

The small intestine and irritable bowel syndrome (IBS): a batch process model

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Abstract

Faults in a batch process model of the small intestine create the symptoms of all types of irritable bowel syndrome. The model has three sequential processing sections corresponding to the natural divisions of the intestine. A brain controller divided into four sub-controllers, each with a unique neurotransmitter, governs it.

Each section has a sub-controller to manage transport. Sensors in the walls of the intestine provide input and output goes to the muscles lining the walls of the intestine. The output controls the speed of food soup, moves it in both directions, mixes it, controls absorption, and transfers it to the next section at the correct speed (slow).

The fourth sub-controller manages the addition of bile. It obtains input from the first section of the process via the signaling hormone Cholecystokinin and sends output to the muscle that empties the gall bladder. The correct amounts of bile are then added to the first section.

The sub-controllers produce output only when input is received. When output is missing, the enteric nervous system (division of the autonomic nervous system) applies a 'default' condition. The 'default' condition is normally active only when there is no food in the intestine. If food is present and a transport sub-controller fails to provide output, then the 'default' condition moves the food soup to the end of that section. The movement is in one direction only (forward), at a speed dependent on the amount & type of food eaten, and environmental factors affecting the autonomic nervous system.

When the 'default' transport speed is 'too fast', then the symptoms of irritable bowel syndrome are produced.

Introduction

This hypothesis evolved from observation of a case of IBS-D over a period of decades. Medical tests and examinations could find nothing wrong with the patient's digestive system. Dietary trials were carried out. The progressive worsening of the disorder was consistent with the gradual loss of neuro-transmitter(s) in the brain. Cases of IBS-C and IBS-A also provided evidence.

Explanations

Here you will find background information necessary to understand the hypothesis:

Autonomic nervous system

This is the interconnected networks of nerves that control automated systems in the human body. Activity levels in these networks increase when adrenal hormones are released on awakening, and when stress occurs. After the morning peak of activity, a gradual relaxation occurs, that is completed overnight.

Bicarbonate

This is made by the pancreas and walls of the small intestine. It neutralizes stomach and food acids so that the pH of food soup is neutral to slightly alkaline. This is necessary because human digestive enzymes do not work in acid conditions.

Bile

This is made in the liver, stored in the gall bladder, and secreted into the duodenum. Bile emulsifies fats into droplets a few nanometers in diameter that can then be digested by lipase

enzymes. Bile salts can be reabsorbed in the ileum and returned to the gall bladder.

Borborygmii

These are loud gurgling sounds that come from the small intestine. They are common in some types of IBS.

CCK (Cholecystokinin)

When food soup is pumped from the stomach into the duodenum, CCK, a peptide hormone [3], is released into the bloodstream. CCK stimulates the release of bile from the gall bladder, and lipase enzymes from the pancreas, into the duodenum. It also inhibits gastric emptying.

Enteric nervous system

The network of nerves controlling automated functions in the digestive system. It is a division of the autonomic nervous system.

Glucose-dependent insulinotropic peptide (GIP)

This hormone is released from the duodenum when food soup is pumped in from the stomach. It stimulates the release of insulin from the pancreas and inhibits gastric emptying.

Gall Bladder

A storage organ for bile produced in the liver. It is emptied by a muscle controlled by the brain that delivers bile into the duodenum.

Irritable bowel syndrome

IBS is a common digestive disorder that has three widely recognized types; IBS-C (constipation predominant), IBS-D (diarrhoea predominant), and IBS-A (alternating constipation and diarrhoea). The

author has identified a fourth type and has labeled it IBS-B (bile deficient). Rates of occurrence have been measured at 5-25% in the general population. The financial burden for the USA has been estimated at tens of billions of dollars annually [1,2].

Insulin

A hormone made in the pancreas that moves glucose into cells.

Large intestine (colon)

This 2 metre muscular tube accepts food soup from the small intestine and prepares it for release via the anus. The soup is processed with the assistance of bacteria.

Liver

An organ that manufactures bile salts from cholesterol and stores them in the gall bladder.

Neuro-transmitters

These are chemicals that the nervous system uses to transmit signals between nerve cells. There are a considerable number of them, and most are unknown to science.

Pancreas

An organ that, when stimulated by the hormones CCK & Secretin, delivers protease enzymes, lipase enzymes and bicarbonate into the duodenum. It also manufactures the hormone insulin.

Secretin

This hormone is released from the duodenum when food is pumped in from the stomach. It stimulates the pancreas to release protease enzymes and bicarbonate into the duodenum. It also inhibits gastric emptying.

Small intestine

This is a narrow flexible muscular tube that accepts acidified food soup from the stomach, digests it, and moves it into the large intestine. It is about 6-9 m long and divided into three physically different sections;

1. *Duodenum* is first. It is about 25 centimetres.
2. *Jejunum* is second. Its length can be between 2 and 3 metres.
3. *Ileum* is last. Its length can be between 4 and 6 metres.

The Hypothesis

Digestion in the small intestine is a batch process, with sequential sections corresponding to the three natural divisions of the intestine; duodenum, jejunum and ileum. The process is governed by a brain controller divided into four sub-controllers, each equipped with a unique neuro-transmitter.

Control faults in this process cause the disorder Irritable Bowel Syndrome (IBS).

The batch process

The duodenum is the first section of the process. It accepts acidified food soup from the stomach and releases CCK, GIP and Secretin into the bloodstream. Bile, enzymes & bicarbonate are mixed into the soup, so that acids are neutralized, and fats are emulsified. The soup is then moved slowly to the next section.

The jejunum is the second section of the process. It accepts slowly moving food soup from the duodenum. The

soup is mixed so that digested nutrients are efficiently absorbed at the correct rate. It is then moved slowly to the next section.

The ileum is the third section of the process. It accepts slowly moving food soup from the jejunum. The soup is mixed so that bile salts and enzymes can be efficiently absorbed and returned to the gall bladder and pancreas. The food soup is dehydrated and readied for release into the colon.

Process control

There are two levels of control;

The primary level is a brain controller. This is divided into four sub-controllers, each with a unique neurotransmitter. Three sub-controllers manage transport and mixing, and the fourth controls the addition of bile. These sub-controllers produce output only when input is received.

The transport sub-controllers accept input from sensors in the walls of the intestine. These sensors respond to the amount of food soup and the amount and type of fibre present in the soup. Cereal (except corn) and whole legume fibers cause high stimulation.

Output from the sub-controllers controls transport and mixing in the small intestine. Correct control happens regardless of the variable input caused by different foods. The food soup is moved backwards and forwards to mix it, the rate of absorption is controlled, and it is then transferred to the next section at the correct speed (slow).

The fourth sub-controller adds the correct amount of bile to the food soup. Cells in the wall of the duodenum release a peptide hormone, Cholecystokinin (CCK), into the bloodstream, when food is pumped in from the stomach. One of the tasks of this hormone is to travel to the brain where the fourth sub-controller detects it, and outputs a signal to the muscle that empties the gall bladder.

A secondary (default) level of transport control is provided by the enteric nervous system. This control system takes over when brain sub-controller outputs are missing. Normally this is when there is no food soup in the intestine. When food soup is present and output from the brain is missing, then the enteric nervous system moves the soup to the end of that section. The speed of this default transport control is dependent on;

- The amount and types of food fibre eaten.
- Food selection, food preparation, and the timing of eating.
- Environmental and behavioural factors that affect the autonomic nervous system.

There is no control of mixing or timing by the enteric nervous system, the transport is in one direction (forward) and the speed is determined by the above factors. When it is 'too fast', IBS symptoms occur.

The enteric nervous system also adds chemicals when it detects; the amount and types of fibre eaten, cooked proteins (meats, fish, & eggs), fruit acids (alpha hydroxy acids), dairy proteins, and some herbs & spices

(e.g. ginger). However the amount of bile added is usually insufficient and extra needs to be provided by the fourth sub-controller, in order to complete the digestion of fats.

Control Faults

1. One or more of the four neurotransmitters in the primary controller may be deficient or absent. This reduces or eliminates output from one or more of the sub-controllers.
2. A toxic insult may destroy intestinal sensors that secrete hormones, and provide input to the primary controller. This can reduce the amounts of bile, enzymes, and bicarbonate entering the intestine, and reduce or eliminate output from the transport sub-controller(s) of the affected part(s) of the intestine.
3. Surgical procedures may sever input nerves to the primary controller or output nerves from the primary controller to the intestine.
4. Misalignment of neck vertebrae may put pressure on nerves connecting the primary controller to the intestine.
5. In infancy, development of nerve connections from the brain to the intestine, may fail to be completed.
6. Any other fault that interrupts communication between the small intestine and the brain.

The IBS Barrier

This is created when food soup is present in the small intestine, and a section governed by the secondary controller precedes a section governed by the primary controller. When the

secondary controller moves food soup 'too fast' then the primary controller constricts the intestine to stop the fast flow. It is programmed only to accept soup that travels at the correct speed (slow). This Barrier causes the characteristic IBS symptoms of bloating and constipation.

The Barrier is caused by parts of the autonomic nervous system. Variation in the level of activity in this system causes variation in Barrier strength. It is strongest on arising in the morning when adrenal hormones are released to kick-start the metabolism. The symptoms of the Barrier are at their worst when breakfast is eaten. The Barrier increases in strength when stress releases adrenal hormones during the day. It can relax only after the autonomic nervous system returns to a low level of activity.

Diarrhoea

This symptom results when the terminating section of the process is under the control of the secondary controller. When food soup is moved too fast into the colon, it may contain high levels of acids, bile salts & enzymes, and may not have been conditioned correctly. The colon is automatically evacuated when this insult occurs.

The valve at the end of the small intestine is not under brain control. Its state is changed by the level of activity in the autonomic nervous system. When adrenal hormones are released on arising, the valve is easy to open, and a defective ileum immediately pushes its contents into the colon (the morning rush).

When stress releases adrenal hormones during the day, the valve is again easier to open. Overnight the valve becomes firmly closed when the autonomic nervous system relaxes. In severe cases of IBS-D & A, the ileum can push food soup through the valve at any time.

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The following sections explain what happens when neuro-transmitter levels are deficient in the four brain sub-controllers:

IBS-B (bile deficient IBS)

When the output from the fourth sub-controller is missing or deficient, then insufficient bile salts are added to the food soup. Undigested fats impair nutrient uptake in the jejunum, and reabsorption of chemicals in the ileum. Indigestion is followed by fast, loose, grey bowel movements containing fat (steatorrhea). The absence of the brown bile pigment stercobilin causes the grey colour, and when fat is present in the colon, the enteric nervous system automatically evacuates it. IBS-B may occur alone but usually it accompanies one of the other types of IBS. When it does, all IBS symptoms become severe.

IBS-C (constipation dominant IBS)

There are six forms of IBS-C;

1. *The duodenum sub-controller output is deficient or missing;* causes a Barrier to form at the start of the jejunum. When breakfast is eaten, immediate severe bloating may occur. Back-pressure in the duodenum keeps the valve from the gall bladder and pancreas closed, and insufficient

chemicals are added to the food soup.

2. Form 1 together with IBS-B.
3. *The jejunum sub-controller output is deficient or missing;* causes a Barrier to form at the start of the ileum. When breakfast is eaten, borborygmii may occur followed by hard to detect, slight to moderate bloating. Onset of these symptoms is delayed by a few minutes.
4. Form 3 together with IBS-B.
5. *The duodenum & jejunum sub-controller outputs are deficient or missing;* causes a Barrier to form at the start of the ileum. When breakfast is eaten, borborygmii may occur followed by hard to detect, slight to moderate bloating. Onset of these symptoms is immediate.
6. Form 5 together with IBS-B.

IBS-D (diarrhoea dominant IBS)

There are six forms of IBS-D;

1. *The ileum sub-controller output is deficient or missing;* when breakfast is eaten, borborygmii may begin when food soup reaches the ileum several hours later. Diarrhoea occurs immediately food soup reaches the end of the ileum or on arising next morning.
2. Form 1 together with IBS-B.
3. *The ileum & jejunum sub-controller outputs are deficient or missing;* When breakfast is eaten, borborygmii may begin when food soup reaches the jejunum a few minutes later. Diarrhoea occurs immediately food soup reaches the end of the ileum or on arising next morning.
4. Form 3 together with IBS-B.

5. *The ileum, jejunum & duodenum sub-controller outputs are deficient or missing*; when breakfast is eaten, borborygmii may begin immediately. Diarrhoea occurs as soon as food soup reaches the end of the ileum or on arising next morning.
6. Form 5 together with IBS-B.

IBS-A (alternating or mixed constipation and diarrhoea)

There are two forms of IBS-A;

1. *The duodenum & ileum sub-controller outputs are deficient or missing*; causes IBS-C plus IBS-D. Constipation & diarrhoea alternate irregularly. The state of the autonomic nervous system controls the alternation.
2. Form 1 together with IBS-B.

Summary

The 4 types and 15 forms of neurotransmitter deficient IBS created from combinations of defects in the four sub-controller outputs are presented in Table 1.

Other Causes of IBS

The part(s) of the intestine that are affected will not be precisely defined as they are when a neuro-transmitter is missing.

Damage to sensors in the intestinal walls will cause symptoms that may be quite different from IBS caused by other problems. If motor sensors are damaged, symptoms will be similar. If sensors that release CCK are damaged, IBS-B occurs. If sensors that release GIP are damaged, symptoms similar to type II diabetes may occur. If sensors that release Secretin are damaged,

the small intestine becomes acidic (as if the pancreas is malfunctioning), and diarrhoea occurs.

Current theories of IBS

Some are;

- Bacterial overgrowth
- Over-regulation by the brain of the small intestine.
- Food allergies.

None explain why there are multiple types and forms of IBS, with constipation or diarrhoea or both, and why borborygmii, bloating and cramping occur. This theory explains how all symptoms are created and points to under-regulation by the brain as the cause of IBS.

Importance of the theory

The theory explains;

- How the nervous system controls the digestion.
- The cause of the common digestive disorder Irritable Bowel Syndrome.
- How to heal IBS.

Evaluation of the hypothesis

Supporting evidence:

No apparent damage

Medical examinations of most IBS patients show no apparent damage to the small intestine. The problem is likely to be in its control systems.

Cereal & Legume Fibre

Diets that remove cereals and whole legumes [4] reduce symptoms. Stimulation by the fibre in these foods causes 'too fast' speeds when the secondary controller is regulating transport.

Stress

This aggravates IBS symptoms showing that the disorder is likely to involve the autonomic nervous system.

Difficulty digesting fats

This theory identifies five causes;

1. Those with IBS-C & A (severe bloating = defective duodenum sub-controller and resulting jejunum barrier) have large back-pressures in the duodenum that prevent the release of sufficient bile salts.
2. Continual diarrhoea can cause a total loss of bile and enzyme stores.
3. A defective gall bladder sub-controller, preventing the release of sufficient bile (IBS-B).
4. Damage to sensors in the duodenum that release CCK hormone (IBS-B).
5. Damage to sensors in the duodenum that release Secretin hormone. Now insufficient bicarbonate is delivered into the duodenum, the small intestine becomes acidic, and lipase enzymes do not work.

Diarrhoea

IBS-D & A subjects can have high levels of protease enzymes in their bowel movements. The enzymes attack the skin around the anus, causing irritation. They are not being removed by the ileum before food soup is discharged into the colon. This must be an ileum control problem.

The 'morning rush' is a characteristic symptom of IBS-A & D. A fast, loose bowel movement can occur soon after arising. This happens when the level of

adrenal hormones rises, and the valve at the end of the small intestine becomes easy to open. A defective ileum immediately pushes its entire contents prematurely into the colon. High levels of acids, enzymes & fats cause the enteric nervous system to evacuate the colon at speed.

Progressive onset

A case of IBS-D started to suffer symptoms as a teenager. Symptoms were intermittent until about age 40, when they started to increase in frequency. At age 45, they were present every day, and at age 55, there appeared to be little or no control of the small intestine left. This progression is symptomatic of the gradual loss of neuro-transmitter(s) in the brain.

Intestinal bloating

Severe IBS bloating starts on arising when adrenal hormones are released and breakfast is eaten. Stress during the day triggers it again. Overnight it can disappear. The autonomic nervous system is at a high level in the morning, high in response to stress and low overnight. It is likely to be causing the bloating.

The symptom of bloating displays two degrees. It is either severe, or slight to moderate and hard to detect. The duodenum is short (25 centimetres), so when the Barrier is at the start of the jejunum and the stomach continues to pump in food soup, bloating is severe. The jejunum is long (2 to 3 metres), so when the Barrier is at the start of the ileum, it causes only slight to moderate bloating that is hard to detect.

Intestinal cramping

- Cramping associated with bloating is caused by the secondary transport controller trying to move food soup through a Barrier created by the primary controller. The secondary controller pushes in one direction only (forward). The strength of the pushing depends on the type of food eaten, and the state of the autonomic nervous system.
- Cramping associated with loud borborygmii is the secondary controller moving food soup 'too fast' in the small intestine.
- Cramping followed immediately by diarrhoea, occurs when the secondary controller causes the ileum to move food soup prematurely into the colon. The soup contains acids, enzymes and/or fat, and the colon is then rapidly evacuated.

Borborygmii

These gurgling sounds occur when the secondary controller moves food soup 'too fast'. The hypothesis explains the wide variation in expression, intensity and timing of borborygmii.

When only the duodenum transport sub-controller is defective, borborygmii is not a symptom. When only the jejunum transport sub-controller is defective, borborygmii occur, but are delayed by a few minutes. When both the duodenum and jejunum transport sub-controllers are defective, immediate borborygmii occur. When the ileum transport sub-controller is defective, borborygmii occur, but are delayed by a few hours.

Visual hallucinations

These are kaleidoscopic moving patterns in front of the eyes.

- The human body manages circulating free cholesterol levels with the ileum.
- Bile salts are made from cholesterol, stored in the gall bladder, then used to emulsify fats in the duodenum, and later reabsorbed in the ileum.
- The ileum transport sub-controller manages this reabsorption process. When the body's cholesterol level is low, bile salts are recycled. When cholesterol level is high, bile salts are allowed to escape in the stool.

A cholesterol deficit occurs when the ileum transport sub-controller is defective (IBS-A & IBS-D). Bile salts can no longer be recycled and instead are lost in the stool. Now cholesterol is urgently required for making more bile salts. The brain contains large amounts and has to supply some. The process of moving cholesterol out of the brain causes visual hallucinations.

A cholesterol excess occurs; when IBS-B is present, and when back-pressure in the duodenum created by a jejunum Barrier prevents the release of bile. The gall bladder becomes full, and now cholesterol cannot be reduced by making bile salts. Any excess has to be moved into the brain for storage and this process also causes visual hallucinations.

Evidence against the hypothesis

All of the key IBS symptoms, fit neatly into the hypothesis, but the number of cases studied is limited.

Many IBS subjects report that their symptoms vary somewhat from the ones reported here. But differences in age, constitution, diet, lifestyle, disease state, and cause of IBS, mean that there is wide variation in expression of symptoms.

Since this is a hypothesis, some or all of it may be wrong. It should serve nevertheless, to direct researchers into areas they have not visited yet.

Testing the hypothesis

Clinicians may be able to confirm the existence of the three kinds of neuro-transmitter deficient IBS-C predicted by the theory:

1. *The duodenum transport sub-controller output is missing or deficient.* Key symptoms are immediate, severe bloating on eating breakfast, constipation, and in all cases difficulty digesting fats.
2. *The jejunum transport sub-controller output is missing or deficient.* Key symptoms are borborygmii delayed for a short time after starting breakfast, slight to moderate bloating (that is hard to detect), and constipation. Fat digestion will be normal unless IBS-B occurs in tandem.
3. *Both the duodenum and jejunum transport sub-controller outputs are missing or deficient.* Key symptoms are borborygmii starting

immediately after beginning breakfast, slight to moderate bloating (that is hard to detect), and constipation. Fat digestion will be normal unless IBS-B occurs as well.

The two barriers producing neurotransmitter deficient IBS-C may be able to be detected by feeding a breakfast that contains cereal fibre (not corn) and a signaling compound, followed by scanning of the abdomen.

IBS-D may also show three variations of neuro-transmitter deficient IBS. The symptom of borborygmii will allow the clinician to differentiate. If digestive chemicals have been exhausted by continual diarrhoea, or if IBS-B is present, this may not be possible:

1. *The ileum transport sub-controller is malfunctioning.* Borborygmii will start several hours after breakfast. Diarrhoea occurs immediately food reaches the end of the ileum, or on arising the next morning. Cramping is possible.
2. *Both the ileum and jejunum transport sub-controllers are malfunctioning.* Borborygmii will begin a few minutes after starting breakfast. Diarrhoea occurs immediately food reaches the end of the ileum, or on arising the next morning. Cramping is possible.
3. *The ileum, jejunum, and duodenum transport sub-controllers are malfunctioning.* Borborygmii will begin immediately after starting breakfast. Diarrhoea occurs when food reaches the end of the ileum many hours later, or on arising the next morning. Cramping is possible.

Researchers studying brain function can look for the four small intestine brain controllers, with their unique neuro-transmitters, and for under-regulation by one or more of them.

Consequences of the Hypothesis

Stress and IBS

Environmental and lifestyle factors can cause stress, and when the primary brain controller(s) fail then IBS symptoms will aggravate stress. Stress raises the level of activity in the autonomic nervous system by causing the release of adrenal hormones and;

- Any Barrier becomes stronger and its associated constipation, cramping and bloating, are increased in intensity.
- The valve at the end of the small intestine becomes easier to open and diarrhoea may occur.

This explains why psychological treatments that reduce stress [5, 6] have good success in treating IBS.

Diagnosis

Diagnosis of IBS caused by a neuro-transmitter deficiency will be improved if these key criteria are used;

- IBS-C is indicated when constipation occurs but there is NO diarrhoea.
- IBS-D is indicated when diarrhoea occurs but there is NO bloating.
- IBS-A is indicated when diarrhoea AND severe bloating occur.
- IBS-B is indicated when steatorrhea and/or severe symptoms of IBS-C, D, or A occur.

These criteria eliminate the effects of age, constitution, diet, lifestyle, and disease state, from the diagnosis.

Diagnosis of IBS-B is more difficult as steatorrhea can be caused by other factors. Dietary trials may be the only way of confirming that it is occurring. However the presence of severe symptoms is indicative of IBS-B.

If IBS is caused by damage to intestinal sensors then diagnostic criteria will not be so clear cut.

Treatment

Diets free from cereals and whole legumes [4] reduce the symptoms of IBS. These are often called low carb, low fibre, or low starch diets.

However when the small intestine becomes acidic, then treatment is more difficult. A low acid, low fat, low fibre, high carb diet, needs to be eaten. The carbs must not be from cereals (except corn) or whole legumes.

Environment & lifestyle factors, that cause the activity of the autonomic nervous system to be at high levels, increase symptom severity.

Relaxation therapies, that teach how to keep the autonomic nervous system at low levels, are needed. These lower the activity of the autonomic nervous system, weaken the IBS Barrier and keep the valve into the colon more firmly closed.

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Table 1: A summary of the four types and 15 forms of neuro-transmitter deficient IBS predicted by the hypothesis.

#	Primary controller				IBS Type(s)
	Bile	Duodenum	Jejunum	Ileum	
1	X	O	O	O	B
2	O	X	O	O	C
3	X	X	O	O	C, B
4	O	O	X	O	C
5	X	O	X	O	C, B
6	O	X	X	O	C
7	X	X	X	O	C, B
8	O	O	O	X	D
9	X	O	O	X	D, B
10	O	O	X	X	D
11	X	O	X	X	D, B
12	O	X	X	X	D
13	X	X	X	X	D, B
14	O	X	O	X	A
15	X	X	O	X	A, B

Legend: X = defective and O = functioning