharides in food”

- Not affected by food processing
- Includes a mixture of polymers such as cellulose, pectin, and hemicellulose
- Resistant to human digestive enzymes and escape breakdown in the small intestine
- May be classified as
 - “soluble” (becomes gelatinized in water, especially when heated)
 - “insoluble” (remains unchanged in water)

Dietary Fiber (continued)

- Fibre resists digestion
- All types of fibre pass completely undigested through the small intestine and into the colon
- All carbohydrates that are not digested and absorbed from the small intestine move into the large intestine where they are fermented by micro-organisms

Accessibility of Starch and Fibre in the Colon

- Physical accessibility
 - Cell walls of plant cells entrap starch
 - Prevents its swelling and dispersion
 - Delays or prevents digestion by enzymes
 - Includes whole grains, nuts, seeds:
 - vegetables with “skins”: sweet corn, peas, beans
 - partly milled grains and seeds: “whole grain” breads and cereals
 - If the rigid structures of the plant are physically removed, the starch is exposed to the action of enzymes in the small intestine

Role of Food in IBS

- Food does not cause IBS
- Food passing through “damaged organ” continues or exacerbates the condition
- Food interacts with gastrointestinal tissues in several ways:
 - Immunologically
 - Physiologically
 - Biochemically



Dietary Management of IBS

- Symptoms seem to be based on TWO principal mechanisms:
 1. Inflammation
 2. Fermentation
- Dietary management strategies are designed to reduce the effects of these