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 the 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 applies a default condition. The default
condition is normally active when no food is in the intestine. If food is in the
intestine 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’, irritable bowel syndrome is
caused.
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 a neurotransmitter 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:**
A network of nerves controlling automated functions. Activity levels in this network rise when adrenal hormones are released on awakening, and when stress occurs. After the morning peak of activity, a gradual relaxation 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 the food soup is neutral to slightly alkaline. Human digestive enzymes will not work in acid conditions.

**Bile:**
Bile is made in the liver, stored in the gall bladder, and secreted into the duodenum. The bile salts it contains emulsify 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.

**Borborygmi:**
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.

**Enteric nervous system:**
The network of nerves controlling automated functions in the digestive system. It is a sub-division of the autonomic nervous system.

**Glucose-dependent insulino tropic peptide (GIP):**
This hormone is released from the duodenum when food soup is pumped in from the stomach. It travels to the pancreas where it stimulates the release of insulin.

**Gall Bladder:**
The storage organ for bile produced in the liver. It is emptied by a muscle controlled by the brain and delivers bile into the duodenum.

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

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

*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 makes bile salts from cholesterol and stores them in the gall bladder.

*Neurotransmitters:*
These are chemicals that the nervous system uses to transmit signals between nerve cells. There are a considerable number of them, and many 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 insulin hormone.

*Secretin:*
This hormone is released from the duodenum when food is pumped in from the stomach. It stimulates the pancreas to release 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 to the large intestine. It is about 6-9 m long and divided into three physically different sections:

1. The first section is the duodenum; it is about 25cm long.
2. The second section is the jejunum; it is about 2 to 3m long.
3. The third section is the ileum; it is about 4 to 6m long.

**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 neurotransmitter.

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 and bicarbonate are mixed into the soup, so that acids are neutralized, & 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 nutrients released by enzyme action 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 reabsorbed and returned to the gall bladder and pancreas. The food soup is 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. 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 legume fibre causes high stimulation. Fruit, vegetable and animal fibre causes normal 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, control the rate of absorption, and then transfer it 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 Cholecystokinin (CCK), a peptide hormone, 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 then 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 system takes over when brain sub-controller outputs are missing. Normally this is when there is no food soup present. When food soup is present and an 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 level of control is principally dependent on;

- The amount and types of fibre eaten.
- Environmental 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 occurs.

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 insufficient and extra must be provided by the fourth sub-controller, in order to complete the digestive process.
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 reduces the amounts of bile, enzymes, and bicarbonate entering the intestine, and reduces or eliminates output from the transport sub-controller of the affected part 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:
The Barrier 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 cramping, 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 overnight if the autonomic nervous system returns to a low level of activity.

Diarrhoea:
Diarrhoea 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 can contain high levels of acids, bile salts & enzymes, and has not 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 as 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 neurotransmitter 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, insufficient bile salts are added to the food soup. Undigested fats impair nutrient uptake in the jejunum, and the 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 often it accompanies one of the other types of IBS. When it does, 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. This causes a Barrier to form at the start of the jejunum. When a breakfast containing cereal is eaten, immediate severe bloating occurs. Backpressure in the duodenum keeps the valve from the gall bladder and pancreas closed, so that 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. This causes a Barrier to form at the start of the ileum. When a breakfast containing cereal is eaten, borborygmi occurs 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. Both the duodenum and jejunum sub-controller outputs are deficient or missing. This causes a Barrier to form at the start of the ileum. When a breakfast containing cereal is eaten, borborygmi occurs 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 a breakfast containing cereal is eaten, borborygmi begin when food soup reaches the ileum several hours later. Diarrhoea occurs immediately food soup reaches the end of the ileum or on arising.
2. Form 1 together with IBS-B.
3. The ileum and jejunum sub-controller outputs are deficient or missing. When a breakfast containing cereal is eaten, borborygmi 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.
4. Form 3 together with IBS-B.
5. The ileum, jejunum and duodenum sub-controller outputs are deficient or missing. When a breakfast containing cereal is eaten, borborygmi begin immediately. Diarrhoea occurs as soon as food soup reaches the end of the ileum or on arising.

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 and ileum sub-controller outputs are deficient or missing. This causes IBS-C plus IBS-D. Constipation and 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 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 neurotransmitter is missing.

Damage to sensors in the intestinal walls will cause symptoms that may vary somewhat 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, and diarrhoea results.

**Current theories of IBS:**
Some are;
- Bacterial overgrowth
- Overregulation 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 borborygmi, bloating and cramping occur. This theory explains how all symptoms are created and shows that it is likely to be under-regulation that causes IBS.

**Importance of the theory:**
The theory explains how the nervous system controls the small intestine.

**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 the control systems.

**Cereal & Legume Fibre:**
Diets that remove cereals and legumes [4] reduce symptoms. Stimulation by these foods causes ‘too fast’ speeds when the secondary controller is regulating transport.

**Stress:**
Stress 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 and severe bloating (defective duodenum sub-controller and resulting jejunum barrier) have large backpressures 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. The hypothesis of a defective bile 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 bile salts and enzymes in their bowel movements. The protease 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 is an ileum control problem.

The ‘morning rush’ is a characteristic symptom of IBS-A & D. A fast, loose bowel movement may 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 a neurotransmitter in the brain.

**Intestinal bloating:**
IBS bloating starts on arising when adrenal hormones are released and breakfast is eaten. Stress during the day further increases it. 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 (25cm). 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 2-3m long. 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 the secondary transport controller trying to move food soup through the 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 borborygmi 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 rapidly evacuated with cramping.

**Borborygmi:**
This gurgling sound occurs when the secondary controller moves food soup ‘too fast’. The hypothesis explains the wide variation in expression, intensity and timing of borborygmi.

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

**Visual hallucinations:**
These are kaleidoscopic moving patterns of colour that start near the centre of the visual field and radiate outwards. They occur in many cases of IBS-B, IBS-D & IBS-A.

The human body manages circulating cholesterol levels with the ileum:

Cholesterol is used to make bile salts which are then stored in the gall bladder. This process removes cholesterol from circulation.

Bile salts emulsify fats in the first and second sections of the small intestine and later on they can be reabsorbed in the third section (ileum). The ileum transport sub-controller manages this recycling process. When the body’s cholesterol level is low, most bile salts are recycled. When cholesterol level is high, bile salts are allowed to escape via the stool.

When the ability to eliminate cholesterol via the digestive process is compromised, a high level of cholesterol can build up and cause the brain to output visual hallucinations.

This happens when IBS-B is present and when backpressure created by a jejunum Barrier prevents the release of bile. The gall bladder becomes full. Excess cholesterol can no longer be reduced by making bile salts.

When excessive amounts of bile salts are lost, all available cholesterol is diverted to bile salt manufacture. The resulting low level causes the brain to output visual hallucinations.

This happens when the ileum transport sub-controller is defective (IBS-A & IBS-D). Bile salts can no longer be recycled and are lost when the ileum empties too soon.
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.

Testing the hypothesis

Clinicians may be able to confirm the existence of the three kinds of neurotransmitter 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 a breakfast containing cereal, constipation and difficulty digesting fats in all cases.
2. The jejunum transport sub-controller output is missing or deficient. Key symptoms are borborygmi delayed for a short time after starting to eat a breakfast containing cereal, slight to moderate bloating (that is hard to detect), and constipation. Fat digestion will be normal unless IBS-B occurs.
3. Both the duodenum and jejunum transport sub-controller outputs are missing or deficient. Key symptoms are borborygmi starting immediately after commencing a breakfast containing cereal, slight to moderate bloating (that is hard to detect), and constipation. Fat digestion will be normal unless IBS-B occurs.

The two barriers producing neurotransmitter deficient IBS-C may be able to be detected by feeding meals for breakfast that contain cereal fibre and a signaling compound, then immediate scanning of the abdomen.

IBS-D may also show three variations. The symptom of borborygmi will allow the clinician to differentiate. If digestive chemicals have been exhausted by continual diarrhoea, OR if IBS-B is present, OR if the small intestine is acidic, this may not be possible:

1. The ileum transport sub-controller is malfunctioning. Borborygmi will start several hours after a breakfast containing cereal is eaten. 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. Borborygmi will begin a few minutes after starting to eat a breakfast containing cereal. 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. Borborygmi will begin immediately after starting to eat a breakfast containing cereal. Diarrhoea occurs as soon as food reaches the end of the ileum, or on arising the next morning. Cramping is possible.

Researchers studying brain function can look for under-regulation, and for the four small intestine brain controllers with their unique neurotransmitters.

**Consequences of the Hypothesis**

**Stress and IBS:**
Environmental and lifestyle factors can cause stress, and when the primary small intestine controller fails, the symptoms will aggravate stress. Stress raises the level of activity in the autonomic nervous system by causing adrenal hormone release. The IBS barrier then becomes stronger and constipation, cramping and bloating are increased in intensity. The valve into the colon becomes easier to open and diarrhoea occurs.

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

**Diagnosis:**
Diagnosis of IBS caused by a neurotransmitter deficiency will be improved if these key criteria are used;

- IBS-C; constipation and NO diarrhoea.
- IBS-D; diarrhoea and NO bloating.
- IBS-A; diarrhoea AND bloating.
- IBS-B; steatorrhea and/or severe symptoms of IBS-C, D or A.

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

Diagnosis of IBS-B is more difficult as steatorrhea can be caused by other factors. Dietary trials may be the only way of definitely finding out if 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 legumes [4], reduce the symptoms of IBS. These are often called low carb, or low fibre, or low starch diets.

When the small intestine becomes acidic, treatment is more difficult. The diet needs to be low acid and low fat, as well as low fibre.

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, weaken the IBS barrier and keep the valve into the colon firmly closed.
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References


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

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<th>#</th>
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<th>Jejunum</th>
<th>Ileum</th>
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Legend: X = defective and O = functioning