# The Japanese Low FODMAP Diet Manual

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By

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# CONTENTS

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*The authors would like to praise the efforts of the final proofreader, Jo Parsons, United Kingdom.*

# PREFACE

## YOSHIHARU UNO

I have been working as a bowel disease specialist for 30 years; however, I was not interested in IBS at all until a few years ago because the cause of IBS was difficult to understand. I could only believe what I could actually see with my eyes. For that reason, I was devoted to research that used endoscopies. However, after researching endoscopic caecostomy in 2003, I became interested in constipation therapy. This was because, I noticed in an antegrade enema from a caecostomy that the transit time was in proportion to the width of the intestinal lumen. In addition, I noticed that the transit time was prolonged by colon gas.

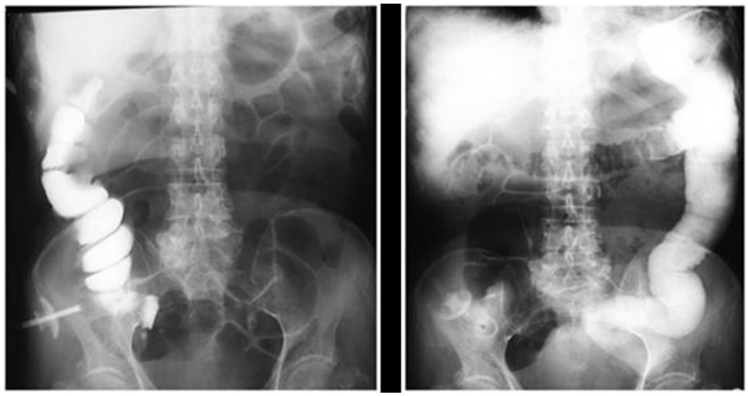

Figure 1. *ACE from caecostomy (left). Twenty minutes after ACE (right). The liquid contrast agent moved to the descending colon. However, the gas stayed in the transverse colon from caecum (Uno: 2003, 2004, 2006).*

After this, I treated many patients who were suffering from constipation. In addition, my sister, who had been taking laxatives for a long time, passed away due to colon cancer. Therefore, I read the past 100 years of research papers on constipation thoroughly. I found that there are many patients who are laxative dependent, and these patients have excessive dilation of the colon. Moreover, I found from abdominal X-rays that, in

addition to the quantity of stool, the quantity of gas was also significant in constipation. Therefore, I started to think that patients should reduce the total amount of both stool and gas. I told my patients to reduce their fiber intake, and to avoid wheat products, as these decrease gas production in the intestines. I also told them to avoid fermented foods, xylitol, or any gas producing foods.

In 2013, when I learned about FODMAPs, I realized the effect of dietary restriction of fermented foods. After 2013, I then became interested in low-FODMAP theory and made it my mission to spread this dietary method in Japan and the rest of the world. However, the more I read books or research papers, which were published in Australia and the West, I realized that not all the theories matched the Japanese diet or lifestyle. This made me think there was a necessity for a low-FODMAP diet modified for Japanese people. The most important problem was that low-FODMAP theory contradicts “the good bacteria theory”, which is strongly believed in Japan. Therefore, I thought that for this new theory to be accepted in Japanese society, the establishment of a scientific theory that was logical and without contradiction was needed. Moreover, there were some other difficulties in spreading the word about a low-FODMAP diet as it could have a large impact on Japan’s agriculture, the dairy industry, the restaurant industry, the instant food industry, and the confectionery industry. A vague, incomplete knowledge would not succeed in spreading the low-FODMAP diet in Japan. Therefore, I investigated the root cause of each FODMAP group, then learned all the details of how the research had been completed. However, when you walk into Japanese book stores, the shelves are full of health-related books that only advocate conventional dietary methods with new names, as though they were new inventions. Also, in Japan, fermented foods, which are high in FODMAPs, are very prevalent. Books advocating the health benefits of foods such as burdock, yoghurt, and garlic are widely sold in Japan. In 2015, I obtained the exclusive right to trademark low-FODMAP for education, speeches, publications, broadcasts, and merchandise. In this book, I have written my suggestions for a low-FODMAP diet for Japanese IBS patients. In addition, I will discuss the research that I have carried out on the subject matter.


Figure 2: *T-shirt from a Japanese low-FODMAP diet promotion group (JLFDPG)*

# PURPOSE OF THE ENGLISH VERSION PUBLICATION

YOSHIHARU UNO

No one has doubt regarding the effectiveness of a low-FODMAP diet. However, everyday meals are different, and dependent on the country's ethnic and religious differences. Many people around the world may think that Japanese only eat rice and fish. However, since the end of World War II, Japan has imported a large amount of flour, and bread and milk are also supplied for feeding hospital and school meals. Furthermore, it is currently believed that the high FODMAP content of fermented food is healthy in Japan and, as a result, most FODMAPs are included in Japanese foods. Even with such difficult circumstances, we are trying to help patients with IBS to follow a low-FODMAP diet. To that end, we have provided a lot of information in this book, and we hope to share our knowledge about a low-FODMAP diet around the world.

# INTRODUCTION BY THE SECOND AUTHOR

## MAMI NAKAMURA

I am a Japanese pharmacist living in Melbourne, Australia. I was diagnosed with IBS and was referred to a dietitian specializing in low-FODMAP diets. It took me a while to understand and accept a low-FODMAP as it was so different from what I knew. Since the day I met the dietitian, I have started reading lots of books related to IBS and low-FODMAP diet. However, luckily, I am in the leading low-FODMAP country and, through the program, I experienced the whole process and leaned how to manage IBS with a low-FODMAP diet.

Almost no information was available in Japanese but I managed to find Dr. Uno's column on the internet and I got to know him through his column. I was moved by his substantial contribution to Japanese IBS patients and, as we shared information about low-FODMAP diet in Japan and Australia, we decided to publish his book in English.

# CHAPTER I

# GENERAL DISCUSSION

## 1. Diseases related to FODMAP

### 1) What are FODMAPs?

F → fermentable
O → oligosaccharides
D → disaccharides
M → monosaccharides
P → polyols: these are sugar alcohols.

FODMAPs are poorly absorbed from the small intestine because they are not easily, or even not at all, degraded in the small intestine. They are also highly osmotic, meaning that they attract water into the lumen of the ileum, and then they are fermented in the large intestine, contributing to the production of water and gas. High-FODMAP foods lead to gut distention, resulting in abdominal pain, flatulence, abdominal bloating, diarrhea, and bowel movement disturbance. These are symptoms of irritable bowel syndrome (IBS).

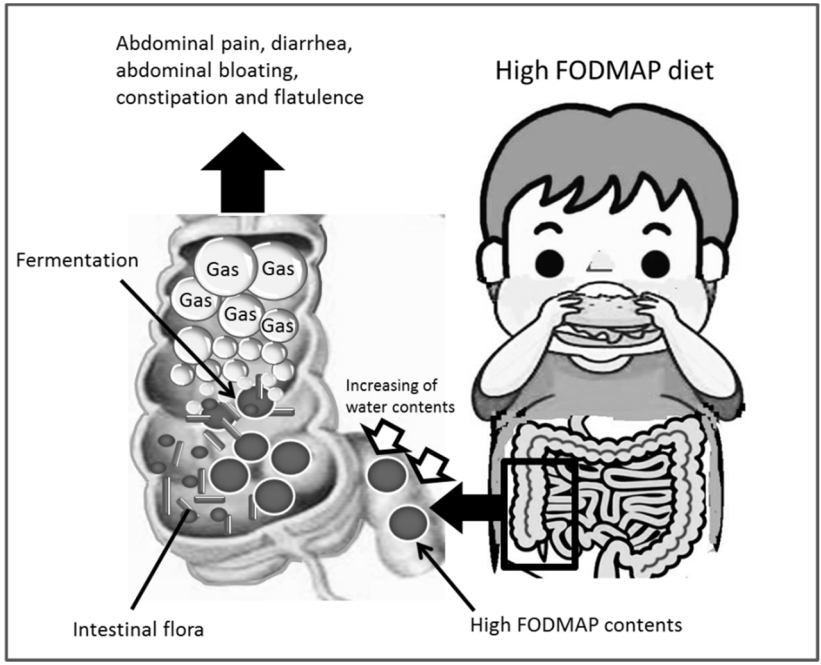


Figure 3: *High-FODMAPs increase the water in the lumen of the ileum, and are fermented in the large intestine. The production of gas by fermentation results in abdominal bloating, flatulence, stomach ache, diarrhea, and bowel movement disturbance.*

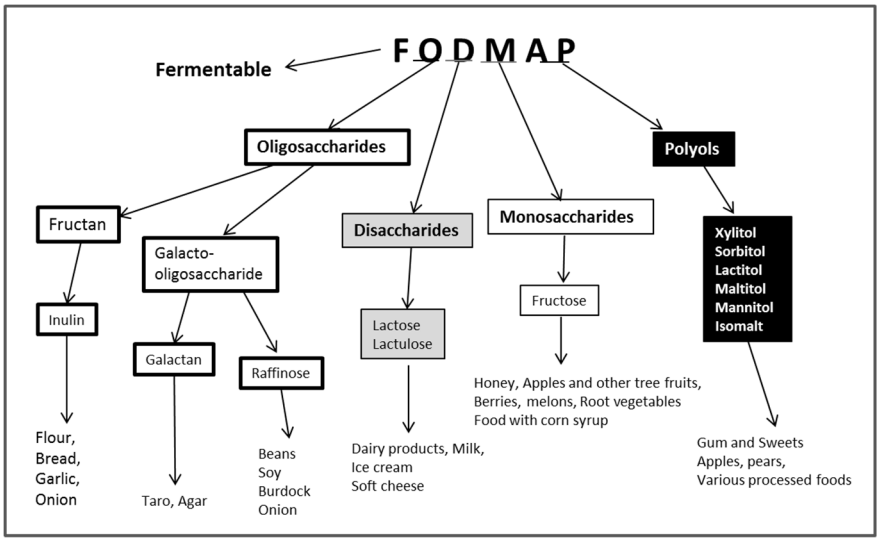


Figure 4: *High-FODMAPs chart*

High-FODMAP foods are also associated with many diseases other than IBS.

## 2) Criteria for diagnosing functional bowel disorder

The international diagnostic criteria for IBS were updated and revised: Rome III Criteria in 2006 and Rome IV for IBS diagnosis in 2016.

**Criteria for IBS diagnosis in 2006 (Rome III)**

Diagnostic criterion*

Recurrent abdominal pain or discomfort** at least 3 days/month in the last 3 months associated with two or more of the following:

(1) Improvement with defecation.

(2) Onset associated with a change in frequency of stool.

(3) Onset associated with a change in form (appearance) of stool.

* Criterion fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis.

** “Discomfort” means an uncomfortable sensation that is not described as pain.

**IBS criteria of Rome IV in 2016**

Diagnostic criterion*

Recurrent abdominal pain on average at least 1 day/week in the last 3 months associated with two or more of the following:

(1) Related to defecation

(2) Associated with a change in frequency of stool

(3) Associated with a change in form (consistency) of stool

* Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

**Diagnostic criteria for IBS subtypes**

IBS with predominant constipation (IBS-C): > 1/4 (25%) of bowel movements with Bristol Stool types 1 or 2 and < 1/4 (25%) of bowel movements with Bristol stool types 6 or 7.

IBS with predominant diarrhea (IBS-D): > 1/4 (25%) of bowel movements with Bristol Stool types 6 or 7 and < 1/4 (25%) of bowel movements with Bristol Stool types 1 or 2.

IBS with mixed bowel habits (IBS-M): > 1/4 (25%) of bowel movements with Bristol stool types 1 or 2 and > 1/4 (25%) of bowel movements with Bristol Stool types 6 or 7.

IBS unclassified (IBS-U): Patients who meet diagnostic criteria for IBS but whose bowel habit cannot be accurately categorized into one of the three groups above should be categorized as having IBS-U.

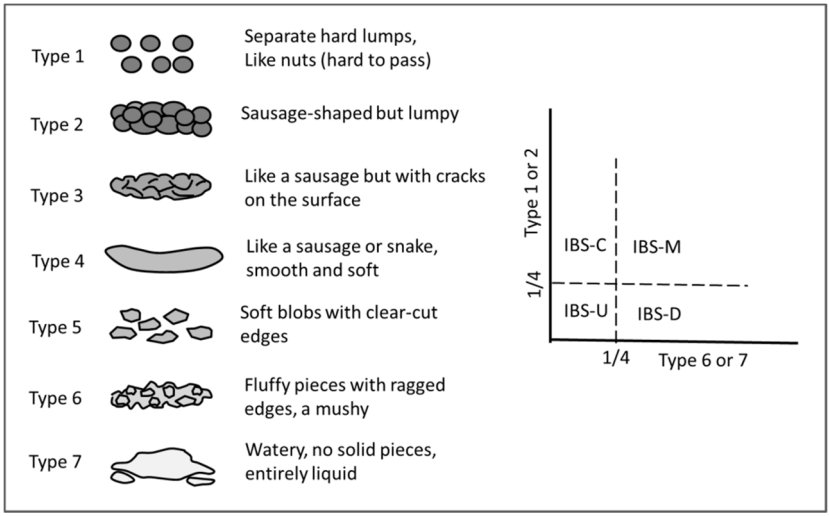


Figure 5: *Bristol Stool Scale (Drossman: 2016)*

**Irritable Bowel Syndrome in Children (Rome IV)**

Diagnostic criteria*

Must include 1 or more of the following:

(1) Abdominal pain at least 4 days per month associated with one or more of the following:

a. Related to defecation

b. A change in frequency of stool

c. A change in form (appearance) of stool

(2) In children with abdominal pain and constipation, the pain does not resolve with the resolution of the constipation (children in whom the pain resolves have functional constipation, not IBS)

(3) After an appropriate evaluation, the symptoms cannot be fully explained by another medical condition

* Criteria fulfilled for at least 2 months prior to diagnosis

**Functional Abdominal Bloating /Distension (Rome IV)**

Diagnostic criteria*

Must include both of the following:

(1) Recurrent bloating and/or distension occurring on average at least 1 days/week; abdominal bloating and/or distension predominates over other symptoms**

(2) There are insufficient criteria for a diagnosis of irritable bowel syndrome, functional constipation, functional diarrhea, or postprandial distress syndrome

* Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

**Mild pain related to bloating may be present as well as minor bowl movement abnormalities

**Functional Constipation (Rome IV)**

Diagnostic criteria*

(1) Must contain 2 or more of the following:**

a. Straining during more than ¼ (25%) of defecations

b. Lumpy or hard stools (Bristol Stool Form Scale 1–2) more than 1/4 (25%) of defecations

c. Sensation of incomplete evacuation more than ¼ ( 25%) of defecations

d. Sensation of anorectal obstruction/blockage more than ¼ (25%) of defecations

e. Manual maneuvers to facilitate more than ¼ (25%) of defecations (e.g., digital evacuation, support of the pelvic floor)

f. Fewer than 3 defecations (spontaneous bowel movement) per week

(2) Loose stools are rarely present without the use of laxatives

(3) Insufficient criteria for irritable bowel syndrome

* Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

** For research studies, patients meeting criteria for opioid-induced constipation should not be given a diagnosis of FC because it is difficult to distinguish between opioid side effects and other causes of constipation However, clinicians recognize that these conditions may overlap.

**Functional Constipation of Child (Rome IV)**

Diagnostic criteria

Must include 1 month of at least 2 of the following in infants up to 4 years of age:

(1) 2 or fewer defecations per week

(2) History of excessive stool retention

(3) History of painful or hard bowel movements

(4) History of large diameter stools

(5) Presence of a large fecal mass in the rectum

In toilet trained children, the following additional criteria may be used:

(6) At least one episode/week of incontinence after the acquisition of toileting skills

(7) History of large diameter stools which may block the toile**t**

**Functional Diarrhea (Rome IV)**

Diagnostic criterion*

Loose or watery stools, without predominant abdominal pain or bothersome boating, occurring in more than 25% of stools**

* Criterion fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

**Patients meeting criteria for IBS-D should be excluded

**Functional Diarrhea of Child (Rome IV)**

Diagnostic criteria

Must include all of the following:

(1) Daily painless recurrent passage of four or more large, unformed stools

(2) Symptoms last more than 4 weeks

(3) Onset between 6 and 60 months of age

(4) No failure-to-thrive if calorie intake is adequate

**Infant Colic (Rome IV)**

Diagnostic criteria

For clinical purposes, must include all of the following:

(1) An infant who is less than 5 months of age when the symptoms start and stop

(2) Recurrent and prolonged periods of infant crying, fussing, or irritability reported by caregivers that occur without obvious cause and cannot be prevented or resolved by caregivers

(3) No evidence of infant failure to thrive, fever, or illness

For clinical research, a diagnosis of infant colic must meet the preceding diagnostic criteria and also include both of the following:

(1) Caregiver reports infant has cried or fussed for 3 or more hours/day during 3 or more days in 7 days in a telephone or face-to-face screening interview with a researcher or clinician

(2) Total 24-hour crying plus fussing in the selected group of infants is confirmed to be 3 hours or more when measured by at least one prospectively kept 24-hour behavior diary

**Fecal Incontinence (Rome IV)**

Diagnostic criterion*

Recurrent uncontrolled passage of fecal material in an individual with a developmental age of at least 4 years

*Criterion fulfilled for the last 3 months previously with 2–4 episodes of fecal incontinence over 4 weeks

## 2. Small intestinal bacterial overgrowth (SIBO) and IBS

The frequency of SIBO in patients with IBS is 4–78% (Ghoshal et al.: 2014). In addition to the IBS disease described in the Rome diagnostic criteria, small intestinal bacterial overgrowth (SIBO) is an important disease. Bacteria are normally present throughout the entire gastrointestinal tract, but relatively few bacteria live in the small bowel when compared with the large bowel. Less than 10,000 bacteria per milliliter of fluid live in the duodenum to the jejunum; however, when it comes to the distal ileum and colon, there are one billion and one trillion, respectively. There are also at least a quadrillion bacteria per kilogram of feces living throughout the gastrointestinal tract (Andoh. 2015).

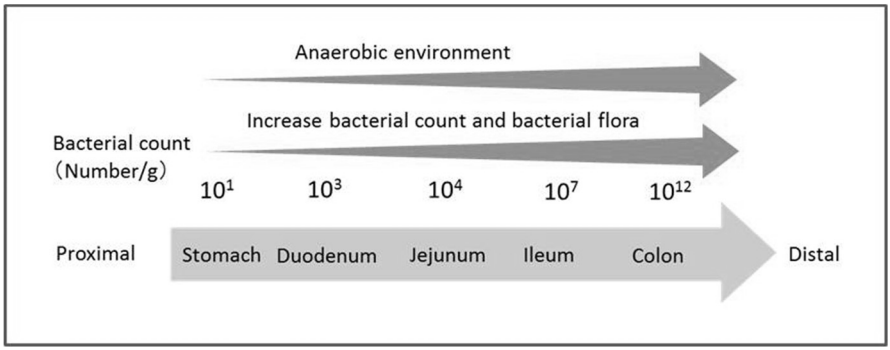


Figure 6: *Number of bacteria in the digestive tract (Andoh: 2015)*

The increased bacterial population due to SIBO causes excessive fermentation in the small intestine. Gas is produced by the fermentation of sugars, including glucose and sucrose, leading to the symptoms seen, such as gas production, bloating, pain, and diarrhea. In a study on patients with colectomy (Rao et al.: 2018), significant differences were found in the type of bacterial flora, with a predominance of aerobic bacterial organisms and fewer anaerobic organisms in post-colectomy SIBO patients when compared to controls. The duodenal cultures grew a variety of organisms primarily including *Streptococcus* species, *Escherichia coli*, *Klebsiella pneumoniae*, and *Lactobacilli*.

**Diagnosis of SIBO**

Historically, the diagnosis of SIBO was performed by the direct aspiration and culture of jejunal fluid, and was defined as an observation of 100,000 CFU/mL (colony forming unit per 1 milliliter of aspirated fluid from the jejunum). However, this cut off was not well-validated and has been a point of controversy. In a systematic review on the diagnosis of SIBO, it was observed that healthy controls have a bacterial concentration of $\leq 10^3$ CFU/mL, while concentrations of $\geq 10^5$ CFU/mL are mostly seen in patients with blind loop syndrome: such as patients with Billroth II procedure (Khoshini et al.: 2008). Currently, a bacterial concentration of > 1,000 CFU/mL is generally considered significant for diagnosis of SIBO (Erdogan et al.: 2015, Jacobs et al.: 2013, Pyleris et al.: 2012, Giamarellos-Bourboulis et al.: 2012).

However, because the aspiration-based method is an invasive test, indirect and non-invasive tests—LHBT (lactulose hydrogen breath test) and GHBT (glucose hydrogen breath test)—have been commonly used as alternatives (Ghoshal et al.: 2011, 2014). In SIBO, gas in the small intestine is increased by the fermentation of carbohydrates in the small intestine. Gas in the small intestine is carried to the blood circulation through capillaries and is excreted from the lungs. Therefore, the most common method for diagnosing SIBO has been to measure the hydrogen gas or methane gas exhaled after ingestion of 100g of glucose or 10g of lactulose.

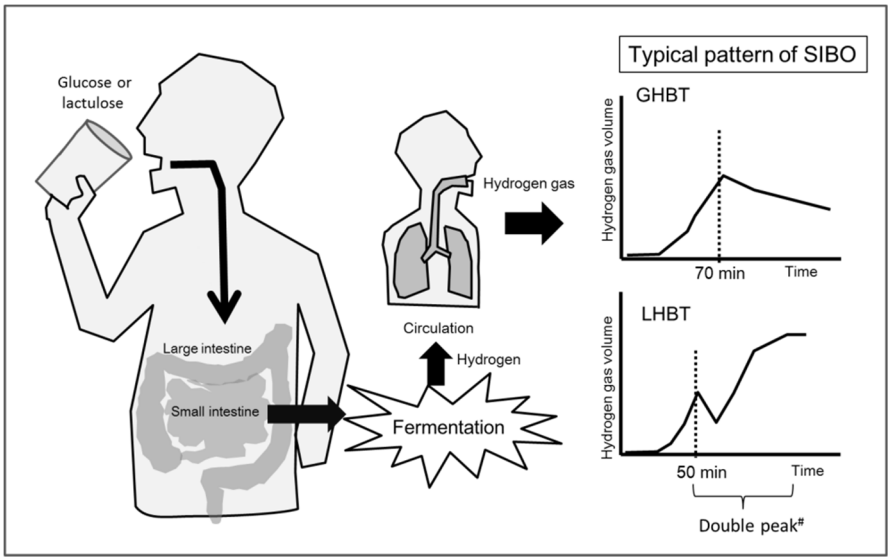


Figure 7: *GHBT and LHBT in SIBO*

*#(Rezaie et al.: 2017)*

**SIBO breath test:** patients drink a sugar solution of glucose or lactulose after a 1- or 2-day preparatory diet. The diet removes much of the food that would feed the bacteria, allowing for a clear reaction to the sugar drink. Breath testing measures the hydrogen gas produced by the bacteria in the small intestine that has diffused into the blood, then lungs, for expiration. Hydrogen is a gas produced by bacteria; therefore, the amount of hydrogen gas can be indirectly used to measure the number of bacteria present in the intestines. However, the credibility of this method has been doubted, since it shows a positive rate of between 4 and 78% (Ghoshal et al.: 2014), so it may not be accurate. In 2015, assessment of over 15,000 lactulose breath tests showed that median and mean gas production levels do not elicit a double peak (Chang et al.: 2015). The North American consensus meeting in 2015 (Rezaie et al.: 2017), suggested using a rise of $\geq$ 20 ppm from the baseline in hydrogen to diagnose SIBO. Additionally, this meeting suggested that, on the basis of current evidence, a double peak should not be used to diagnose SIBO and has no validity, and the test should be performed for at least 3 hours to ensure the presence of colonic

fermentation. Furthermore, these researchers suggested using a cut of ≥ 10ppm of methane positivity for a diagnosis of SIBO.

Usually, sucrose and glucose are absorbed in the small intestine and do not contribute to fermentation in the large intestine. In SIBO, however, these sugars can be fermented in the small intestine and, because of this, when the patient complains of wind-related abdominal symptoms after only the sucrose consumption it is highly likely to be due to SIBO. In a metagenomic analysis study in 2018 (Sundin et al.: 2018), researchers described that the glucose-based hydrogen and methane breath test was not sensitive to the overgrowth of jejunal bacteria. However, they also discussed that a positive breath test may indicate altered jejunal function and microbial dysbiosis. However, in a 2018 study using breath test and scintigraphy in combination, it was shown that the colon transit time increases when there is a high occurrence of methane gas in the small intestine (Suri et al.: 2018).

**Abdominal imaging:** In SIBO—whether methane or strictly hydrogen is produced—gas can have a significant effect on the patient's quality of life. For patients suspected of having SIBO, confirming the increase in small intestinal gas at the time of occurring symptoms will aid diagnostic determination. In order to determine the gas increase, it should be compared using abdominal imaging (CT, MRI, and X-ray) before and after the onset of symptoms. The easiest method would be to induce symptoms by ingestion of glucose and confirm the gas increase at that time (Uno Y: unpublished study).

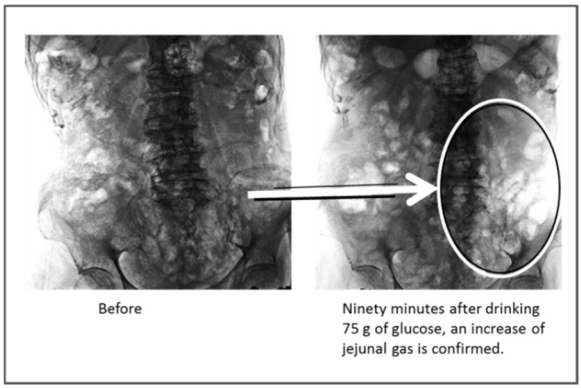


Figure 8: *Diagnosis of SIBO via an X-ray image (Personal data of Uno)*

**Treatment of SIBO**

In 2011, Pimentel et al. in the US indicated that SIBO is the main cause of IBS and advocated the eradication of gut bacteria using rifaximin (Pimentel et al.: 2011). As mentioned, the incidence of SIBO in IBS patients is under debate in terms of testing methods and facilities. However, as rifaximin suppresses bacterial growth: *E. coli* (85.4% inhibition rate), *Klebsiella* (43.6% inhibition rate), *Enterococcus* (100% inhibition rate), and *Staphilococcus aureus* (100% inhibition rate) efficiently, the effects could be promising. The problem in SIBO is usually sugars (carbohydrates), which are fermented by the bacteria in the small intestine before their absorption. If IBS symptoms are not relieved with low-FODMAP diet and if there is a chance that it could be SIBO, then the dietary restriction of all carbohydrates may be effective.

**Drug-induced SIBO**

**Non-steroidal anti-inflammatory drugs (NSAIDs)**: In 2014, the Osaka Medical University in Japan reported that chronic non-steroidal anti-inflammatory drug users were susceptible to SIBO due to damage to the small intestinal mucosa (Muraki et al.: 2014).

**Proton pump inhibitors (PPI)**: PPIs are used to treat gastric ulcers and the eradication of *Helicobacter pylori*. However, in 2010 it was reported that about 50% of patients treated with PPIs for a year develop SIBO and the incidence of SIBO is eight times higher than normal (Lombardo et al.; 2010). The diagnosis of SIBO in that study was done with a breath test. Also, a study into the diagnosis of SIBO via the culture of duodenal aspirate in 2013 also showed a similar increase in SIBO with PPI use (Jacobs et al.: 2013). In a 2018 meta-analysis study (Su et al.: 2018), a total of 19 articles met the eligibility criteria for the meta-analysis in 7055 subjects. The pooled odds ratio (OR) showed a statistically significant association between increased risk of SIBO and PPI use (OR 1.71). In a systematic review and meta-analysis of 50 studies in 2018 (Chen et al.: 2018), more than 1/3 of IBS patients tested positive for SIBO and the odds of SIBO in IBS were increased by nearly fivefold. The prevalence of SIBO varied according to the diagnostic modality performed. When the pH in the small intestine increases by one, then the bacteria in the small intestine is increased by 13.8%. Long-term use of PPIs causes an increase in the pH of the stomach, and the barrier function in the stomach decreases so that the number of bacteria in the small intestine increases. Omeprazole,

Lansoprazole, Pariet (rabeprazole), and Nexium (esomeprazole) are used clinically for the treatment of gastric ulcers, duodenal ulcers, reflux esophagitis (which increases after the eradication of *H. pylori*), and non-erosive gastro-esophageal reflux disease (GERD) in Japan. In particular, lansoprazole is commonly used in conjunction with aspirin or NSAIDs. $H_2$ blockers (famotidine) also increase the gastric pH. Unlike other countries, because Japanese people take PPIs frequently it is estimated that there are more potential SIBO patients in Japan. When PPIs were first used in Japan, the period of use was limited to 2 weeks. However, more recently there has been no limit on the length of administration period. PPIs are mainly formulated for chronic abdominal pain and continuous heartburn but, in practice, the symptoms (abdominal pain and heartburn) may be side effects from the administered drug.

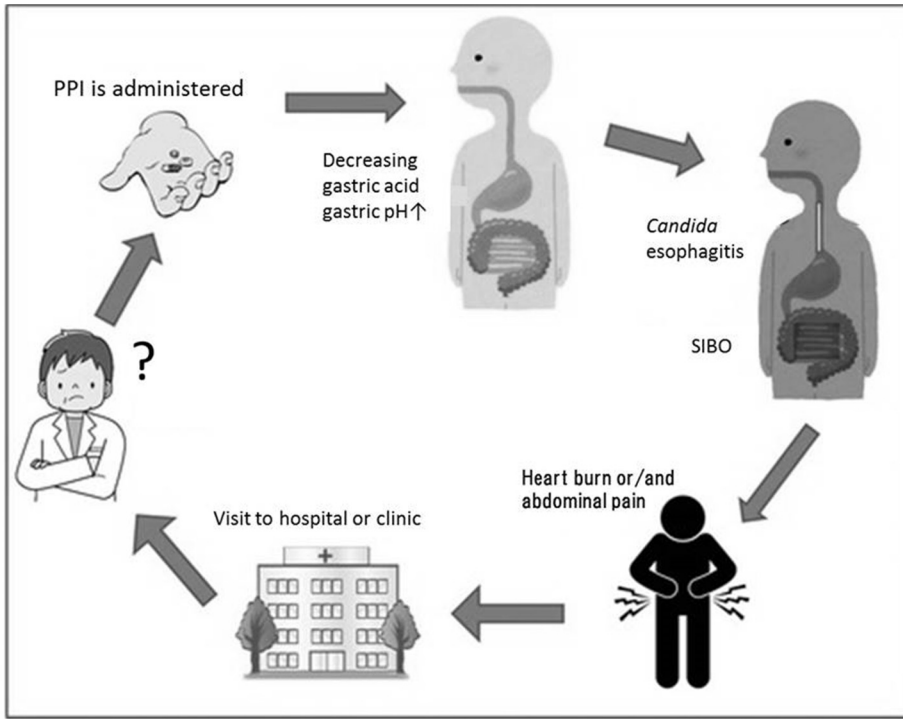


Figure 9: *The cycle between PPI and SIBO in Japan*