Western Herbal Medicine and Digestion

Thomsonism and Physiomedicalism

According to 19th century herbalist Samuel Thomson, whose practices would profoundly influence the later development of herbal medicine in the Western world, good digestion is the foundation of health. In contrast to the dominant medical theories of the day, which relied entirely upon a simplistic understanding of the circulatory system, Thomson conceived of an alternative medical approach in which the property of "heat" was synonymous with health, and the property of "cold" serving as the primary cause of disease. Thomson believed that good health arose when the heat of digestion radiated outwards from the stomach to the periphery of the body. "The heat is maintained in the stomach by consuming food; and all the body and limbs receive their proportion of nourishment and heat from that source; as the whole room is warmed by the fire which is consumed in the fireplace" (Thomson 50, 1825).

Fundamental to the conceptual framework established by Thomson was the idea that "concretions" could build up in the stomach, impairing absorption and weakening digestion. Thus, cleaning the stomach of these concretions or accumulations was tantamount to regaining health. To do this Thomson felt that "all the art required" was "to know what medicine will do it, and how to administer it, as a person knows how to clear a stove and the pipe when clogged with soot" (Thomson 190, 1825).

According to Thomson's framework, disease arises when the internal heat of the stomach is obstructed from circulating to the periphery, or when an attack of cold or shock comes from without, obstructing the natural flow of the internal heat. Following this theory, Thomson advocated diaphoresis, or sweating therapies, as a general cure to "remove all obstructions from the system (and) restore the powers of digestion" (Thomson 506, 1841). Thomson felt that to "promote a natural perspiration is universally applicable in all cases of disease, and

therefore may be considered as a general remedy" (Thomson 506, 1841). Thomson believed that the presence of fever was the most important single factor in overcoming disease, and where obstruction or a deficiency of heat promoted disease, he sought to induce a fever to assist the body in its battle to overcome the underlying pathology of coldness.

Throughout Thomson's writing he refers to "canker" as an archetypal form that disease can manifest. What Thomson referred to as a canker were the physical symptoms of coldness, noticed as the "white …coat(ing) that was attached to the mucous membranes" (Thomson 507, 1841). "Canker and putrefaction are caused by cold, or want of heat, for whenever any part of the body is so affected with the cold so as to overpower the natural heat, putrefaction commences; and if not strong enough to overcome its progress, it will communicate with the blood, when death will end the contest between heat and cold, or the powers of life and death by deciding in favor of the latter" (Thomson 507, 1841).

The remedy Thomson considered of prime importance to restore the natural heat of the body was lobelia (*Lobelia inflata* herb), what he would later call No. 1 in his patented system of healing. Using the analogy of the wood stove, Thomson compared the activity of lobelia to dry wood shavings added to a dying fire, enkindling and ensuring a proper burn to draw the smoke out through the chimney. Thus lobelia was stimulant to digestion, and promoted the movement of the stomach's heat to the periphery, correcting circulation. And just as the cause of a fire that will not burn properly can be the accumulation of soot in the stovepipe, lobelia could also throw off the canker through its emetic activity. "It is searching, enlivening, quickening, and has great power in removing obstructions" (Thomson 589, 1841). But just as kindling burns quickly, Thomson found that the activity of lobelia "…soon exhausts itself, and if not followed by some other medicine, to hold the vital heat till nature is able to support itself by digesting the food, it will not be sufficient to remove a disease that has become seated" (Thomson 589, 1841). Thus began his search for such a remedy.

Thomson tried various pungent-tasting herbs, such as ginger (Zingiber officinalis rhizome), mustard (Brassica nigra seed), and peppermint (Mentha piperita leaf) to hold the heat in the body, and while he had good results with these herbs, found them to be "...more or less volatile, and would not have the desired effect" (Thomson 591, 1841). One day in 1805, on a trip to visit some friends on a farm in Massachusetts, Thomson stopped in at a local residence. When he entered the house he saw a large string of cayenne peppers hanging on the wall. Although he knew them to be pungent in nature, he wasn't sure of their medicinal activity. He purchased the string of peppers and took them home, prepared a powder from them, and experimented upon himself. The taste and resulting effect was remarkable to Thomson, and was certainly the most pungent and heating herb he had ever tasted. Thomson later put cavenne to the test when he felt a cold coming on, and found that it promoted a good perspiration and dispelled his symptoms. Thomson felt at last he had found his No.2 medicine, cayenne (Capsicum annuum fruit), something to sustain the fire of digestion after lobelia had enkindled it. According to Thomson, cayenne "...is no doubt the most powerful stimulant ever known; its power is entirely congenial to nature, being powerful only when raising and maintaining the heat, on which life depends. It is extremely pungent, and when taken sets the mouth as it were on fire; this lasts, however, but a few minutes, and I consider it essentially a benefit, for its effects on the glands causes the saliva to flow freely, and leaves the mouth clean and moist" (Thomson 593, 1841).

While lobelia and cayenne were to become the mainstay of many of his treatments, there were many more herbs that Thomson found useful in practice. In deep-seated conditions, after he employed lobelia to stimulate the fires of the body and cayenne to hold it, Thomson found he needed something else to continue the work of removing the canker, without necessarily promoting emesis. For this purpose he employed the pungent and astringent bayberry (Myrica cerifera bark). "This valuable article may be taken separately, or compounded with other substances, and is the best remedy for canker that I have ever found." Thomson soon found other plants that could do the job as well as bayberry, and began to use other, similarly astringent and warming herbs such white pond lily (Nymphaea odorata root), sumac (Rhus glabra leaf), hemlock (Pinus canadensis bark), witch hazel (Hamamelis virginiana bark), raspberry (Rubus idaeus leaf), and marsh rosemary (Statice limonium root). Having established these three primary classes of therapeutic activity, Thomson used this approach repeatedly, with excellent results. Thomson was by no means limited however to these three simple approaches however, and as his experience grew so did the subtly of his practice. We find that he added plants to his therapeutic armamentarium continually. After using No.3 to remove the canker, Thomson then gave his No.4 remedy to correct the digestive organs and enhance secretion, which could be dosed as a single or combination of bitter tasting herbs such as balmony (Chelone glabra root), bitter root (Apocynum androsaemifolium root), poplar (Populus tremuloides bark), and barberry (Berberis vulgaris root).

From a modern perspective, Thomson's belief that all disease is a subset of poor digestive function may appear simplistic and quaint. It must be remembered, however, that Thomson used this theory of disease as the basis of treatment in a wide variety of conditions, from arthritis to yellow fever, and published many successful case histories in his autobiography. Thomson's therapeutic success was later replicated later by his many followers, who published their own case histories using his methods. Although the legal scope of practice for modern herbal medicine makes some of Thomson's techniques impractical, the basic concept of restoring the natural heat of the stomach as a way to resolve disease remains valid. The development of the later Physiomedical tradition that found inspiration in Thomson's approach, exemplified by the work of W.H. Cook (*The Physiomedical Dispensatory* 1869) and T.J. Lyle (*Physiomedical Therapeutics, Materia Medica and Pharmacy*, 1897), gradually moved away from the emetic and purgative therapies advocated by Thomson, using instead a modified technique that is less invasive and more suited to a clinical setting. This approach also allowed for the development of more complex and sophisticated formulas, such as Robert's formula.

Robert's formula is used in the treatment of duodenal ulcer and digestive inflammation, originally mentioned in a short book called *The Herbal Care of Duodenal Ulcers* (1952), written by British herbalist Captain Frank Roberts. Robert's formula is comprised of equal parts roots of goldenseal root, purple coneflower root, pokeroot, cranesbill geranium root, and marshmallow root. Reaching beyond Thomson's simple method of using emetics, stimulants, and cholagogues in a successive fashion, Robert's formula utilizes a multi-pronged approach to simultaneously address different elements of digestion inflammation:

• <u>Robert's Formula</u>

- 20 mL purple coneflower (*Echinacea angustifolia* root)
- o 20 mL poke root (*Phytolacca americana* root)
- 20 mL marshmallow (Althaea officinalis root)
- 20 mL goldenseal (*Hydrastis canadensis* root/rhizome)
- o 20 mL cranesbill geranium (*Geranium maculatum* root)
 - Rx: 20-40 gtt. bid-tid

The demulcent marshmallow root is used to soothe mucosal inflammation, whereas the astringents cranesbill geranium root and goldenseal root act to remove morbid accumulations, stop bleeding, and promote healing. As a bitter-tasting herb, goldenseal root also helps to restore gastric and hepatic secretions, and along with purple coneflower root, inhibits bacterial pathogens such as *H. pylori* that play a role in ulcerogenesis. Herbs including poke root and purple coneflower root also help to modulate the immune response in the gut by decongesting the lymphatic capillaries in the lamina propria.

Over the decades the approach of Western Clinical herbal medicine has been expanded to include a great diversity of approaches, involving a large materia medica for digestive disease classified according to their specific actions. The holistic approach taken by Western herbal medicine includes a variety of rationales, each used to counter a specific symptom or group of symptoms present in the disease. The following is an overview of the basic actions of botanicals used in digestive disorders, and examples of each:

Botanical action	Example botanicals	
demulcents	marshmallow root, slippery elm bark, licorice root, comfrey root, Irish moss	
bitters	gentian root, barberry root, goldenseal root, centaury root, calendula	
astringents	oak bark, bayberry bark, cinnamon bark, witch hazel bark, cranesbill geranium root	
stimulants	ginger rhizome, horseradish root, cayenne, prickly ash	
carminatives	fennel seed, mint leaf, ginger rhizome, cardamom seed, caraway seed, dill seed	
bulk laxatives	psyllium husk, flax seed, hemp seed	
aperients and laxatives	cascara bark, turkey rhubarb root, senna pod, aloe leaf	
antispasmodics	crampbark, hops strobile, lobelia, wild yam root, henbane, belladonna	
hepatics and cholagogues	bitters (see above); dandelion root, artichoke root, milk thistle seed, fringetree bark, barberry root, poplar bark, greater celandine root	
antimicrobials	purple coneflower root, wild indigo root, myrrh resin, poke root, western red cedar leaf	
antihelminthics	pumpkin seed, garlic, wormwood leaf, male fern root	

Table 1: Herbal actions and examples

Each action listed above contains specific indications and contraindications, as follows:

Botanical action	Indications	Contraindications
demulcents	mucosal inflammation, hyperacidity	excessive phlegm/catarrh
bitters	poor appetite, gastric reflux, hypochlorhydria, bilious dyspepsia, steatorrhea	duodenal ulcers
astringents	catarrhal conditions, diarrhea, hemorrhage	constipation, iron-deficiency anemia, malnutrition

stimulants	catarrhal conditions, nausea and emesis	active inflammation, hyperacidity	
carminatives	colic, spasm, bloating, gas, flatulence	gastric reflux	
bulk laxatives	occasional constipation	chronic constipation, iron- deficiency anemia, malnutrition, cereal grain intolerance (allergy)	
aperients and laxatives	atonic constipation	constipation with mucosal inflammation, diarrhea	
antispasmodics	colic, spasm, bloating, gas, flatulence, diarrhea	none; some antispasmodics are potentially toxic, e.g. belladonna root	
hepatics and cholagogues	bilious dyspepsia, gall bladder disease, pancreatic insufficiency, steatorrhea, constipation, hepatic pain, hepatomegaly	use with caution in cholelithiasis	
antimicrobials	bacterial or fungal infections, diarrhea	overuse may unfavorably alter the gut ecology; some antimicrobials are potentially toxic	
antihelminthics	abdominal bloating and malabsorption, presence of eggs or parasites in stool	some antihelminthics may be toxic; always use with aperients and laxatives	

Table 2: Herbal actions, indications and contraindications

Excess and Deficiency

One of the innovations of physiomedicalism was to expand upon Thomson's relatively simple ideas and incorporate them within the Brunonian concept²⁰ of sthenia and asthenia, or excess and deficiency, respectively. In recent years this concept of two primary states and which herbs act to counter them has been further clarified in the work of herbalist Michael Moore, whose text *Herbal Energetics in Clinical Practice* is particularly useful in this regard. In this text, Moore states that the term 'excess' refers to the heightened metabolic activity of a particular organ system, "usually from hormonal or neurologic causes" (2002). Moore takes care to point out that an excess state in one system will typically rob the vital energy from other systems in order to sustain this state. Thus an excess state of one organ system is typically conjoined with a deficiency state within another.

To illustrate this concept, let us consider the musculoskeletal system in an excess state, expressing signs and symptoms that include muscular tightness, spasm, inflammation, and pain. When this imbalance happens, Physiomedical principles suggest that it comes at the expense of the viscera, which suffers from impaired stimulus and blood flow. Measures are thus taken to correct this imbalance by using by using herbs to activate visceral function, including digestive stimulants and bitter cholagogues. In contrast, the medical treatment of joint inflammation and muscle pain relies upon powerful anti-inflammatory agents, muscle-relaxants, and sedatives. These drugs exert their properties by inhibiting or modifying certain metabolic or neurological pathways, such as cyclooxygenase or the opioid receptor, and are often toxic to the gut and liver, and create issues with dependency. Such an approach confuses the cause of the imbalance with the mechanism of disease, and completely ignores and even worsens the underlying issue of impaired digestive function.

²⁰ Brunonian or "Brownian" referring to the work of Scottish physician John Brown, outlined in his 1780 publication *Elementa Medicinae*.

A vital 'deficiency' in an organ system refers to a decrease in the overall metabolic activity of a particular organ system. Physiomedicalists often referred to this as an 'atonic' condition, typically caused by insufficient stimulation, but this deficiency could also relate to some kind of inherited weakness, the accumulation of environmental and drug toxins, chronic infection, or nutritional deficiency. According to Physiomedical principles, a true deficiency state can only be addressed through trophorestoration – long term measures to rejuvenate a weakened organ system – whereas atonic states are corrected by proper stimulation.

According to Michael Moore, most deficiency states:

"... derive from the necessary diverting of energy to other organs or functions. This may result from the lifelong accommodations between inherited organ strengths and weaknesses. Most frequently it is caused by the habitual and learned induction of stress or emergency responses in the nervous and endocrine systems as a lazy or even necessary means of summoning a usable substitute for missing motivation, creativity, emotional health, passion, or psychic energy. We all use these auto-induction stress devices. It isn't always possible to control our lives, to always have the energy to do what needs doing, to always WANT to do what is necessary...and sometimes Shit Happens. Some of us, however, become too reliant on jump-starting ourselves with emergency stress measures, on the rolodex of fears, frustrations and angers that can be thumbed through to induce a knee-jerk twitch of adrenalin. Since the first symptom of metabolic imbalance is usually increased central nervous system irritability, some of us may prefer to stay just a little sick or autotoxic, if only to push the stress button more easily" (2002, 8).

Upper digestive tract: normal function

The gastrointestinal tract (GIT) can be divided into two basic parts: the upper digestive tract, including the mouth, pharynx, stomach and small intestine, and the lower digestive tract, consisting of the colon. These two parts of the digestive tract are separated largely because their physiological activities are distinctly different.

Activities in the upper digestive tract are initiated by salivary secretion in the mouth and the stimulation of neuroendocrinal reflexes that prime the secretions of the stomach, liver, pancreas and small intestine. Saliva is secreted in response to a variety of stimuli, including the sight, smell and even the sound of food, as well as the actual act of chewing and tasting in the mouth. Saliva is a partially mucoid secretion that while assisting with the first stages of carbohydrate and fat digestion, also helps to lubricate the food as it passes through the pharynx into the stomach.

As the food enters into the stomach and begins to fill the stomach, stretch receptors in the stomach wall stimulate parasympathetic fibers that promote the first waves of peristalsis and the flow of gastric juices. This process is assisted by chemoreceptors that detect an increase in pH from salivary secretions, as well as the hormone gastrin, and paracrine histamine, which

are released by certain cells in the gastric mucosa to upregulate digestive secretions. Gastrin and histamine also assist in partially closing off the lower esophageal sphincter, allowing the stomach to thoroughly mix the food with gastric secretions to form chyme.

As the chyme is slowly squirted into the duodenum through the pyloric sphincter, stretch receptors in the duodenum, as well as the presence of fatty acids and sugars in the chyme, stimulate the release of gastric inhibitory polypeptide, secretin, and cholecystokinnin. Generally speaking, these hormones act to delay gastric emptying and slow down peristalsis, while simultaneously stimulating the secretions of the liver, gall bladder and pancreas. In turn, these secretions, as well as the brush border enzymes of the small intestine, act upon each macronutrient to break it down into its constituent parts: fats are emulsified by bile, and carbohydrates and proteins by the secretions of the gut, they are absorbed into the bloodstream by a variety of processes, and enter into portal circulation to be transported to the liver for further processing. Fats however are broken down and suspended as chylomicrons, absorbed into the lymphatic system, and then transported through the subclavian vein into the blood, and then the liver.

Upper digestive tract: deficiency symptoms and treatment

The patient with an upper GIT deficiency typically complains of a dry mouth and throat, and often has a history of dental problems including receding gums from diminished salivary production. The tongue is typically coated and dry upon rising, and the appetite is poor, with bloating and a sense of fullness after meals that gets worse throughout the day. In most cases patients prefer to skip breakfast, eating small, frequent meals later in the day, relying on lighter carbohydrate-rich foods. Proteins and fats are usually poorly digested because of the lack of gastric stimulus, a dynamic that usually extends to the colon with symptoms of constipation, or loose motions with putrefaction and steatorrhea. Such people will often choose vegetarianism if exposed to it, and may feel better eating in this fashion, but over the long term a protein-deficient diet often leads to diminished gastric acid production and an upper GIT deficiency in a vicious cycle. As a result, such patients also end up being very sensitive to a wide variety of foods, and often complain of multiple food sensitivities or allergies. Moore states that regular, excessive alcohol consumption can also induce deficiency symptoms, and that some cigarette (and marijuana) smokers often use these drugs as a stimulant to upper GI activities (2002, 10). This dynamic of deficiency counts for most problems with digestion, rather than excess states.

Herbs to stimulate

Therapy in herbal medicine for an upper GI deficiency rests primarily upon the stimulation of gastric function by stimulating the bitter reflex on the back of the tongue, and by stimulating mucus membrane secretion, increasing blood supply to the viscera. It is important to visually apprehend the food, to fully activate the sensory nerves, which in turn activates the first stages of digestion. In some cases additional measures need to be taken to initiate parasympathetic stimulation, retraining the nervous habit of relying upon fight or flight mechanisms to get them through the day.

- bitter stimulants, e.g. gentian (*Gentiana lutea* root), goldenseal (*Hydrastis canadensis* root/rhizome), barberry (*Berberis vulgaris* root)
- vasodilators, e.g. ginger (*Zingiber officinalis* rhizome), prickly ash (*Zanthoxylum americanum* bark), cayenne (*Capsicum annuum* fruit)
- parasympathomimetics, e.g. skullcap (*Scutellaria lateriflora leaf*), milky oat (*Avena sativa seed*), passionflower (*Passiflora incarnata herb*)

Upper digestive tract: excess symptoms and treatment

The signs and symptoms of upper gastrointestinal excess are a moist mouth with abundant salivary secretion, and the rapid evacuation of the bowels first thing in the morning without any kind of stimulus. Upon observation the tongue may appear to be pointed, with the tip and the sides of the tongue appearing red and/or swollen. The appetite is usually quite strong, with a preference for fatty and protein-rich foods. In some cases there may be symptoms of nausea, especially in the morning, or when a meal is delayed or missed altogether due to the release of gastric secretions. Additional symptoms of GIT excess include irritability, burning sensations, colic, and loose motions.

<u>Herbs</u> to relax

The primary class of herbal medication used to down-regulate gastrointestinal functions are typically astringent in nature, acting upon the mucosa as a local vasoconstrictor. Common examples include tea and coffee, usually consumed after meals, or in India, the chewing of betel nut. Demulcents are called for in irritability and symptoms of burning sensation, and act to protect the mucosa from excessive acid secretion. Likewise, for the colic that can accompany upper GI excess antispasmodics are indicated, to relax and normalize hyperperistalsis.

In general, gastrointestinal excess is usually a constitutional manifestation, found in the kind of people that claim to have an "iron" stomach, who have consistently strong appetites and can eat just about anything. In some cases, however, and for short periods of time, the nervous system may need to be addressed directly, down-regulating parasympathetic activities. This approach is ultimately suppressive however, and long-term measures are directed towards constitutional aspects, using relaxing nervines and gentle antispasmodics to pacify the hyperactivity and irritability of the digestive tract. As Moore points out, it is far easier to accidentally over-stimulate than suppress this state, so ensure that the herb chosen will not stimulate gastrointestinal function (2002, 10). Sympathomimetics such belladonna that contain potentially toxic tropane alkaloids may also be indicated in acute cases, but should be used with exceptional caution, in very small doses, and only for short periods of time.

- astringents, e.g. cranesbill geranium (*Geranium maculatum* root), oak (*Quercus alba* bark), goldenseal (*Hydrastis canadensis* root/rhizome)
- demulcents, e.g. slippery elm (*Ulmus fulva* inner bark), marshmallow (*Althaea officinalis* root), licorice (*Glycyrrhiza glabra* root)

- antispasmodics, e.g. wild yam (*Dioscorea villosa* root), fennel (*Foeniculum vulgare* seed), hops (*Humulus lupus* strobile)
- sympathomimetics, e.g. belladonna (*Atropa belladonna* root/leaf), henbane (*Hyoscyamus niger* root/leaf), datura (*Datura stramonium* root/leaf)

Lower digestive tract: normal function

The passage of chyme into the colon is regulated by the actions of the ileocecal sphincter, which in turn is regulated by the activities of the stomach and the secretion of gastrin, as well as the stretch receptors in the small intestine. Once the chyme is deposited into the colon, a mechanism called haustral churning allows each haustra to accumulate the chyme until it is completely distended, before it is passed along to the next haustrated segment of the colon by peristalsis. The chyme is moved along the colon in this way until the transverse colon, when an event called mass peristalsis propels the chyme into the descending colon, driving the contents into the rectum. The movement of materials through the colon is in large part stimulated by gastric emptying. Thus many people will observe that a bowel movement is often initiated a short time after eating. The defecation reflex is under control of the parasympathetic nervous system, but the actual evacuation of feces is under conscious control. Too much conscious control, however, and the natural autonomic defecation reflux becomes weaker over time, leading to chronic constipation.

For years the importance of the colon in human health has been underestimated by modern medicine. Technically, the colon isn't very important to the process of digestion, responsible only for the absorption of water and small amounts certain B vitamins, vitamin K, and electrolytes. Unlike the stomach and small intestine, the colon produces no enzymes, and only secretes mucus to lubricate the feces. It is for this convenient reason that the surgical resection of the colon is still considered a cure for inflammatory bowel disease, despite the impact to quality of life. Likewise, the appendix that hangs down from the cecum is often stated as being a vestigial organ that has no intrinsic importance.

While seemingly unimportant, the colon and appendix both represent importance places of interface between our human cells and the body's microbiome. After the food as been properly digested by the stomach and small intestine, the chyme itself doesn't comprise much volume. But when it is excreted through the ileocecal spincter into the cecum of the colon, it is inoculated by the awaiting bacteria, and through the process of haustral churning is fermented (and putrefied in the presence of proteins) into the feces. As such, by the time it is excreted from the body, approximately 60-80% of the fecal volume is bacteria. This increase in fecal bulk and the gases produced through fermentation helps propel the feces through the large intestine for elimination, and alterations in fecal volume and gas production are usually good indications of unfavorable ecological changes in the digestive tract, generally referred to by the term dysbiosis – or "wrong of life."

The process of bacterial fermentation also generates nutrients that are vital to the health of the colonic cells, such short-chain fatty acids (e.g. acetic acid, propionic acid, butyric acid), paid as a kind "rent" by the colonic bacteria for occupying what otherwise seems to be an

unimportant organ. Not only do these commensal bacteria provide a source of nutrients, by interacting with toll-like receptors on the colonic cell surface, they enhance the ability of colonic epithelial surface to withstand injury, and help to promote healing.

Lower digestive tract: deficiency symptoms and treatment

The symptoms of a lower GIT deficiency are characterized by constipation, with poor stimulation of colonic reflexes, prolonged transit time, and the excessive dehydration of the feces. Very often examination of the tongue will show a creamy yellow or brown-colored region at its root. In almost all cases a lower GIT deficiencies originates with an upper GIT deficiency. The improper digestion of protein by the stomach favors the growth of potentially pathogenic bacteria that putrefy it, resulting in the production of neurotoxic metabolites including skatole and indole that are absorbed into the bloodstream. Likewise, excess sugar as well as flour product consumption promotes ecological changes that favor the pathogenic growth of opportunistic organisms such as *Candida albicans*. Any kind of alteration of the normal colonic flora also causes a disruption of toll-like receptor signaling, compromising the ability of the intestinal surface to withstand insult and repair damage. Dysbiotic changes that arise in the colon results in moderate inflammation of the cecum and appendix, comprising the ileocecal sphincter, leading to the retrograde flow of bacteria into the ileum, i.e. small intestine bacterial overgrowth (SIBO).

Impaired hepatic function also plays a role in lower GIT deficiency. A liver that is relatively overwhelmed by toxic metabolites (of endogenous or xenobiotic origin) or that is under stimulated (e.g. from a lack of bitter taste) will tend to produce a backlog in portal circulation. As a result blood tends to pool in the lower pelvic cavity, congesting the veins and especially the hemorrhoidal tissues. Thus in the treatment of a lower GIT deficiency liver function must also be simultaneously stimulated. In some cases a GIT deficiency state is reflective of a relative thyroid deficiency, and a symptom of generalized coldness (e.g. hypothyroidism). A lower GIT deficiency can also be a manifestation of intense feelings of fear and anxiety that been internalized, e.g. post-traumatic stress disorder.

Herbs to stimulate

The primary treatment in lower GIT deficiency is to enhance the digestive secretions of the upper GIT, liver and pancreas, support fecal lubrication, and correct the colonic flora. In acute cases it may be necessary to provide direct stimulus with the use of aperients and laxatives, but these are short term measures only. Given the connection between emotions such as fear and anxiety and deficient colonic function, nervine trophorestoratives should also be considered.

- bitter cholagogues, e.g. gentian (*Gentiana lutea* root), yellowdock (*Rumex crispus* root), barberry (*Berberis/Mahonia spp.* root)
- vasodilators, e.g. ginger (*Zingiber officinalis* rhizome), prickly ash (*Zanthoxylum americanum* bark), cayenne (*Capsicum annuum* fruit)

- demulcents, e.g. slippery elm (*Ulmus fulva* inner bark), marshmallow (*Althaea officinalis* root), licorice (*Glycyrrhiza glabra* root)
- laxatives, e.g. cascara (*Rhamnus purshianus* wood), senna (*Cassia angustifolia* pod), turkey rhubarb (*Rheum palmatum* root)
- thyrostimulants, e.g. barberry (*Berberis vulgaris* root), seaweed (e.g. *Fucus vesiculosus*, *Nereocystis luetkeana*, *Palmaria palmata*), cayenne (*Capsicum annuum* pods), blue flag rhizome (*Acorus calamus* rhizome)
- prebioitcs, e.g. beet root, chicory root, psyllium husk, inulin powder
- probiotics, e.g. live culture foods, *Lactobacillus*, *Bifidobacterium*
- nervine trophorestoratives, e.g. American ginseng (*Panax quinquefolium* root), skullcap (*Scutellaria lateriflora* leaf), milky oat (*Avena sativa* seed), passionflower (*Passiflora incarnata* herb)

Lower digestive tract: excess symptoms and treatment

The symptoms of a lower GIT excess are seen as the rapid transit of chyme through the digestive tract, a heightened gastric reflex, and frequent loose motions. There may be symptoms of colon spas and rectal irritability. The bowel movement appears more fully formed in the first portions of the material, but becomes progressively moist and liquid towards the end (Moore 2002, 12). The color of the bowel movement in excess states will range from normal to dark brown, and can even be greenish in color from excess gastric acid production (excluding vegetable fiber).

<u>Herbs</u> <u>to</u> <u>relax</u>

The primary approach to a lower GIT excess is to down-regulate the parasympathetic activities of the gut. As a result of this transit time will be slowed, and the smooth muscles in the colon, bile and pancreatic ducts will begin to relax. Astringents may also be indicated, especially where there is diarrhea or excessive mucus secretions. In many cases a GIT excess state is reflective of a relative thyroid excess (Moore 2002, 12), requiring the use of thyrodepressant herbs. In a similar vein, lower GIT excess can be related to the internalization of feelings related to aggression and anger, which benefit from the use of relaxing nervines.

- antispasmodics, e.g. wild yam (*Dioscorea villosa* root), fennel (*Foeniculum vulgare* seed), hops (*Humulus lupus* strobile)
- astringents, e.g. cranesbill geranium (*Geranium maculatum* root), oak (*Quercus alba* bark), goldenseal (*Hydrastis canadensis* root/rhizome), *CapsicumCapsicum*
- thyrodepressants, e.g. bugleweed (*Lycopus virginicus* herb), motherwort (*Leonurus cardiaca* herb)
- relaxing nervines, e.g. linden (*Tilia cordata* flower), skullcap (*Scutellaria lateriflora* leaf), passionflower (*Passiflora incarnata* herb)

Gastrointestinal infection and ecological status

As we have established, colon function is largely dependent upon the proper health of the microbiome. In addition, we have learned that diet is among the most important factors in the maintenance of this ecology, and thus where dysbiosis exists, dietary measures are always a key to restore ecological health. Apart from the diet itself, the microbiome is affected by other factors such as seasonal change, travel, hygiene, and contamination, exposing our GIT to an array of pathogenic organisms including helminths (worms), protozoans, bacteria, fungi, and viruses. The primary defense against these incursions is the strength of digestion, including the secretion of gastric HCl that can effectively denature these organisms before they can influence the microbiome. As a result, there will be a greater tendency to intestinal infection in any kind of GIT deficiency state, and prophylactic measures should be taken when these stressors arise. This includes measures to restore proper digestion and the use of small doses of antimicrobial herbs. For active infection, and depending on the pathogenic involved, specific classes of herbs can be chosen to limit the growth of these organisms, including:

- antibacterials, e.g. wild indigo (*Baptisia tinctoria* root), echinacea (*Echinacea angustifolia* root), goldenseal (*Hydrastis canadensis* root/rhizome)
- antifungals, e.g. pau d'arco (*Tabebuia spp.* bark), barberry (*Berberis spp.* root), western red cedar (*Thuja plicata* leaf), sweet annie (*Artemisia annua* herb)
- antihelminthics, e.g. sweet annie (*Artemisia annua* herb), black walnut (*Juglans nigra* green hull), quassia (*Quassia amara* wood)
- antivirals, e.g. St. John's wort flower (*Hypericum perforatm* flower), osha (*Ligusticum porteri/grayi.* root), yerba mansa (*Anemopsis californica* leaf)
- pre/probiotics, e.g. beet root, chicory root, inulin, *Lactobacillus, Bifidobacterium, Saccharomyces boulardii,* live culture foods

Asian Concepts of Digestive Health

Ayurveda

The simple approach to digestive disease outlined by Samuel Thomson, developed further by the later physiomedicalists, is remarkably similar to the approaches used in Ayurveda. The "heat" of the stomach referred to by Thomson is more or less synonymous with the concept of *agni* in Ayurveda. Beyond the role assigned to it by Thomson, the concept of *agni* includes metabolic functions, and so also relates to specific organs such as thyroid, liver, skeletal muscles, and the skin. In its more subtle form, *agni* represents the mind's ability to "digest" sensory information, including our ability to perceive, comprehend, and discriminate between all the different facets of experience.

Qualities and characteristics of agni

Agni is characterized by the qualities of ushna ('hot'), tikshna ('sharp'), and laghu ('light'), extending outwards from in the amashaya (stomach and small intestine) as the jatharagni. Here the jatharagni attends to the separation of food into its 'subtle essence' (sukshma rasa, which feeds the mind), its 'gross nutrient' portion (rasa, which feeds the body), and 'waste products' (kitta, including both feces and urine).

According to Ayurveda, when a *dosha* is in an increased state, the qualities that each *dosha* manifests can have negative effects upon digestion:

- In *vattika* conditions the *jatharagni* is *vishamagni*: digestion that is erratic and irregular.
- In *paittika* conditions the *jatharagni* is *tikshnagni*: extremely intense, with a strong appetite, burning sensations, and thirst.
- In *kaphaja* conditions the *jatharagni* is *mandagni* (*agnimandya*), characterized by weak digestion, poor appetite, nausea, and epigastric heaviness.

In the absence of *dosha* increase or vitiation, the *jatharagni* is *samagni*: correct, proper and normal.

Qualities and characteristics of ojas

Ojas is a bodily substance that counter-balances the activities and qualities of *agni*. There are two types of *ojas*: one called the *para ojas* (the 'superior essence'), and the called *apara ojas* (the 'inferior essence'). *Para ojas* is also referred to as the 'eight drops' (*ashtabindu*), and is an unchanging substance that is intrinsic to the manifestation of life that only dissipates upon death. In contrast, *apara ojas* maintains the strength of the body and mind, and is in a continual state of flux, dependent upon factors such as proper breathing, a healthy diet, and good digestion.

Once the ingested food is processed by *agni* the nutrient portion (*rasa*) is circulated to each of the bodily tissues (*dhatus*), nourishing and supporting their various functions. Each bodily tissue also maintains its own subset of *agni*, and some portion of the received nourishment is further refined by this *agni* into *apara ojas*. Simply referred to as *ojas*, this fluctuating aspect of the vital essence plays an important role in supporting digestive function, and just as *ojas* is dependent upon *agni*, so does *ojas* sacrifice itself to nourish *agni*. Factors that enhance the status of *ojas* include rest, sleep, breath control (*pranayama*), and meditation, whereas *ojas* is depleted by poor digestion, over-thinking, strong emotions, insomnia, physical exercise, work, toxins, disease, and sexual activity.

The status of *ojas* can be assessed by the luster of the eyes, the strength of limbs, and the function of the mind and senses. The greatest concentration of *ojas* is found in the reproductive tissue, which uses this *ojas* to create life. In health *ojas* is for the most part distributed equally all over the body, or is directed to support specific functions, such as the senses or digestive organs, when they are active. In acute disease or trauma the flow of *ojas* is blocked, and in chronic disease the flow of *ojas* gradually becomes deficient. When diminished, the lack of *ojas* (called *ojakshaya*) produces symptoms such as fear, anxiety, weakness of the senses, poor complexion, poor memory, poor concentration, and emaciation, all of which correspond to an increase in *vāta*.

Qualities and characteristics of ama

According to Ayurveda, a primary factor in digestive illness is the accumulation of *ama*, or 'undigested food,' which primarily is the result of *mandagni*. As the by-product of poor digestion, *ama* is opposite in nature to *agni*, displaying qualities such as *guru* ('heavy'), *shita* ('cold'), *snigdha* ('greasy'), *sthira* ('stable'), *picchila* ('slimy'), and *manda* ('slow'). As *ama* accumulates, it counters the light, hot, and sharp qualities of *agni* and impairs digestion further. This leads to a diminishment of *ojas*, and because *ojas* is the substrate upon which *agni* itself is nourished, a vicious cycle is established, resulting in a progressive increase in *ama* and a commensurate decrease in both *agni* and *ojas*. *Ama* thus represents a state of digestive entropy, and its accumulation over a sustained period eventually robs *ojas* and *agni* of their functionality, and hastens the degenerative process.

As they share qualities in common there is some confusion between *ama* and *kapha*, and often they are mistakenly believed to be the same thing. *Kapha* represents an aspect of disordered

homeostasis as an **endogenous** mechanism that leads to the expression its characteristic qualities. In contrast, *ama* is an **exogenous** substance derived from weak digestion (*agnimandya*), and hence, typically arises from the influence of *kapha*. Once *ama* is generated, however, it can associate with any of the *doshas* to cause their increase and vitiation. In such a state a *dosha* is said to be *sama*, or 'with *ama*', whereas in the absence of *ama*, a *dosha* is said to be *nirama*, or 'without *ama*'. Sometimes the term *ama dosha* is used more generally to describe a state in which *ama* has accumulated and is causing an imbalance.

Although *ama* is not the only cause of disease in Ayurveda, it is the most frequent cause, and thus at the outset of treatment for almost any condition the elimination of *ama* and the enhancement of *agni* is implemented, in a process called *ama pachana* (*'ama cooking'*). Quite often simply by dispelling *ama* and restoring *agni* the condition will resolve, but if the persists beyond the use of *ama pachana*, a specific line of treatment is administered to the vitiated *dosha(s)*.

Sama conditions	Nirama conditions
mucus congestion, catarrh	mucus normal, thin, clear
poor appetite	good appetite
indigestion, symptoms after eating	digestion occurs unnoticed
lethargy and lassitude after eating	energized and revitalized after eating
constipation	at least two bowel movements daily
sinking, dark-colored stools with mucus	floating, yellowish brown stools; no mucus
increased urination, urgency, frequency	normal urination
thick tongue coating	clear or thin white coating
headache	no headache
circulatory congestion, feeling of coldness,	circulation normal
numbness, tingling, neuralgia	nervous function normal
loss of strength	normal strength
joint swelling, pain, and inflammation	normal joint function
orbital edema, eyes appear dull	no orbital swelling, eyes bright/shining
symptoms worse with cold/damp weather/climates, worse at night; better with heat and dryness	health unaffected by changes in weather or climate

The following table describes the differences between *sama* and *nirama* conditions:

Table 3: Sama and nirama

In a broader context, the accumulation of *ama* is the impairment of one's ability to derive nourishment from life, be it physical, emotional, mental, or spiritual. A correctly functioning *agni* thus confers a harmonious benefit to the whole organism, with proper discrimination of the body, mind, and senses. It is important to remember that Ayurveda considers the partaking of food to be a *yagya*, or spiritual sacrifice. According to Ayurveda, *agni* is a sacrificial fire that resides within each of us, and when we consume food, our digestion becomes a catalyst that allows us to receive a great blessing of abundance. Proper digestion in Ayurveda thus depends upon the proper degree of mindfulness, as well as reverence, for this blessing obtained from the consumption of food.

Qualities and characteristics of a wholesome diet

In Ayurveda there is a clear distinction between a wholesome diet (*hita ahara*) and an unwholesome diet (*ahita ahara*), and the impact that each has upon health. As is retold in the *Charaka samhita*, during the first great medical conference held thousands of years ago in the Himalayas, there was a debate amongst the assembled sages as to the fundamental cause of disease. After each in attendance had expressed their own theories, one sage named Punarvasu Atreya gently chided his colleagues for not comprehending the importance of complete view of the subject. "It is the wholesome use of food that promotes the health of a person", says Atreya, "and that which is unwholesome is the cause of disease."

Some foods in Ayurveda are generally regarded as more or less wholesome when compared to others, but this determination is usually made in context with the needs of the patient. This takes into account factors such as climate (*deha*), the time of day and the local season (*kala*), the quantity of food (*rashi*), the quality of food (*prakriti*), the preparation of food (*karana*), food combinations (*samyoga*), the method of consumption (*upayoga*), and the health status of the recipient (*upayukta*). The underlying algorithm to understand most of these relationships is based upon the *tridosha* model, which seeks to resolve imbalances by pacifying the affected *dosha*(*s*) and restoring balance. For example, a diet comprised of mostly *vata*-reducing foods (*prakriti*) is generally followed when living in a dry climate (*deha*), when transitioning through autumn and winter (*kala*), and in patients with a *vata* imbalance (*upayukta*).

The other aspects of diet described in Ayurveda, such as the quantity of food consumed at each meal (*rashi*), how it is prepared (*karana*), the various food combinations (*samyoga*), and the method of its consumption (*upayoga*), all appear to have separate considerations, but at their core, each is based on the principles of *tridosha*. With regard to quantity (*rashi*), this is in large part a subjective determination based on how the patient feels after eating. Proper digestion requires some space in order for the food to mixed, and so it is generally advised to leave the stomach at least ¼ part empty after meals.

The subject of food preparation (*karana*) is exceptionally vast, and involves a huge variety of techniques, including steaming, boiling, stewing, frying, grilling, smoking, and fermentation. Each method of preparation has a particular influence on the *doshas*, and can be used intentionally to modulate the qualities of different food items. For example, grilling or roasting meat dehydrates the tissues, making it hard, dense, and more difficult to digest. For *mandagni* and especially in *vata* conditions it is always better to stew the meat, which serves to hydrate the tissues, making it tender, soft, and easier to digest. Likewise, fresh milk is cooling and nourishing, and helps to reduce *pitta*, but when it is fermented into yogurt serves to increase *pitta*.

Ayurveda also describes how different foods affect each other in combination (*samyoga*). For example, meat is generally not eaten with legumes in Ayurveda because it weakens digestion. In this case, the dry, light nature of the legumes is opposite in nature to the moist, dense nature of the meat, and instead of counter-balancing each other as some combinations do, creates a state of disharmony. This observation has been validated by the relatively recent discovery of naturally occurring protease inhibitors found in legumes that impair the digestion of animal proteins. Likewise, there are recommendations in Ayurveda to avoid

combinations of milk with foods such as fish, garlic, sour-tasting fruits (e.g. lime), certain legumes such as *kulattha* (horsegram), and coconut. For a more complete rendering of the concept of *samyoga* please refer to Lesson Eight of the Inside Ayurveda program.

Lastly, the concept of *upayoga* or the method of food consumption refers to a set of practices recommended by Ayurveda to support good digestion. This includes following a proper 'order' (*krama*) for the consumption of each of the six flavors (*rasa*). Generally, sweet, sour and salty flavored foods and beverages are consumed first, followed by pungent, bitter and astringent flavors. The concept of *upayoga* also refers to the proper 'procedure' (*vidhi*) for eating food, and elaborates many considerations, including proper hygiene, the temperature of the food and its relative moistness, whether or not the previous meal has been digested, and the setting in which the food is consumed. With regard to the latter, and referencing the notion described earlier that eating is a *yagya* or spiritual ritual, Ayurveda suggests that the eating environment be free of distractions, strong emotions, and other disturbances to mindful eating.

Botanicals for digestive health in Ayurveda

Similar to the approach of Physiomedicalism, Ayurveda describes a large number of therapeutic actions (*karma*), and among those that affect digestion there are many different categories. Described earlier, the fundamental measure to restore digestion in Ayurveda is encompassed by the terms *dipana* and *pachana*, referring to an ability to enkindle the digestive fire and cook the ingested food, respectively. A great many herbs combine this activity and so are called *dipanapachana*, such as Trikatu, or the 'three pungents' formulation comprised of equal parts long pepper fruit, ginger rhizome, and black pepper fruit. Likewise there are categories for other therapeutic aspects of digestive function, including sialagogues (*asyasravana*, e.g. tumburu bark), emetics (*vamana*, e.g. madana fruit), antiemetics (*chardinigrahana*, e.g. ginger rhizome), carminatives (*shulaprashamana*, e.g. ajwain seed), laxatives (*rechana*, e.g. trivrit root), astringents (*purishasangrahaniya*, e.g. kutaja root), and antihelminthics (*krimighna*, e.g. vidanga fruit).

Despite using these different categories of therapeutic action, the primary approach of using herbal medication to support digestive health is based upon the *tridosha* model. Recalling the earlier discussion of how each of *dosha* impacts digestion, there are three fundamental states of disturbance to *agni* called *mandagni* (slow digestion), *tikshnagni* (fast digestion), and *vishamagni* (irregular digestion). Understanding the *dosha*(*s*) for each type of disturbance, specific herbs and formulas can be chosen address these fundamental imbalances:

- 1. *mandagni*, i.e. heavy, slow, sluggish digestion (*kapha*)
 - symptoms: sweet taste, epigastric heaviness, nausea, orbital puffiness, burping with sweet taste and/or taste of previous meal
 - treatment: appetizing and digestive (*dipanapachana*) remedies that are bitter, pungent, and astringent in flavour; emetic therapy (*vamana karma*)
 - herbs, e.g. yavani (*Trachyspermum ammi* fruit), chavya (*Piper chaba* fruit/stem), chitraka (*Plumbago zeylanica* root), shunthi (*Zingiber officinalis* rhizome), pippali (*Piper longum* fruit), maricha (*Piper nigrum* fruit), ela (*Elettaria cardamomum* fruit), hingu (*Ferula narthex* gum), tumburu fruit (*Zanthoxylum alatum* pericarp/bark)
 - <u>Trikatu churna</u>

- 1 part shunthi (*Zingiber officinalis* rhizome)
- 1 part maricha (*Piper nigrum* fruit)
- 1 part pippali (*Piper longum* fruit)
 - Rx: 2-3 g bid-tid
- <u>Amalakyadi churna</u>
 - o 1 part amalaki (*Phyllanthus emblica* fruit)
 - 1 part chitraka (Plumbago zeylanica root)
 - o 1 part haritaki (Terminalia chebula fruit)
 - 1 part pippali (Piper longum fruit)
 - 1 part saindhava (rock salt)
 - Rx: 2-3 g bid-tid
- <u>Chitrakadi vati</u>
 - 1 part chitraka (*Plumbago zeylanica* root)
 - 1 part pippalimula (*Piper longum* root)
 - 1 part yavakshara (Hordeum vulgare, purified ash of grass)
 - 1 part swarjikshara (sodium bicarbonate)
 - 1 part sauvarchala (black salt)
 - 1 part saindhava (pink salt)
 - 1 part vida lavana (salt made from *Phyllanthus emblica* fruit)
 - 1 part samudra lavana (sea salt)
 - 1 part audbhida lavana (earthen salt)
 - o 1 part shunthi (*Zingiber officinalis* rhizome)
 - 1 part maricha (*Piper nigrum* fruit)
 - 1 part pippali (*Piper longum* fruit)
 - 1 part hing (Ferula narthex gum)
 - o 1 part ajamoda (*Trachyspermum roxburghianum* seed)
 - 1 part chavya (*Piper chaba* fruit/stem)
 - 1 part dadima (Punica granatum juice)
 - Rx: 2 pills bid-tid
- 2. *tikshnagni*, i.e. hot, sharp, fast digestion (*pitta*)
- symptoms: strong appetite, bitter taste, burning sensation in stomach, sour-tasting reflux, dizziness, thirst
- treatment: appetizing (*dipana*) remedies that are sweet, bitter, and astringent in flavour; purgative/choleretic therapy (*virechana karma*)
- herbs, e.g. dhaniya (*Coriandrum sativum* seed), daruharidra (*Berberis nepalensis* root), kumari (*Aloe vera* leaf), katuka (*Picrorhiza kurroa* rhizome), bhunimba (*Andrographis panniculatus* leaf), lavanga bud (*Syzigium aromaticum* flower bud), musta (*Cyperus rotundus* tuber/rhizome)
- Avipattikara churna
 - 44 parts trivrit (*Operculina turpethum* root)
 - 11 parts lavanga (*Syzigium aromaticum* flower bud)
 - 1 part musta (*Cyperus rotundus* tuber/rhizome)
 - 1 part vidanga (Embelia ribes seed)
 - 1 part sthula ela (Amomum subulatum fruit)

- 1 part patra (*Cinnamomum tamala* leaf)
- 1 part pippali (*Piper longum* seed)
- 1 part maricha (*Piper nigrum* seed)
- 1 part shunthi (*Zingiber officinalis* rhizome)
- 1 part amalaki (*Phyllanthus emblica* fruit)
- 1 part bibhitaki (*Terminalia bellerica* fruit)
- 1 part haritaki (*Terminalia chebula* fruit)
- 1 part vida lavana (salt made from amalaki fruit)
- 66 parts sugar
 - Rx: 2-3 g bid-tid
- <u>Punarnava asava</u>
 - 16 g shunthi (*Zingiber officinalis* rhizome)
 - 16 g pippali (*Piper longum* fruit)
 - 16 g maricha (*Piper nigrum* fruit)
 - o 16 g haritaki (*Terminalia chebula* fruit rind)
 - o 16 g darvi (Berberis aristata stem)
 - o 16 g bibhitaki (*Terminalia bellerica* fruit rind)
 - 16 g amalaki (*Phyllanthus emblica* fruit)
 - 16 g gokshura (*Tribulus terrestris* root/plant)
 - 16 g kantakari (*Solanum xanthocarpum* root/plant)
 - 16 g brihati (Solanum indicum root/plant)
 - 16 g vasaka (Adhatoda vasica root)
 - 16 g erandamula (*Ricinus communis* root)
 - 16 g katuki (Picrorhiza kurroa root)
 - 16 g punarnava (Boerhaavia diffusa root)
 - 16 g gajapippali (Scindapsus officinalis fruit)
 - 16 g pichumarda neem (Azadirachta indica stem bark)
 - 16 g guduchi (*Tinospora cordifolia* vine)
 - 16 g patola (*Trichosanthes dioica* leaf)
 - o 16 g shushka mulaka (*Raphanus sativus* root)
 - 16 g duralabha (*Fagonia cretica* root)
 - 256 g dhataki (Woodfordia fruticosa flower)
 - \circ 1.6 kg sita (sugar)
 - 320 g draksha (*Vitis vinifera* fruit)
 - \circ 800 g madhu (honey)
 - for *amlapitta* (gastric ulcer)
 - Rx: 12 24 mL
- <u>Mahatiktaka ghrita</u>
 - o 192 g saptaparna (Alstonia scholaris stem bark)
 - 192 g ativisha (Aconitum heterophyllum, purified root)
 - o 192 g shampaka (Cassia fistula fruit pulp)
 - 192 g katuka (*Picrorhiza kurroa* rhizome)
 - o 192 g patha (Cyclea peltata / Cissampelos pariera root)
 - 192 g musta (*Cyperus rotundus* rhizome/tuber)
 - o 192 g ushira (*Vetiveria zizanioides* rhizome)

- 192 g <u>Triphala</u> (equal parts *Terminalia chebula* fruit, *Terminalia bellerica* fruit, *Phyllanthus emblica* fruit)
- 192 g patola (*Trichosanthes dioica* leaf)
- 192 g nimba (Azadirachta indica stem bark)
- 192 g parpataka (Fumaria indica herb)
- o 192 g dhanvayasa (Alhagi pseudalhagi herb)
- o 192 g chandana (Santalum album heartwood)
- 192 g pippali (*Piper longum* fruit)
- o 192 g gajapippali (Piper chaba fruit)
- o 192 g padmaka (Prunus poddum heartwood)
- o 192 g ĥaridra (*Curcuma longa* rhizome)
- 192 g daruharidra (*Berberis aristata* stem)
- 192 g ugragandha (*Acorus calamus* rhizome)
- 192 g vishaka (*Citrulus cholocynthis* herb)
- 192 g shatavari (Asparagus racemosus root)
- 192 g sariva (*Hemidsemus indicus* root)
- 192 g vatsakabija (Holarrhena antidysenterica root)
- 192 g vasa (Adhatoda vasica root)
- o 192 g murva (Marsdenia tinescsima root)
- 192 g amruta (*Tinospora cordifolia* stem)
- 192 g kiratatikta (Swertia chiraita herb)
- 192 g yashtimadhu (*Glycyrrhiza glabra* root)
- o 192 g trayamana (*Gentiana kurroo* herb)
- 1.536 liters amalaki (*Phyllanthus emblica* juice)
- 768 ml ghrita (clarified butter)
 - Rx: 3-6 g bid
- 3. *vishamagni*, i.e. irregular, unstable, dry digestion (*vata*)
 - symptoms: irregular appetite, astringent taste, bloating, colic, constipation
 - treated with digestive and appetizing (*dipanapachana*) remedies that are sweet, sour, and salty in flavor; enema therapy (*vasti karma*)
 - herbs, e.g. yavani (*Trachyspermum ammi* seed), ajamoda seed (*Trachyspermum roxburghianum*), adraka (*Zingiber officinalis* rhizome), twak (*Cinnamomum zeylanicum* bark), fennel (*Foeniculum vulgare* seed), hingu (*Ferula narthex* gum), shweta jiraka (*Cuminum cyminum* seed), shatapushpa (*Anethum graveolens* seed), rock salt
 - <u>Hingwastak churna</u>
 - 1 part shweta jiraka (*Cuminum cyminum* seed)
 - o 1 part krishna jiraka (*Nigella sativa* seed)
 - 1 part saindhava (mineral salt)
 - 1 part hingu (Ferula narthex gum)
 - 1 part ajamoda seed (*Trachyspermum roxburghianum*)
 - 1 part pippali (*Piper longum* seed)
 - 1 part maricha (*Piper nigrum* seed)
 - 1 part shunthi (*Zingiber officinalis* rhizome)
 - Rx: 2-3 g bid-tid

- <u>Trikatu rasayana vati</u>
 - gandhaka (purified sulfur)
 - o jiraka (*Cuminum cyminum* seed)
 - saindhava (pink salt)
 - <u>Trikatu</u> (equal parts *Piper longum* fruit, *Piper nigrum* fruit, *Zingiber officinalis* rhizome)
 - hingu (Ferula narthex gum)
 - o lashuna (Allium sativum bulb)
 - o nimbu (*Citrus limonium* juice)
 - Rx: 2 pills bid-tid
- <u>Chandraprabha vati</u>
 - 3 g karpura (*Cinnamomum camphora* leaf)
 - 3 g vacha (Acorus calamus rhizome)
 - 3 g mustaka (*Cyperus rotundus* rhizome)
 - 3 g bhunimba (Andrographis paniculata herb)
 - 3 g guduchi (*Tinospora cordifolia* stem)
 - 3 g devadaru (Cedrus deodara wood)
 - 3 g haridra (*Curcuma longa* rhizome)
 - 3 g ativisha (Aconitum heterophyllum purified root)
 - 3 g daruharidra (Berberis nepalensis root bark)
 - 3 g pippalimula (*Piper longum* root)
 - 3 g chitraka (Plumbago zeylanica root)
 - 3 g dhaniya (*Coriandrum sativum* fruit)
 - 3 g haritaki (*Terminalia chebula* fruit)
 - 3 g amalaki (Pyllanthus emblica fruit)
 - o 3 g bibhitaki (Terminalia bellerica fruit)
 - 3 g chavya (*Piper retrofractum* stem)
 - 3 g vidanga (*Embelia ribes* fruit)
 - 3 g gajapippali (Scindapsus officinalis fruit)
 - 3 g pippali (*Piper longum* seed)
 - 3 g maricha (*Piper nigrum* seed)
 - 3 g shunthi (Zingiber officinalis rhizome)
 - 3 g swarna makshika (purified copper/iron pyrite)
 - 3 g yava kshara (*Hordeum vulgare*, calcinated grass)
 - 3 g sarjika kshara (sodium bicarbonate)
 - 3 g saindhava (pink salt)
 - \circ 3 g sauvarchala (black salt)
 - o 3 g vida lavana (salt made from *Phyllanthus emblica* fruit)
 - 12 g trivrit (Operculina turpethum root)
 - 12 g danti (Baliospermum montanum root)
 - 12 g twak (Cinnamomum zeylanicum bark)
 - o 12 g patra (*Cinnamomum tamala* leaf)
 - \circ 12 g ela (*Elettaria cardamomum* seed)
 - o 12 g vamshalochana (Bambusa arundinacea inner bark)
 - 24 g lauha bhasma (calcinated iron oxide ash)
 - \circ 48 g sita (sugar)
 - 96 g shilajatu (purified shilajit)

- 96 g guggulu (*Commiphora wightii*, purified oleo-gum resin)
 - Rx: 2 pills bid-tid

Traditional Chinese medicine

Digestive function in Traditional Chinese medicine is largely ruled by the Stomach (*wei*) and Spleen (*pi*), which are responsible for generating the Food Essence (*gu qi*) that forms the basis of the Post-Natal Essence (*hou tian zhi qi*). Like *apara ojas* in Ayurveda, the Post-Natal Essence is responsible for sustaining the energy of the body. Traditional Chinese medical theory states that the Stomach is a *yang* organ, whereas the Spleen is a *yin* organ. Based on this dichotomy, *yin* is thus an important element to counter-balance the function of the Stomach, whereas *yang* is a vital component to counter-balance the Spleen.

Stomach

The function of the Stomach in Chinese medicine is to denature the ingested food, analogous to the way a compost pile breaks down plant material, only requiring moisture and heat in the process. After this rotting and ripening process the Stomach propels the ingested food downwards into the Small Intestine (*xiaochang*), which separates out the nutrient portion of the food. After this, the nutrient portion is the transported to the Spleen for further processing, and the wastes are sent to the Large Intestine (*dachang*) to be eliminated. Similar again to Ayurveda, the Stomach is the root of health in Chinese medicine, and the first step in transforming the ingested food into the energy that feeds the body. Thus if the Stomach is in a weakened state, the *qi* cannot be maintained and will eventually be lost.

The Stomach is particularly sensitive to any irregularities in diet, and if excessively cold foods are consumed, such as raw vegetables and cold water, this has the effect of weakening *yang*, promoting symptoms such as poor appetite, loose motions, and generalized coldness. Conversely, when excessively spicy or drying foods are consumed this can promote a deficiency of *yin* within the Stomach, with symptoms such as afternoon fever, thirst, and constipation. If excessively spicy and greasy foods are consumed the result may be excess heat within the Stomach, which similarly weakens the *yin* component, but leads to specific symptoms such as epigastric burning, thirst, constant hunger, nausea, and bad breath.

If the Stomach lacks the *qi* to properly ripen the ingested food, this manifests as a Stomach *qi* deficiency, leading to a general weakening of *qi* in the body, manifesting symptoms such as poor appetite, epigastric discomfort after eating, loose motions, and weakness of the limbs. This latter syndrome is often referred to as 'food stagnation', in which the ingested food cannot be ripened, leading to symptoms such as burping, epigastric heaviness, and poor appetite. When the Stomach *qi* is 'rebellious' and flows upwards instead of downwards, usually in association with excess Stomach heat, there may be symptoms of gastric reflux, burping, hiccoughs, and vomiting.

Based on the correct identification of these various patterns, herbs and formulas can be chosen to restore balance to the Stomach, including:

- 1. Stomach *yang* deficiency:
 - herbs, e.g. gan jiang (*Zingiber officinalis* rhizome), wu zhu yu (*Evodia rutaecarpa* fruit), chuan jiao (*Zanthoxylum bungeanum* pericarp), ding xiang (*Syzigium aromaticum* flower bud), gao liang jiang (*Alpinia officinarium* rhizome), bi ba (*Piper longum* fruit), hu jiao (*Piper nigrum* fruit)
 - Li Zhong Wan (Regulate Middle Pill)
 - 9 g gan jiang (Zingiber officinalis rhizome)
 - o 9 g ren shen (Panax ginseng root)
 - 9 g bai zhu (Atractylodes macrocephala root)
 - 9 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - granules, Rx: 2-4 g bid
 - <u>Wu Zhu Yu Tang (Evodia Decoction)</u>
 - 9-12 g wu zhu yu (Evodia rutaecarpa seed)
 - 18 g gan jiang (*Zingiber officinalis* rhizome, recently dried)
 - 9 g ren shen (Panax ginseng root)
 - o 12 pieces da zao (Ziziphus jujuba fruit)
 - decoction, Rx: 200 mL bid
- 2. Stomach *yin* deficiency
 - herbs, e.g. sa shen (Adenophora verticillata root), mai men dong (Ophiopogon japonicus root), shi hu (Dendrobium nobile stem), yu zhu (Polygonatum odoratum root)
 - <u>Yi Wei Tang (Benefit Stomach Decoction)</u>
 - 9 g sha shen (Adenophora verticillata root)
 - 15 g mai men dong (*Ophiopogon japonicus* root)
 - o 15 g sheng di huang (Rehmannia glutinosa root)
 - 4.5 g chao yu zhu (*Polygonatum odoratum*, dry-fried root)
 - 3 g bing tang (rock sugar)
 - Rx: 200 mL bid-tid
 - <u>Mai Men Dong Tang (Ophiopogon Decoction)</u>
 - 15-64 g mai men dong (Ophiopogon japonicus root)
 - 9 g ren shen (Panax ginseng root)
 - 6-15 g jing mi (non-glutinous rice)
 - o 12 piece da zao (Ziziphus jujuba fruit)
 - 6 g gan cao (Glycyrrhiza uralensis root)
 - o 6-9 g zhi ban xia (*Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
 - decoction, Rx: 200 mL qid
 - granules, Rx: 2-4 g bid-tid
- 3. Stomach Fire
 - herbs, e.g. ge gen (Pueraria lobata root), sheng di huang (Rehmannia glutinosa root), zhi mu (Anemarrhena asphodeloides rhizome), gua lou ren (Trichosanthes kirilowii seed), huang lian (Coptis chinensis rhizome), tian hua fen (Trichosanthes kirilowii root)
 - Ban Xia Xie Xin Tang (Pinellia Decoction to Drain the Epigastrum)
 - 9-12 g zhi ban xia (*Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
 - 9 g gan jiang (*Zingiber officinalis* rhizome, recently dried)

- 9 g huang qian (Scutellaria baicalensis root)
- 3 g huang lian (*Coptis chinensis* rhizome)
- 9 g ren shen (*Panax ginseng* root)
- 12 pieces da zao (*Ziziphus jujuba* fruit)
- 9 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - harmonizes Stomach, directs Rebellious *qi* downwards, disperses distension
 - granules, Rx: 3-4 g bid-tid
- Mai Men Dong Tang (Ophiopogon Decoction)
 - o decoction, Rx: 200 mL qid
 - o granules, Rx: 2-4 g bid-tid
- 4. Stomach *qi* deficiency (Food Stagnation)
 - herbs, e.g. shan zha (*Crataegus pinnatifida* fruit), mai ya (*Hordeum vulgare*, fermented sprout), gu ya (*Oryza sativa* fermented sprout), lai fu zi (*Raphanus sativus* seed), chen pi (*Citrus reticulata* peel), pei lan (*Eupatorium fortunei* herb), cao dou kou (*Alpinia katsumadai* seed)
 - Bao He Wan (Preserve Harmony Pill)
 - 9-15 g shan zha (Crataegus pinnatifida fruit)
 - o 9-12 g shen qu (massa fermentata, medicated leaven)
 - \circ 6-9 g lai fu zi (Raphanus sativus seed)
 - 6-9 g chen pi (*Citrus reticulata* peel)
 - o 9-12 g zhi ban xia (*Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
 - 9-12 g fu ling (*Poria cocos* fruiting body)
 - 3-6 g lian qiao (Forsythia supensa seed)
 - tea pills, Rx: 5-8 pills bid-tid
 - granules, Rx: 3-5 g bid-tid
 - Xiang Sha yang Wei Pian (Saussurea Amomum Nourish Stomach Pill)
 - o 1.5 g ren shen (Panax ginseng root) or dang shen (Codonopsis pilosula)
 - 3 g bai zhu (*Atractylodes macrocephala* rhizome)
 - 2.4 g fu ling (*Poria cocos* fruiting body)
 - 2.4 g cang zhu (*Atractylodes lancea* rhizome)
 - 2.4 g jiang zhi chao hou po (*Magnolia officinalis*, fried with ginger)
 - 2.4 g chen pi (*Citrus reticulata* peel)
 - 2.4 g chao xiang fu (*Cyperus rotundus*, dry-fried rhizome)
 - 2.1 g bai dou kou (*Amomum kravanh* seed)
 - 1.5 g mu xiang (Saussurea costus root)
 - 2.4 g sha ren (Amomum villosum seed)
 - 3 g Angelica sinensis
 - 1.5-3 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - 1.5-3 g da zao (Ziziphus jujuba fruit)
 - tablets, Rx: 3-5 tabs bid-tid
 - granules, Rx: 2-4 g bid-tid
- 5. Rebellious Stomach qi
 - herbs, e.g. sha ren (Amomum villosum seed), mu xiang (Saussurea costus root), ding xiang (Syzygium aromaticum flower buds)

- Xiao Ban Xia Tang (Minor Pinellia Decoction)
 - 9 g zhi ban xia (*Pinellia ternata*, prepared rhizome)
 - o 9 g sheng jiang (Zingiber officinalis freshly dried rhizome)
 - harmonizes Stomach, descends Rebellious qi, stops vomiting
 - Rx: 200 mL bid
- <u>Ding Xiang Shi Di Tang (Clove and Persimmon Calyx Decoction)</u>
 - 9-12g chen pi (*Citrus reticulata* peel)
 - 9-12 g zhu ru (Bambusa brevifolia shavings)
 - 3 g zhi ban xia (*Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
 - 18 g sheng jiang (*Zingiber officinalis* freshly dried rhizome)
 - 15 g gan cao (Glycyrrhiza uralensis root)
 - 30 pieces da zao (Ziziphus jujuba fruit)
 - augments *qi*, warms middle, descends Rebellious *qi*, relieves hiccup
 - decoction, Rx: 200 mL bid
 - granules, Rx: 3-4 g bid-tid

<u>Spleen</u>

After digestion by the Stomach, the nutrient portion of the ingested food is passed along to the Spleen from the Small Intestine (*xiao chang*). Within the Spleen, the digesting food is refined and rendered into the vital Essence (*qi*), which is then passed along to the rest of the body through the channels of circulation. Energetically, the Spleen manifests the intellect, referring to the ability to think, memorize and concentrate. It is said that the Spleen opens into the mouth, and thus when the lips are moist and pink, the Spleen is functioning correctly.

The general function of *qi* in the body is to lift and raise, and in particular, this is the job of the Spleen. When the *qi* of the Spleen is deficient, from mental strain, a poor diet, or eating too much, the result is an impairment in the upward-moving force, causing symptoms such as poor appetite, abdominal distension after meals, fatigue, weakness, and loose stools. In cases of long standing Spleen *qi* deficiency this may manifest as a 'sinking' of the Spleen *qi*, similar in nature to the symptoms of a Spleen *qi* deficiency, with additional symptoms of pelvic heaviness, frequent urination, as well as rectal, vaginal, or uterine prolapse.

Similar to Spleen *qi* deficiency is when the Spleen is invaded by dampness, caused by cold and wet weather, or foods that are too cold and wet in nature. When the Spleen cannot fully transform the food into *qi* the result is dampness accumulating in the *fei* ('lungs'), manifesting as catarrh and congestion. A Spleen *yang* deficiency has a similar etiology, but is specifically caused by too much cold in the diet, which weakens *yang* and promotes coldness in the body.

Based on the correct identification of these various patterns, herbs and formulas can be chosen to restore balance to the Spleen, including:

- 1. Spleen *qi* deficiency
 - herbs, e.g. ren shen (*Panax ginseng* root), dang shen (*Codonopsis pilosula*), tai zi shen (*Pseudostellaria heterophylla* tuber), huang qi (*Astragalus membranaceus* root), shan yao (*Dioscorea opposita* rhizome), bai zhu (*Atractylodes macrocephala* rhizome), da zao (*Ziziphus jujuba* fruit), gan cao (*Glycyrrhiza uralensis* root), huang jing (*Polygonatum sibiricum* rhizome)

- <u>Si Jun Zi Tang (Four Gentlemen Decoction)</u>
 - 3 g ren shen (*Panax ginseng* root) or dang shen (*Codonopsis pilosula*)
 - 4.5 g bai zhu (*Atractylodes macrocephala* rhizome)
 - 3 g fu ling (*Poria cocos* fruiting body)
 - 3 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - decoction, Rx: 200 mL bid
 - tablets, Rx: 4-6 tabs bid-tid
 - granules, Rx: 2-3 g bid-tid
- Liu Jun Zi Tang (Six Gentlemen Decoction)
 - 3 g ren shen (Panax ginseng root) or dang shen (Codonopsis pilosula)
 - 4.5 g bai zhu (*Atractylodes macrocephala* rhizome)
 - 3 g fu ling (*Poria cocos* fruiting body)
 - 3 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - 3 g chen pi (*Citrus reticulata* peel)
 - 4.5 g zhi ban xia (*Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
 - decoction, Rx: 200 mL bid
 - tablets, Rx: 2-3 tabs bid-tid
 - granules, Rx: 2-4 g bid-tid
- 2. Sinking Spleen qi
 - herbs, e.g. dang shen (Codonopsis pilosula), chai hu (Bupleurum falcatum)
 - Bu Zhong Yi Qi Wan (Tonify Middle Augment Essence Decoction)
 - o 12-24 g huang qi (Astragalus membranaceus root)
 - 9-12 g dang shen (Codonopsis pilosula)
 - o 9-12 g bai zhu (Atractylodes macrocephala rhizome)
 - 3-6 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - 9-12 g dang gui (Angelica sinensis root)
 - 6-9 g chen pi (*Citrus reticulata* peel)
 - 3-6 g sheng ma (*Cimicifuga heracleifolia* root)
 - 3-9 g chai hu (Bupleurum falcatum root)
 - decoction, Rx: 200 mL bid-tid
 - tea pills, Rx: 8 pills bid-tid
 - granules, Rx: 2-4 g bid-tid
 - <u>Gui Pi Tang (Restore Spleen Decoction)</u>
 - 3-6 g ren shen (*Panax ginseng* root)
 - 9-12 g chao huang qi (*Astragalus membranaceus*, dry-fried root)
 - o 9-12 g bai zhu (Atractylodes macrocephala rhizome)
 - 9-12 g fu ling (*Poria cocos* fruiting body)
 - o 9-12 g chao suan zao ren (Ziziphus spinosa, dry-fried seed)
 - 6-9 g long yan rou (*Euphoria longan* fruit)
 - 3-6 g mu xiang (Saussurea lappa rhizome)
 - 3-6 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - 6-9 g dang gui (Angelica sinensis root)
 - 3-6 g zhi yuan zhi (*Polygonatum odoratum*, steamed root)
 - decoction, Rx: 200 mL bid-tid

- tea pills, Rx: 8 pills bid-tid
- granules, Rx: 2-4 g bid-tid
- 3. Spleen Dampness
 - herbs, e.g. chen pi (*Citrus reticulata* peel), sha ren (*Amomum villosum* seed), shi chang pu (*Acorus gramineus* rhizome), cao dou kou (*Alpinia katsumadai* seed), cao guo (*Amomum tsaoko* fruit), fu ling (*Poria cocos* fruiting body)
 - Ping Wei San (Calm Stomach Powder)
 - o 12-15 g chao cang zhu (*Atractylodes macrocephala* rhizome, dry fried)
 - 9-12 g jiang zhi chao hou po (*Magnolia officinalis*, fried with ginger)
 - 9-12 g chen pi (*Citrus reticulata* peel)
 - 3-6 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - powder, Rx: 6-9 g bid-tid
 - granules, Rx: 2-4 g bid-tid
 - Liu He Tang (Harmonize Six Decoction)
 - 1 part ren shen (*Panax ginseng* root)
 - 1 part sha ren (Amomum villosum seed)
 - 1 part zhi ban xia (*Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
 - 1 part xing ren (*Prunus armeniaca* seed)
 - 2 parts bai zhu (*Atractylodes macrocephala* rhizome)
 - 2 parts huo xiang ye (*Agastache rugosa* herb)
 - 2 parts bian dou (Dolichos lalab seed)
 - 2 parts chi fu ling (*Poria cocos*, red portion of fruiting body)
 - 2 parts mu gua (Chaenomeles lagenaria fruit)
 - 4 parts jiang zhi chao hou po (*Magnolia officinalis* bark, fried with ginger)
 - 4 parts xiang ru (Elsholtzia splendens herb)
 - 1 part zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - decoction, Rx: 12 g powder decocted with one piece da zao and three slices ginger, given in divided doses
 - granules, Rx: 2-4 g bid-tid
- 4. Spleen *yang* deficiency
 - herbs, e.g. bai dou kou (*Amomum kravanh* seed), wu yao (*Lindera aggregate* root), rou gui (*Cinnamomum cassia* bark), fu long gan (soot from fire pit), ding xiang (*Syzygium aromaticum* flower bud), yi zhi ren (*Alpinia oxyphylla* seed), rou dou kou (*Myristica fragrans* seed)
 - Fu Zi Li Zhong Pian (Aconite Regulate Middle Pill)
 - 1 part zhi fu zi (Aconitum carmichaeli, processed lateral roots)
 - 1 part pao jiang (*Zingiber officinalis* rhizome, recently dried)
 - 1 part ren shen (Panax ginseng root)
 - 1 part bai zhu (*Atractylodes macrocephala* rhizome)
 - 1 part zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - pills made with honey, Rx: 2-3 g, bid
 - <u>Da Jian Zhong Tang (Major Construct Middle Decoction)</u>
 - hua jiao (*Zanthoxylum bungeanum* pericarp)

- gan jiang (*Zingiber officinalis*, dried rhizome)
- ren shen (Panax ginseng root)
- yi tang (barley malt)
 - decoction, Rx: 200 mL bid
 - granules, Rx: 3-5 g bid

Liver and Gall Bladder

The Liver (gan) is responsible for ensuring the proper flow of *qi* in the body. It serves this purpose by 'dredging' out impurities in the blood that impairs the flow of *qi*, discharging these wastes as the bilious secretions of the Gall Bladder (*dan*). When the Liver *qi* is stagnant the Stomach *qi* cannot descend and the Spleen *qi* cannot rise, resulting in abdominal distension, poor appetite, belching, borborygmi, and loose motions. When the Liver *yang* rises upwards, as can occur in a Stomach *yin* deficiency, or from long-standing Liver *qi* stagnation, the result may be a bitter taste in the mouth, constipation, and emotional symptoms such as sudden outbursts of anger. In cases of Spleen *qi* deficiency the result may be Liver-Gall Bladder (*gandan*) damp heat, manifesting as epigastric fullness and pain, a bitter taste in the mouth, nausea, jaundice, and a loss of appetite.

Please refer to **The Inner Alchemist: Hepatobiliary System** for a complete review of Liver-Gall Bladder patterns in Chinese medicine.

Small Intestine and Large Intestine

The Small Intestine (*xiao chang*) and Large Intestine (*da chang*) are hollow organs that serve to transport the digesting food through the digestive tract, but each also plays a role in digestive activity. After the Stomach completes its process, the pure nutrient portion is passed along to the Spleen, and what is left over is passed along to the Small Intestine. The Small Intestine then refines this into a pure fluid that is absorbed and directed to the Spleen, and an impure fluid that is directed downwards to the Large Intestine. If the Liver *qi* is stagnant or if there is excess cold the result can be *qi* stagnation of the Small Intestine. This causes the Small Intestine to become spasmodic, knotted, and painful, causing lower back and groin pain. For herbs and formulas used to address Small Intestine *qi* stagnation, please refer to module two, under Liver *qi* stagnation.

After receiving the impure fluids from the Small Intestine, the Large Intestine then extracts any remaining water to supplement the bodily fluids, and eliminate the remaining portion as feces. Unlike the Small Intestine, there are several patterns of Large Intestine dysfunction. Heat or damp-heat of the Large Intestine is often the result of a diet containing too much spicy and greasy foods, and thus is an extension of Stomach heat, but it can also be caused by intense emotions. Symptoms of Large Intestine heat include constipation, thirst, fever, and a burning anus. In contrast, Damp-Heat of the Large Intestine is recognized by symptoms such as diarrhea, bloody stools, and abdominal distension. Dryness of the Large Intestine can be caused by excess heat or from a deficiency of *yin*, with symptoms of constipation, dryness, and thirst. A deficiency of *qi* in the Large Intestine usually develops from a *qi* deficiency of the Stomach and/or Spleen. Symptoms can include both diarrhea and constipation, as well as organ prolapse, hemorrhoids, exhaustion, and a desire for warm fluids. Cold of the Large Intestine

usually derives from a cold diet or climate, with symptoms such as loose stools, borborygmi, copious pale-colored urine, and general coldness of the body. Wind of the Large Intestine arises when Wind and Heat are trapped within the bowel, leading to bright red blood that precedes or follows a bowel movement.

Based on the correct identification of these various patterns and how they are linked to Stomach and Spleen function, herbs and formulas can be chosen to restore balance to the Small and Large Intestines, including:

- 1. Large Intestine Heat
 - herbs: jin yin hua (*Lonicera japonica* flower), bai jiang cao (*Patrinia scabiosaefolia* herb), huang lian (*Coptis chinensis* rhizome), yu xing cao (*Houttuynia cordata* herb), chuan xin lian (*Andrographis paniculata* herb), bai tou weng (*Anemone chinensis* root)
 - <u>Bai Tou Weng Tang (Pulsatilla Decoction)</u>
 - 6 g bai tou weng (Anemone chinensis root)
 - 9 g huang lian (*Coptis chinensis* rhizome)
 - 9 g huang bai (*Phellodendron amurense* bark)
 - 9 g qin pi (Fraxinus rhynchophylla bark)
 - decoction, Rx: 200 mL bid-tid
 - granules, Rx: 3-5 g bid-tid
- 2. Large Intestine Damp-Heat
 - herbs, e.g. huang qian (*Scutellaria baicalensis* root), huang lian (*Coptis chinensis* rhizome), ku shen (*Sophora flavescens* root), qin pi (*Fraxinus rhynchophylla* bark), hu huang lian (*Picrorhiza kurroa* rhizome)
 - <u>Huang Qin Tang (Baikal Skullcap Decoction)</u>
 - 9 g huang qin (Scutellaria baicalensis root)
 - \circ 9 g shao yao (Paeonia alba root)
 - 9 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - 9 g da zao (Ziziphus jujuba fruit)
 - decoction, Rx: 200 mL bid-tid
 - granules, Rx: 3-4 g bid-tid
- 3. Large Intestine Dryness
 - herbs, e.g. huo ma ren (*Cannabis indica* seed), tao ren (*Prunus persica* seed), zhi ke (*Citrus aurantium* seed), yu li ren (*Prunus japonicus* seed), mai men dong (*Ophiopogon japonicus* root)
 - <u>Run Chang Wan (Moisten Intestines Pill)</u>
 - 15 g huo ma ren (*Cannabis indica* seed)
 - 9 g tao ren (Prunus persica seed)
 - 9 g dang gui (Angelica sinensis root)
 - 30 g sheng di huang (*Rehmannia glutinosa* root)
 - 9 g zhi ke (*Citrus aurantium* seed)
 - Rx: 15 g mixed with honey, in divided doses

- 4. Large Intestine Cold
 - see Spleen yang deficiency
 - Ji Chuan Jian (Benefit River Decoction)
 - 6-9 g jiu cong rong (*Cistanche deserticola* stem, prepared herb)
 - 9-15 g dang gui (Angelica sinensis root)
 - 6 g niu xi (Achyranthes bidentata root)
 - 4.5 g ze xie (Alisma plantago-aquatica rhizome)
 - 3 g zhi ke (Citrus aurantium seed)
 - 1.5-3 g sheng ma (*Cimicifuga heracleifolia* root)
 - decoction, Rx: 200 mL bid-tid
 - granules, Rx: 3-4 g bid-tid
 - 5. Large Intestine Wind
 - Yi Zi Tang (Decoction 'B')
 - 6 g dang gui (Angelica sinensis root)
 - 5 g chai hu (Bupleurum falcatum root)
 - 3 g huang qin (Scutellaria baiclanesis root)
 - 3 g gan cao (Glycyrrhiza uralensis root)
 - 1 g sheng ma (*Cimicifuga heracleifolia* root)
 - 1 g da huang (*Rheum palmatum* root)
 - decoction, Rx: 200 mL bid-tid
 - granules, Rx: 3-4 g bid-tid

Nutrition and Digestive Health

For the average person, it's not too difficult to imagine how diet might affect or interfere with the normal activities of digestion, let alone the health of the body. Despite this apparently obvious truth it often runs counter to the perspective held in modern medicine that diet plays very little role in digestive health. Specifically, this perspective is most crystalized within the field of gastroenterology, in which practitioners routinely dismiss any benefit of dietary therapy at the outset of treatment, reaching instead for anti-inflammatory and immunosuppressive drugs – or if these don't work – surgical intervention. If dietary advice is given, the recommendations of most gastroenterologists are cursory and simplistic, typically consisting of measures such as increasing or decreasing dietary fiber, or relying on preformulated liquid meal replacements.

This perspective is reflective of a bias that is deeply rooted in modern medicine, evolving in part due to a wholesale rejection of traditional medical practices that have always stressed the importance of diet. Instead of the Hippocratic Oath, in which practitioners are exhorted to "... use those dietary regimens which will benefit my patients," the pharmaceutical and allied medical industries have successfully orientated modern physicians away from sustainable dietary therapies towards expensive and often invasive proprietary technologies. A good example of this is the expensive immunosuppressive drug humira (adalimumab) used in the treatment of inflammatory bowel disease (IBD), which in 2014/2015 was the top selling drug with global sales topping \$8.2 billion USD.²¹ The reality is that apart from developing novel foods for impulse purchase, the health industry has little financial incentive to study (and make public) the importance of diet in the prevention and treatment of disease.

²¹ Brown T. 2015. The 10 Most-Prescribed and Top-Selling Medications. WebMD. Available from: http://www.webmd.com/drug-medication/news/20150508/most-prescribed-top-selling-drugs

The obvious ignorance that physicians have when it comes to diet is in part a reflection of the paternalistic culture in which modern medicine evolved, which assigns matters of the kitchen to women, and hence not worth the time of a serious professional. During their education physicians receive only a few hours of training in nutrition, and even after practicing for many years, few can speak with any authority on the subject of diet. This has left academics and non-physicians with the task of developing dietary guidelines, such as the original "food pyramid" developed by researchers at the US Department of Agriculture that exhorted the population to consume most of their calories from carbohydrates. Dietary guidelines were also influenced by the pharmaceutical and food industries, often working in conjunction with self-serving non-profits such as the American Heart Association. Perhaps the best example of this collusion is the decades long battle against cholesterol and saturated fat, which has now largely been disproven.^{22,23} It was a convenient fiction, however, that fit neatly with the pharmaceutical industry's development of cholesterol-lowering drugs, and all the "heart-smart" low-fat alternatives developed by the food industry.

Years ago I when I lived in Calgary, Alberta, I was invited by the secretary of the local Crohns/Colitis Foundation to give a lecture, along with a gastroenterologist, on the holistic treatment of inflammatory bowel disease (IBD) at the University of Calgary. The gastroenterologist spoke first, and had an impressive slideshow of inflamed and ulcerated intestines, and provided a thorough review of the medical therapies used to treat IBD. When he finished to polite applause, I began my lecture by telling the doctors and patients in attendance that it was irrational to try to treat IBD without first addressing diet, and that in my practice, I wouldn't consider working with any IBD patient if they refused to change their diet. Almost immediately there was an uproar in about half the audience, and I had several physicians stand up and tell me that there was no scientific evidence to suggest that diet had any role to play in IBD.

But as I referred to earlier, this accusation is a part of a vicious cycle of wilful ignorance. The reason for the dearth of solid science is obvious: a diet cannot be patented so easily, and thus there is very little money to do the clinical research in the first place. This is apart from the fact that the science itself, while often complicated and difficult to understand, is still remarkably unsophisticated, struggling in confusion with relatively simple issues like the health benefits of coffee or the role of antioxidants. Adding to this is the modern media that prefers to promulgate controversy and confusion, fostering an ill-informed and disempowered public that becomes increasingly dependent upon these gatekeepers for information, rather than following the common sense of traditional knowledge.

Despite the relative paucity of scientific data, I told the doctors who questioned my assertions that in my clinical experience and those of my colleagues, diet is the single most important factor when it comes to the maintenance of digestive health. And what's more I told the audience, this perspective is supported by every system of medicine except that of modern gastroenterology, representing thousands of years of empirical knowledge. This realization, I

²² Berger S1, Raman G1, Vishwanathan R2, Jacques PF2, Johnson EJ3. 2015. Dietary cholesterol and cardiovascular disease: a systematic review and meta-analysis. *Am J Clin Nutr.* 102(2):276-94

²³ Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. 2010. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. *Am J Clin Nutr.* 91(3):535-46

told them, is exceptionally good news for both the open-minded physician and the patient alike, as it provides for a workable, cost-effective strategy that can be implemented at the outset of treatment to provide relatively quick results without having to rely upon invasive medications and procedures. As my lecture continued I went on to describe a basic dietary intervention for IBD, and by the end of the lecture had won over much of the audience. Although I noted a bit of a boost in my patient load over the next while, I was very pleased when several months later when I received a phone call from the secretary of the Crohn's and Colitis Foundation that originally invited me to speak. Without telling me she had put into practice what I had outlined during the lecture, and three months later, was told by her gastroenterologist that she didn't have any sign of IBD. While she was obviously thrilled with the results, I would never suggest that IBD is an easy condition to treat, or that diet alone is always sufficient to promote remission. Nonetheless, I have had over the years and continue to have many cases in which dietary changes bring about profound changes in digestive disorders such as IBD.

Food Allergy

A food allergy is an abnormal immune response to food, with signs and symptoms that range from mild to severe. Mediated by a type I hypersensitivity response that involves the release of IgE antibodies, food allergies usually have a rapid onset, anywhere from a few seconds up to one hour after exposure. Typical symptoms include a rash, hives, itching (of mouth, lips, tongue, throat, eyes, skin, or other areas), swelling (of lips, tongue, eyelids, or the whole face), dysphagia, rhinitis, hoarseness, wheezing, shortness of breath, diarrhea, abdominal pain, lightheadedness, fainting, nausea, and vomiting. If the reaction is particularly severe it can affect the respiratory tract and the circulation of blood causing a life-threatening allergic reaction called anaphylaxis. During an anaphylactic reaction the allergic response promotes bronchial constriction and impaired respiration that leads to cyanosis, along with systemic vasodilation that causes the blood pressure to drop, leading to a loss of consciousness.

Risk factors for food allergies include a family history of allergies, vitamin D deficiency, and obesity. In particular, food allergies are associated with atopy, a chronic condition with a familial basis that is characterized by the manifestation of allergic rhinitis (hay fever), eczema, or asthma. Some research suggests that atopic individuals may have a deficiency of an enzyme called delta-6-desaturase that is involved in the conversion of dietary linoleic acid (n-6) into gamma linoleic acid (GLA). This issue is particularly relevant given the relatively recent shift in the modern Western diet away from traditional fats such as butter, ghee, lard, fish fat, and tallow, towards the consumption of seed oils such as safflower oil, corn oil, and cottonseed oil fats that are all rich in n-6 fatty acids.

Another recently described factor in the development of food allergy found primarily in developed nations relates to a loss in the diversity and resilience of the human microbiome. In part this has arisen due to an irrational and obsessive fear of "germs" in the developed world, fostering the prolific use of antimicrobial compounds in the environment (e.g. cleaning products) and in our food (e.g. antibiotics). This ecological shift has also been facilitated by a diet rich in refined carbohydrates that favors the growth of pathogenic bacteria such as

Shigella and *Escherichia* that not only inhibit probiotic organisms but produce bacterial endotoxins that damage the integrity of the gastrointestinal tract.

A food allergy is usually triggered by a dietary peptide or protein that either resists enzymatic degradation or is improperly denatured. This results in the synthesis and the fixation of IgE antibodies to histamine-containing mast cells and basophils. Upon a second exposure to the same peptide or protein IgE antibodies promote the degranulation of mast cells and basophils and the release of histamine, increasing smooth muscle contraction and vascular permeability. A food allergy may also involve non-IgE mediators due to the migration of leukocytes to the initial site of inflammation as well as cytokines released from mast cells, usually seen 2–24 hours after the original reaction.

Although care is often taken to distinguish allergy from autoimmunity there is a spectrum between them that relates to the role of two broad classes of T helper cells called type 1 (Th1) and type 2 (Th2). An IgE-mediated allergy is generally regarded as a classical Th2 disease, whereas autoimmune diseases are dominated by the activation of Th1 cells. When an allergy becomes chronic, however, it begins to display features of Th1 cell activation. Patients suffering from chronic allergic inflammation have been found to react against a variety of human peptides and proteins that have a structural similarity to exogenous allergens. Once activated by the allergic response, allergen-specific IgE antibodies cross-react with endogenous peptides and proteins, inducing an IgE-mediated allergic response. Chronic exposure to exogenous environmental allergens causes prolonged inflammation and the liberation of autoantigens, resulting in the activation of Th1 cells and IgE-independent tissue damage. For example, several autoantigens that have been identified in autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus, and Sjorgen's syndrome that cross-react with IgE antibodies.

Identifying a food allergy can be a challenge, and to make the diagnosis of allergy and isolation of dietary triggers easier, physicians typically employ a few different methods of food allergy testing. The most common used allergy tests are the skin prick test and radioallergosorbent test (RAST), and although they are effective for isolating environmental allergies (e.g. pollen, animal dander, dust mites), they are much less reliable when it comes to isolating potential food allergens. More recently the enzyme-linked immunosorbent assay (ELISA) and antigen leukocyte antibody test (ALCAT) tests have been developed as a way to test for food allergies, and even though they have been enthusiastically embraced in some circles, their reliability and efficacy has come under serious question.²⁴ Non-laboratory tests such as electro-acupuncture ("vega-testing"), kinesiology ("muscle-testing"), and the use of a pendulum are also frequently used by holistic practitioners, but none of these methods have a scientific or traditional basis.

The most effective method for determining if a particular food is playing a role in a health issue is to perform an elimination-challenge diet. To undertake this test, the patient completely eliminates any suspect food items from the diet for a period of at least 4-6 weeks. If and when the symptoms resolve, the suspect foods are re-introduced, one at a time over a

²⁴ Miller, SB. 1998. IgG Food Allergy Testing by ELISA/EIA What Do They Really Tell Us? *Townsend Lett Doc Pat.* 174:62-65

period of 1-2 weeks, and any health changes are noted. The most common triggers for food allergy include:

- peanut
- tree nuts (e.g. almond, brazil nut, cashew, hazelnut, macadamia, pecan, pine nus, pistachio, walnut)
- dairy
- egg
- cereal grains (e.g. wheat, rye, barley, corn, etc.)
- legumes (e.g. soy)
- fish
- shellfish
- sesame
- sulfites
- oranges
- coffee and tea
- food preservatives
- artificial flavors and colors
- fish
- beef
- nuts and seeds
- tomatoes and other nightshade vegetables (e.g. potatoes, eggplant, capsicum peppers, etc)

Intestinal Permeability

There is an increasing amount of evidence that damage to the intestinal wall plays a role in autoimmune diseases including multiple sclerosis, ankylosing spondylitis, Bechet's syndrome, type one diabetes and rheumatoid arthritis (Cuvelier et al 1987; De Keyser et al 2002; Fresko et al 2001; Malosse et al 1992; Vaarala et al 2002; Yacyshyn et al 1996). Almost all patients suffering such disorders demonstrate some degree of digestive dysfunction, and even if they do not experience overt gastrointestinal disease, when the tissues of the digestive tract are examined by techniques such as colonoscopy they will often display indications of chronic inflammation. Researchers have also noted that in many cases the remission of autoimmunity often occurs in tandem with the remission of digestive inflammation, and vice versa. These clinical findings lend support to a theory called intestinal permeability syndrome and its role in the pathogenesis of autoimmune disorders.

The theory of **intestinal permeability (IP)** syndrome or "leaky gut" is that some agent or combination of agents initiates an inflammatory response in the digestive tract that eventually disrupts the integrity of the mucosal lining. The gut lining maintains a crucially important role as a selectively permeable membrane that serves to separate self from non-self, only allowing for the absorption of dietary nutrients after they have been broken down into sufficiently

small, uncomplicated molecules. The pathogenesis of intestinal permeability syndrome involves a degenerative process in which tiny perforations in the gut lining leads to a loss of this selective permeability. This allows for proteins and peptides derived from the diet as well as from bacteria, parasites, and fungi, to pass through the gut lining. In response to this infiltration, an immune response is initiated that results in the synthesis of specific antibodies against these antigens. Once activated, these antibodies enter into general circulation and cross-react with endogenous peptides and proteins, initiating an immune response that results in tissue destruction. If this process continues unchecked the result is the development of an autoimmune disease.

Chronic gut inflammation damages the protective coating of IgA, an antibody that serves to maintain the integrity of the mucous membranes. This inhibition of immune function in the gut allows for an increased risk of viral, bacterial, fungal, and parasitic infection. Toxins produced by these microorganisms not only damage the gut ling further, they can inhibit liver detoxification pathways, promoting a heightened sensitivity to antigenic triggers such as cigarette smoke and strong perfumes. Chronic gut inflammation also leads to the antibody-mediated destruction of transport proteins, playing an important role in nutrient deficiency and a broad range of health issues including fatigue, irritability, depression, anxiety, poor concentration, headache, a loss of bone density, an increased risk of dental caries, and infertility.

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y	Conditions linked with IP	
	 food allergies and 	
e	sensitivities	
e	 atopic conditions 	
е	 arthritis 	
	 rheumatoid arthritis 	
is	asthma	
st	chronic fatigue	
al	chronic urticaria	
11	Crohn's disease	
s,	diabetes mellitus	
If	 ulcerative colitis 	
	 irritable bowel 	
n	thyroiditis	
	 Raynaud's syndrome 	
	acute gastroenteritis	
	cystic fibrosis	
n	multiple sclerosis	
IS	 spondyloarthropathies 	
	• acne	
r	• eczema	
1.	dermatitis herpetiformis	
ıt	• psoriasis	
	autism	
s,	ADD/ADHD	
IS	environmental illness	
	 pancreatic dysfunction 	
0	poor digestion	
s,	 iron deficiency howel concert 	
of	 bowel cancer vitilizo 	
1	• vitiligo	

• vaculitis (Galland 1995)

Factors that promote intestinal permeability

There are a number of factors that can promote intestinal irritation and inflammation, by either indirect or direct means. Indirect factors include mental and emotional stress, as well as the use of psychostimulants such as caffeine, amphetamines, and cocaine. Other indirect factors include vitamin, mineral, and protein deficiency, as well as impaired hepatic detoxification. Factors that directly cause damage to the gut include dietary lectins (from grains and legumes), antinutrient factors (e.g. polyphenols, protease inhibitors), improper food combinations, peroxidized fats, flour products, micro/nano-particles, food preservatives, food additives, enzyme deficiencies (e.g. lactose intolerance), alcohol, antibiotics, aspirin, NSAIDs, corticosteroids, oral contraceptives, intestinal parasites, pathogenic bacteria, and mycotoxins.

Hypochlorhydria

Hypochlorhydria refers to a deficiency of hydrochloric acid (HCl) secreted by the parietal cells of the stomach, and is an important factor in both food allergy and leaky gut syndrome, as well as in nutrient deficiencies such as vitamin B12. With regard to the latter, the secretion of HCl is absolutely necessary for the activation of intrinsic factor, which then assists in the absorption of vitamin B12 necessary for proper nervous function and the formation of red blood cells. Other common nutrient deficiencies found in hypochlorhydria include protein
deficiency, as well as mineral deficiencies such as chromium, copper, iron, magnesium, manganese, molybdenum, selenium and zinc (Bergner 1997, 292).

There are a number of factors that can lead to a diminishment in stomach acid production. The primary stimulus to gastric acid secretion is the consumption of protein, and thus a low-protein diet generally serves to down-regulate HCl secretion. Hypochlorhydria is thus more common in vegetarians and especially vegans. Another prominent factor in hypochlorhydria is chronic stress and the activation of the sympathetic division of autonomic nervous function. The activation of the fight or flight response, which can also be induced by certain drugs such as amphetamines and cocaine, leads to a general diminishment in digestive activity including an impairment in gastric acid secretion. Aging too can lead to hypochlorhydria when the parietal cells of the stomach begin to atrophy, causing gastric HCl levels to decline. Likewise, the chronic ingestion of drugs that suppress acid secretion, such as antihistamines, antacids and proton-pump inhibitors, as well as those that are toxic to the gastric mucosa such as aspirin and acetaminophen, also leads to a diminishment in stomach acid secretion.

Hypochlorhydria is difficult to diagnose, and a few different techniques exist that can provide some indication of the problem. A deficiency of gastric acid can be inferred by an analysis of the case history and the presence of factors pernicious anemia, poor gastric motility, and impaired protein digestion (e.g. foul-smelling flatus). Laboratory methods are also used to detect the presence of undigested proteins in the stool. The most objective assessment to determine hypochlorhydria is the Heidelberg pH capsule gastric analysis, in which a hard plastic capsule containing a miniature radio transmitter with a pH-sensing device is introduced into stomach. The stomach is challenged with a sodium bicarbonate solution and the stomach's ability to return the pH to normal (1.0-2.3 pH). The data collected by the pHsensing device is transmitted, picked up, and stored by a receiver that is fastened to the patient's waist. Within 20 minutes after challenging the stomach the pH should return to normal. A failure to re-establish normal gastric acidity within 20 minutes indicates hypochlorhydria. A complete failure to re-establish normal acidity even after several hours of testing indicates a complete absence of stomach acid, or achlorhydria.

Dysbiosis

Another important factor in the equation of intestinal permeability is the gastrointestinal microbiome, including the presence of beneficial "probiotic" bacteria such as *Lactobacillus acidophilus* and *Bifidobacterium bifidum* that primarily reside in the colon. Researchers have identified up to 400 different species of bacteria in the colon, the vast majority of which (i.e. 99.9%) are strict anaerobes. While not all of these organisms are necessarily beneficial, probiotic organisms such as *Lactobacillus* and *Bifidobacterium* provide a number of important functions, chief of which is to allow the colon to salvage energy from otherwise indigestible nutrients such as fiber. These probiotic microorganisms ferment dietary fiber as a source of energy, and in the process synthesize short chain fatty acids (SCFA) such as acetic, propionic, and butyric acids that are rapidly absorbed by colonic cells. These fatty acids serve as a vital source of nutrients for colonic cells, ensuring the proper replication and turn over of cells, as well as promote water absorption, prevent osmotic diarrhea, and inhibit the growth of pathogenic bacteria such as *Candida albicans* and *Clostridium difficile* (De Roos and Katan 2000).

Probiotic organisms also benefit the colon by interacting with toll-like receptors (TLRs) found on the surface of colonic cells. These bacteria secrete TLR ligands such as lipopolysaccharide and lipoteichoic acid that interact with surface TLRs. The resultant signaling appears to enhance the ability of the epithelial surface to withstand injury and enhance repair, whereas a disruption of TLR signaling compromises the ability of the intestinal surface to withstand and recover from injury. In this way the viability of the colonic microbiome is a crucial consideration in the maintenance of colonic health.

Since the discovery of probiotic organisms only a few decades ago there has been an explosion in probiotic supplements. Bench work and clinical research has demonstrated the therapeutic benefits of probiotic organisms, noting the various activities of different bacterial strains, as well as certain yeasts such as Saccharomyces boulardii that is efficacious in the treatment of Clostridium difficile infection. While especially in the consumer-driven Western world supplements have become popularized, much of the original and continuing research is based on food-sourced probiotics, such as cultured dairy products. Supplements in many cases are not the best source for probiotics, not only due to their relative cost, but because of research which suggests that encapsulated probiotic supplements do not have the shelf stability that consumers are led to believe. In 2009, ConsumerLab reported that 85% of the probiotics tested did not contain the amount of organisms claimed on the label. While there is additional research suggesting that even dead probiotic bacteria can have some immunomodulatory benefits, probiotic supplements should not be used to replace traditional probiotic foods, including lacto-fermented vegetables (e.g. sauerkraut, achar, kimchi), lacto-fermented cereals and legumes (e.g. sourdough, idli, miso), and lacto-fermented meats (e.g. salami, blackforest ham, jinhua ham).

Assessment of Intestinal Permeability

There are several methods that are used to assess the possibility of intestinal permeability. The case history is of great importance, and in most long-standing digestive problems it can be reasonably inferred that leaky gut is a factor. Similarly, chronic extra-intestinal inflammation and tissue degeneration, found in autoimmune diseases such as rheumatoid arthritis are also important indications of intestinal permeability. To determine if such an assessment is correct the condition is treated on the basis that it is intestinal permeability, and the results are then observed over a period of 2-3 months. If the health issue is caused by intestinal permeability, using measures to restore digestive health should improve the condition.

For practitioners requiring a more objective method of assessment called a sugar absorption test can be used to measure the ability of two non-metabolized sugar molecules, mannitol and lactulose, to permeate the intestinal mucosa. Mannitol is readily absorbed and serves as a marker of transcellular uptake, whereas lactulose is only slightly absorbed and serves as a marker for mucosal integrity. To perform the test, the patient mixes a premeasured amount of lactulose and mannitol with water, and then drinks the challenge substance. The test then measures the amount of lactulose and mannitol recovered in a urine sample over the next 6 hours, and the results are compared against a healthy standard. Low urinary levels of mannitol and lactulose indicate malabsorption, whereas high levels of lactulose suggest an increase in intestinal permeability.

Nutritional Treatment of Intestinal Permeability

The key to resolving leaky gut syndrome rests in avoiding factors that produce gut injury, while simultaneously undertaking measures to decrease inflammation, promote epithelial healing, and restore the colonic microbiome. Apart from avoiding substances that cause direct injury to the gut, the best way to ensure intestinal health is to consume a diet that is low in antigenic foods. The most common food allergens are those that have been introduced recently into the human diet, during the advent of the agrarian revolution approximately 10,000 YA. In this respect, it would seem from data comparing the few remaining huntergatherer peoples with post-agricultural societies that a pre-agrarian diet is associated with superior health. This suggests that many foods that are staples in agricultural societies are biologically inappropriate, and counter to our evolutionary history. To this end a diet that reflects the biology of our evolution is a useful place to start in the treatment of intestinal permeability.

The Paleolithic diet

The **Paleolithic diet** reflects the traditional eating patterns of nomadic humans that existed during the Paleolithic period, prior to the Agrarian Revolution and advent of the Neolithic period. It represents a vast diversity of foods and practices, exemplified by the traditional dietary patterns of the last few remaining hunter-gatherer peoples, from the Inuit peoples of northern Canada to the Aboriginal peoples of Australia. The Paleolithic began with the Pleistocene, a geological epoch marked by repeated cycles of extensive glaciation that lasted from about 2.6 MYA to 11,700 years ago. This period time represents approximately 99.97% of our evolution as anatomically modern humans, and while there is good evidence to suggest that genes do evolve over shorter periods of time, our experiment with agriculture has been too short to affect a complete adaptation to this this way of eating. Over the last 25 years a body of clinical experience has been evolving that confirms a number of benefits for this diet, not only in the treatment of digestive disorders, but immune, metabolic, and psychiatric disorders as well.

Beyond the simple fact of our long evolution over hundreds of thousands of years, and the disparity between this and the relatively recent shift in our diet, interest in the Paleolithic diet is based upon several perceived benefits. Skeletal remains show that Paleolithic humans experienced a high degree of health, with body structures similar to that of modern athletes. In contrast, human remains from the Neolithic indicate a marked decrease in health status, with an increased prevalence of malnutrition, infectious disease, iron deficiency anemia, enamel hypoplasia, dental caries, and degenerative joint disease.²⁵ From the Late Paleolithic to the early Neolithic period human physical stature decreased by almost 10%, life expectancy fell by 30%, and the overall health index continued to declined by an average of 2.5 percentage points per millennium until 1500 CE.²⁶ Studies of the few remaining hunter-gatherer peoples

²⁵ Steckel R. 2004. The Best of Times, The Worse of Times: Health and Nutrition in Pre-Columbian America. *NBER*. Available from: http://www.nber.org/papers/w10299

²⁶ Steckel R. 2004. The Best of Times, The Worse of Times: Health and Nutrition in Pre-Columbian America. *NBER*. Available from: http://www.nber.org/papers/w10299

such as the !Kung San of the Kalahari and Aché people of Paraguay have found them to be remarkably free of the chronic degenerative diseases that plague the industrialized world. Likewise, ethnographic reports from the first European explorers in North America consistently refer to the robust health and vitality of the Native American peoples they encountered.^{27,28} Since colonization and the adoption of the modern agricultural diet, however, Native Americans now suffer from the highest rates of diabetes, cardiovascular disease, and cancer in North America.²⁹

Early human societies were centered around a hunter-gatherer lifestyle, following the seasonal migrations of large herds of mammals and the spawning of fish, gathering a large variety of plant foods including sea vegetables, lichen, and edible fungi. While gathered foods were an important contribution to the diet, often reflecting a resourceful and ingenious approach to survival, whenever possible our Paleolithic hunter-gatherer ancestors consumed high amounts of animal food in their diet, comprising between 45–65% of their total caloric intake.³⁰ The estimate for total ancestral fat intake is between 25-60%, depending on latitude, with an estimate of 35% for Paleolithic humans living in northeast Africa, modern humanity's most direct ancestor.³¹ This reliance on animal foods along with the generally low carbohydrate content of wild plant foods tended to shift macronutrient consumption ratios towards protein (19–35% of energy) at the expense of carbohydrates (22–40% of energy).³²

In traditional hunter-gatherer societies the diet was dependent upon the seasons, climate, and geography. In northern temperate climates when solar activity and vegetative cycles peaked during spring and summer, the diet was focused on wild plant foods, complemented by smaller amounts of animal food. In fall and winter, however, when solar influence diminished and most food plants became unavailable, the diet naturally became much more dependent upon animal food. The plant and animals foods consumed by our Paleolithic ancestors, however, are hardly comparable to modern facsimiles. Plant foods eaten by our ancestors contained about 4-5 times the amount of fiber found in the modern diet, with lower levels of carbohydrate, and higher levels of vitamins, minerals, and phytonutrients.³³ And despite the fact that Paleolithic humans ate even more fat than is recommended by the Institute of Medicine (45-65% versus 30%), the amount of saturated fat consumed was lower than is found in the modern diet, with higher levels of both mono- and poly- unsaturated fatty acids, found in the muscle, organs, and brain of wild animals.³⁴

²⁷ Montaigne ML. 1886. *The Essayes of Michael Lord of Montaigne*. Translated by John Florio, edited by Henry Morely. London: George Routledge p. 94

 ²⁸ Lohantan. 1703. New Voyages to North-America: Giving a full account of the Customs, Commerce, Religion and strange Opinions of the Savages of that Country. Vol 2. London. Available from: http://www.archive.org/details/cihm_37430
 ²⁹ Obomsawin R. 1983. Traditional Life Styles and Freedom from The Dark Seas of Disease. Community Dev J. 18(2):187-97

³⁰ Cordain L, Miller JB, Eaton SB, Mann N, Holt S, Speth JD. 2000. Plant-animal subsistence ratios and macronutrient energy estimations in worldwide hunter-gatherer diets. *Am J Clin Nutr.* 71:682–92

³¹ Cordain L, Miller JB, Eaton SB, Mann N, Holt S, Speth JD. 2000. Plant-animal subsistence ratios and macronutrient energy estimations in worldwide hunter-gatherer diets. *Am J Clin Nutr.* 71:682–92

³² Cordain L, Miller JB, Eaton SB, Mann N, Holt S, Speth JD. 2000. Plant-animal subsistence ratios and macronutrient energy estimations in worldwide hunter-gatherer diets. *Am J Clin Nutr.* 71:682–92

³³ Eaton SB. 2006. The ancestral human diet: what was it and should it be a paradigm for contemporary nutrition? In: *Proceedings of the Nutrition Society,* University of East Anglia, 28 June–1 July 2005. 65: 1–6

³⁴ Eaton SB. 2006. The ancestral human diet: what was it and should it be a paradigm for contemporary nutrition?

In many respects it is impossible to completely replicate a Paleolithic diet without also following a Paleolithic lifestyle, but even with some modifications many of the same benefits can be experienced. The goal is to sustainably replicate the nutrient density and macronutrient ratios of the Paleolithic diet, while avoiding immunogenic agriculture staples, including:

- cereals, e.g. wheat, rye, corn, etc.
- legumes, e.g. soy, beans, peas, etc.
- seed oils, e.g. canola oil, safflower oil, sunflower oil
- dairy products, e.g. milk, butter, yogurt
- sweeteners, e.g. sugar, maple syrup, agave syrup
- sweet (hybridized) fruits, e.g. banana, mango, orange
- salt, e.g. refined table salt

Acceptable foods on the Paleolithic diet could thus include:

Animal products

- wild fish, e.g. salmon, halibut, pike, arctic char, mackerel, sardine, trout, pickerel, smelt, herring
- free range organic eggs
- free range organic poultry, e.g. chicken, turkey, duck, partridge, pheasant, quail, emu, ostrich
- free range, grass-fed red meat, e.g. goat, mutton, lamb, pork, beef
- wild game, e.g. elk, venison, bison, caribou, moose
- homemade, nitrite-free fish or meat jerky

Above-ground vegetables

- leafy green vegetables, e.g. cabbages, chard, kale, spinach, lettuce, watercress, dandelion greens, beet greens vegetable flowers, e.g. broccoli, artichokes, Brussel sprouts, cauliflower
- stem vegetables, e.g. celery, asparagus, leeks, rhubarb
- fruiting vegetables, e.g. cucumbers, eggplant, peppers, summer squash, tomatoes
- sea vegetables, e.g. nori, wakame, hijiki, kombu, dulse, kelp
- sprouts, e.g. broccoli, onion, garlic, alfalfa, red clover algae, e.g. chlorella, spirulina
- culinary herbs, e.g. basil, oregano, marjoram, thyme, rosemary, cilantro, mint wild plants (e.g. nettle, fiddleheads, edible fungi)

Starchy vegetables

- roots, e.g. carrot, sweet potato, parsnip, beet, rutabaga, burdock, radish
- bulbs, e.g. onion, garlic, leek

In: Proceedings of the Nutrition Society, University of East Anglia, 28 June–1 July 2005. 65: 1–6

- rhizomes, e.g. ginger, turmeric, galangal
- winter squashes
- edible wild roots, rhizomes, tubers, bulbs

Fats and oils

- animal fats, e.g. lard, tallow, ghee, fish oil
- oily fruits, e.g. extra virgin olive oil, virgin avocado oil, virgin palm oil
- seed oils, e.g. virgin almond oil, virgin coconut oil, virgin palm kernel oil

Fruits

- wild fruits, e.g. saskatoon, huckleberry, juneberry
- cultivated berries, e.g. raspberry, strawberry, blueberry
- pomes: apple, pear, quince
- stone fruits: apricot, peach, plum, cherry
- grape
- melons: cantaloupe, honeydew
- citrus: lime, lemon, grapefruit
- tropical fruits: passionfruit, guava, starfruit, açaí, dragonfruit, mangosteen, pomegranate, prickly pear
- stewed dried fruit: goji, amla, raisin, fig, prune, mulberry

Some exceptions can be made to a strict avoidance of agricultural food products, depending on their properties, on a case-by-case basis. Common interpolations to a strict Paleolithic diet may include:

Cereals and legumes

- mostly fermented; preserve cellular structure (no flour)
- non-grass cereals, e.g. quinoa, buckwheat, amaranth
- grass cereals, e.g. rice, nixtamalized corn
- split/washed dal, e.g. mung, chickpea, urad
- fermented bean products, e.g. tempeh, natto, pickled beans

Dairy products

- non-immunogenic sources, e.g. A2 cow, goat, sheep, buffalo
- fresh/raw or home pasteurized milk
- unripened cheese, e.g. feta, panir, cottage cheese
- butter, ghee
- live culture yogurt, sour cream, buttermilk

While salt (sodium chloride) is often restricted in metabolic disease, recent research suggests that the recommended guidelines for salt consumption of less than 2.5 g per day are too low,

and is correlated with an increase in the risk of morality.³⁵ While a Paleolithic diet can be thought of as a low-salt diet, our Paleolithic ancestors ate salty foods such as seafood and sea vegetables, and may have collected and consumed naturally occurring salt crystals as part of their diet. Most of the risk of excess salt consumption relates to a disturbance in the ratio of potassium to sodium. Although our Paleolithic ancestors consumed salt as part of the diet, they also consumed much higher quantities of potassium from wild plant foods. In contrast, whereas the potassium to sodium ratio of the Paleolithic diet was about 5:1, the ratio in the modern diet is closer to 4:5.³⁶ Another important difference in salt consumption was that any salt consumed by our ancestors was unrefined and rich in trace minerals. This type of salt is similar to unrefined rock salt or sea salt, rather than the pure white crystalline powder of pure sodium chloride that is table salt.

FODMAPs diet

The FODMAP diet has been proposed as a way to resolve chronic gut issues such as irritable bowel syndrome (IBS) by reducing or eliminating foods that contain indigestible long-chain sugars found in foods such as cereals, pulses, root vegetables, and fruits. The term FODMAP is an acronym devised by researchers at Monash University in Australia referring to foods that contain "Fermentable Oligo-saccharides, Disaccharides, Mono-saccharides and Polyols." Examples of high FODMAP foods include:

- vegetables: asparagus, artichoke, onion, leek, garlic, pea, beet, cabbage, celery, sweet corn
- fruits: apple, pear, mango, nashi pear, watermelon, nectarine, peach, plum
- dairy: cow's milk, yogurt, soft cheese, cream, custard, ice cream
- legumes: soy, beans, pea
- cereals: wheat, rye, pasta, crackers, biscuits
- nuts/seeds: cashews, pistachios³⁷

FODMAPs are fermented by the gut bacteria that naturally inhabit our intestines, and in sensitive people, appears to cause symptoms such as gas, bloating, colic, and diarrhea. While many patients following a low FODMAP diet do indeed find that their symptoms diminish after some time, they also find that the diversity and variety of foods in their diet begins to decline dramatically. With these and other restrictions, many patients feel that they have painted themselves into a corner, finding that their intolerances and sensitivities actually worsen over time, or that they experience problems such as chronic constipation.

The notion that indigestible sugars can cause gas and bloating isn't anything new. More than 20 years ago when I began my training as a herbalist, I was taught that a whole foods

³⁵ O'Donnell M, Mente A, Rangarajan S, et al. 2014. Urinary sodium and potassium excretion, mortality, and cardiovascular events. *N Engl J Med.* 371(7):612-23.

³⁶ Eaton SB. 2006. The ancestral human diet: what was it and should it be a paradigm for contemporary nutrition? In: *Proceedings of the Nutrition Society*, University of East Anglia, 28 June–1 July 2005. 65: 1–6

³⁷ Barrett JS 2017. How to institute the low-FODMAP diet. *J Gastroenterol Hepatol.* 32 Suppl 1:8-10.

vegetarian diet was a healthier option than a meat-based diet, and during the first couple years of my practice I encouraged many to make this switch. One of the more common issues I observed, however, was that with the displacement of meat for vegetarian sources of protein such as beans, nuts and seeds, patients very often presented with an increase in gas, bloating, and diarrhea. I learned to anticipate this, and explained that it was a natural result of adding more high-fiber foods to the diet, which in turn, altered the composition of the microflora in the gut. In many cases, the symptoms of gas and bloating that accompanied these changes were temporary, and usually the symptoms would begin to diminish within a few weeks. Often I would discuss ways to improve digestibility of these high fiber foods, such proper cooking techniques, or the use of culinary herbs such as cooking legumes with ginger, garlic, and other culinary herbs. In some patients, however, their digestive symptoms didn't get better over time, and it was for this as well as other reasons that I soon abandoned the idea that a vegetarian diet was necessarily well-suited to everybody. Nonetheless, I learned a great deal about how to deal with digestive issues attributable to a high fiber diet.

It is very clear that some people do note an improvement in their digestive symptoms when they avoid high FODMAP foods. But before we celebrate the success of this intervention, it is important to look at some potential problems. Firstly, the FODMAP diet seeks to remove many of the high fiber foods that researchers have linked to a reduction in the risk of hypertension, stroke, elevated LDL cholesterol, ischemic heart disease, diabetes, and colorectal cancer. Some FODMAPs have also been shown to benefit chronic digestive disorders such as GERD, ulcer, and hemorrhoids, as well as promote mineral absorption, modulate immune function, resist infection, enhance mood and memory, and promote healthy aging. In this context, the indigestible fibers described by the FODMAP system serve as "prebiotics" that provide a substrate for the growth and development of the probiotic bacteria that are crucial in the maintenance of gut health. Further, some high FODMAP foods such as onion and garlic contain powerful antitumor chemicals such as diallyl disulfide, S-allylcysteine, and ajoene, and when consumed regularly are associated with a significant reduction in cancer risk.

Given the weight of evidence in favor of consuming prebiotic foods, a question arises if a FODMAP-restricted diet is a valid approach for managing chronic digestive disease. While it may be empirically true that a FODMAP-restricted diet promotes an improvement in symptoms, are the benefits only attributable to an avoidance of fermentable sugars? Many of the foods listed by researchers at Monash University as being high in FODMAPs are also high in antinutrient factors (ANFs), including constituents such as phytic acid, lectins, protease inhibitors, and polyphenols that interfere with digestion and absorption. It thus seems reasonable to challenge the conclusions made by FODMAP proponents that it is just the fermentable sugars that are the problem, when in actual fact, the issue is a great deal more complicated.

When we reach back into the history of our culinary traditions, it is clear that humans have successfully developed ways to deal with the digestive issues caused by high FODMAP foods that have served as staples for millennia. The red beet for example was widely consumed throughout Eastern Europe as a soup called borscht, despite the fact that contains high levels of fructans that can cause gas and bloating. When consumed irregularly, the indigestible prebiotic fibers in boiled or baked beets are helpful for occasional constipation, and have a

mild laxative activity. When consumed as a staple, however, the high levels of fructans in beets will eventually causes digestive issues. The traditional solution to ameliorate this problem was to ferment the beets first before eating.

Although largely forgotten by contemporary eastern Europeans, a proper beet "borscht" was originally made with fermented beets, a tradition kept alive by some Jewish families as a dish called russel. Fermentation of the beets before cooking utilizes FODMAP-loving bacteria to break down indigestible sugars that only cooking cannot. Among poor Jewish farmers during the 20th century Ukraine beets were very much a staple, but without fermentation it is likely that it would have resulted in severe malnutrition, and hence, would never have become a staple food. Likewise, many other cultures similarly employed fermentation to improve the nutrient bioavailability of their food, including dairy products (e.g. yogurt), cereals (e.g. idli), legumes (e.g. natto), and seeds (e.g. cacao).

In this light, the FODMAPs diet is best used as a temporary measure to restore ecological balance of the GI microbiome, particularly in problems such as small intestine bacterial overgrowth (SIBO). To take advantage of their health benefits over the long term, however, once digestion has been restored high-FODMAP foods should be gradually re-introduced into the diet, but always with a mind to ensure their correct preparation.

Mind-Body Connections in Digestive Health

Modernity has brought us many advantages, but it has also fundamentally changed how we look at the world and see ourselves. Where at one time there was a widely held belief in the interconnectedness of all things, the fragmentary, reductionist approach of the scientific method has upended and severed these connections, fomenting a belief in the fundamental separateness of things. Perhaps nowhere more prominently do we see this disconnect than in the Western conception of mind, where consciousness is viewed as little more than a series of neurons firing away in the brain, isolated and separate from the rest of body. And yet, despite the overwhelming scientific evidence that the brain is the center of consciousness, this knowledge runs counter to the everyday subjective experience that tells us our thoughts and feelings aren't experienced in some discrete part of the brain, but are experienced as a visceral connection to our physical being.

Traditional systems of medicine speak of the profound link between the mind and body, and specifically, the connection between the mind and the visceral organs, such as the heart, lungs, stomach, liver, and bowels. Remnants of this perspective can found in the English language, in which it is still common to hear about someone having "a lot of gall," "venting their spleen," or suffering from a "broken heart." What's important to note about these idioms is that they are not simply quaint attempts to describe emotional states, but an accurate description of where and how these feelings are actually experienced in the body. Although originally antithetical to this notion, the latest science has begun to validate these concepts. This includes the discovery of an enteric nervous system in the gut, with its own secretion of serotonin, providing a direct neurological link between mental and digestive health. The interconnectedness of consciousness is also seen by researchers that have documented how thoughts and emotions often function like a kind of contagion between groups of people.³⁸ And

³⁸ Colino S. Are You Catching Other People's Emotions? US News. Jan. 20, 2016. Available from: https://health.usnews.com/health-news/health-wellness/articles/2016-01-20/are-you-catching-other-peoples-emotions

even beyond neurophysiology and the influence of other people, non-human organisms such as our gut bacteria have been shown to play a key role in disorders such as depression, anxiety, and autism.^{39,40}

The common sense understanding that thoughts and emotions influence aspects of the physical body such as digestion is supported by thousands of years of empirical practice in both Ayurveda and Chinese medicine, and should be part and parcel of any assessment of digestive health. Rooted in the ancient teachings of the Vedas is the notion that the mind actually "creates" the physical universe through its perception. Thus health and disease and even life and death are thought in the ancient teachings of India to be wrought by the mind. Considering the importance of mind, it is necessary to properly appreciate the nature of the patient's mental and emotional state, and how this colors their thoughts, words, and actions.

When the patient's mental and emotional state can be seen to be unproductive, a strategy is shared to ameliorate and pacify the aggravated condition. This can involve a reassessment of personal or career goals resulting in significant lifestyle changes. Personal and professional relationships may need to be addressed, and very often at the core of many mental and emotional issues is unresolved trauma, usually stemming from early childhood experiences. While counselling and psychotherapy are often helpful to resolve such issues, traditional medical practices also made use of creative expression, engaging in activities such as painting, dance, singing, and performance that allow the patient to tap into their own healing potential. In this way, disturbed mental and emotional patterns are really just a form of energy that needs to be recognized, released, and rebalanced.

Dietary therapy, herbal remedies, and supplements can also be used to address mental and emotional issues, restoring digestion and the gut microbiome, as well modulating the immune system, and rebalancing neuro-endocrinal functions. In particular, there are a large array of medicinal plants and other natural products that can be used to simultaneously address mental/emotional issues and digestive problems. Many of the key digestive imbalances described in Ayurveda and Chinese medicine are associated with specific mental and emotional patterns, and thus the indicated herbal formulas for visceral disorder can often be used to address the underlying mental and emotional state.

Thoughts and emotions in both Ayurveda and Chinese medicine are in large related to the function of the heart. In Chinese medicine the heart stores the *shen* or spirit, and in Ayurveda the heart is the foundation of consciousness, containing the nourishing muck and rhizomes connected to a lotus stalk (*manavaha srotas*) that rises up and unfolds in the many petalled blossom of the brain. In Ayurveda, it is the heart (*hrdaya*) that first receives the nutrients digested from food, and thus the nature and quality of the diet has an immediate impact upon consciousness.

³⁹ Foster JA, McVey Neufeld KA. 2013. Gut-brain axis: how the microbiome influences anxiety and depression. Trends Neurosci. 36(5):305-12.

⁴⁰ Kraneveld AD, Szklany K, de Theije CG, Garssen J. 2016. Gut-to-Brain Axis in Autism Spectrum Disorders: Central Role for the Microbiome. *Int Rev Neurobiol.* 131:263-287.

In Ayurveda, specific thoughts and emotions can also be related to each of the *dosha(s)* and the set of qualities that each expresses, whether in isolation or in combination. Thus wherever an imbalanced mental or emotional quality can be found, measures related to the opposite quality or qualities can be undertaken:

Emotion	Quality	Dosha	
loneliness	dry	vata	
stubborn	heavy	kapha	
anger	hot	pitta	
anxiety	cold-dry	vata	
depression	cold-moist	kapha	
jealousy	moist-hot	pitta	
worry	moist-cold	kapha	
irritation	light-hot	pitta	
confusion	light-cold	vata	

In Chinese medicine, specific emotions can be each of the digestive organs, including the Stomach, Spleen, Liver, Gall Bladder, Small Intestine, and Large Intestine:

Digestive organ	Function		Organ Imbalance Emotional Patterns
Stomach	sensing and		yang deficiency: lack of motivation
	feeling		yin deficiency: over-activity, stress, mental strain
			Stomach fire: irritation, anger
			qi deficiency: confusion
			qi stagnation: anger, frustration
Spleen	reflecting	and	Spleen qi stagnation: excess thinking, obsession
	analyzing		Spleen <i>qi</i> deficiency: anxiety, regret
			Spleen dampness: attachment, worry
			Spleen yang deficiency: self-doubt
Small Intestine	judgment	and	Small Intestine qi stagnation: over-excitement, frustration, stress
	discrimination		
Liver	ambition	and	qi stagnation: irritability, mood swings, depression
	planning		Liver yang: irritability, frustration, anger
Gall Bladder	courage	and	qi deficiency: indecision, timidity, discouragement
	initiative		
Large Intestine	letting go		<i>qi</i> stagnation: grief, guilt, anxiety

Etiology, Pathology and Treatment of Digestive Disorders

Indigestion, nausea and vomiting

In traditional systems of medicine such as Ayurveda, Chinese medicine, and the Western herbal tradition, indigestion represents a fundamental disruption of balance that brings about ill-health. According to Ayurveda, the qualities of *agni* (digestive fire) are hot, light, dry, sharp, and penetrating, and thus opposite qualities, i.e. cold, heavy, moist, dull, and dull, represent an obstructing or diminishing influence upon digestion. In Ayurveda this syndrome is called *mandagni*, or 'slow digestion', which arises from an increase of *kapha dosha*. If a food is eaten that increases *kapha*, and the *agni* remains too weak to properly digest it, the result is the formation of *ama*, or 'undigested food'. Like wet leaves that smother a burn pile, the formation and presence of *ama* further impairs digestion, as well as the ability to generate *ojas* ('vitality') from the ingested food, which in a vicious cycle pattern, further diminishes the strength of the digestive fire.

In Chinese medicine lack of appetite is primarily caused by an impairment of the Stomach and Spleen, and similar to Ayurveda, is usually caused by eating difficult to digest or otherwise cold, heavy, moist foods. This results in an impairment to the *yang* energy of both the Stomach and Spleen, promoting symptoms such as weak appetite, mucus congestion, and generalized coldness. As the body becomes deprived of its ability to properly harness the energy contained within the food, the status of *qi* weakens leading to a diminishment of the Stomach and Spleen *qi*. This causes additional symptoms such as epigastric discomfort and generalized fatigue. Eventually this leads to the accumulation of dampness and the syndrome known as Food Stagnation, leading to further symptoms such as burping and abdominal distension after meals.

In the Western herbal tradition the concept of weak digestion is essentially the same as is described in both Chinese medicine and Ayurveda. The early 19th century American herbalist Samuel Thomson visualized all disease as fundamentally related to the pathogenic quality of cold. Similar to the idea of *ama* in Ayurveda, or Food Stagnation in Chinese medicine, Thomson called this fundamental affliction "canker", noted by physical symptoms of cold, weak digestion, and the accumulation of mucus, that eventually leads to a state of "putrefaction." According to Thomson, canker is "... caused by cold, or want of heat, for whenever any part of the body is so affected with the cold so as to overpower the natural heat, putrefaction commences; and if not strong enough to overcome its progress, it will communicate with the blood, when death will end the contest between heat and cold, or the powers of life and death by deciding in favor of the latter" (Thomson 507, 1841).

Lack of appetite

One of the earliest expressions of weak digestion in Ayurveda is *aruchi*, or lack of appetite. There are three primary subtypes of *aruchi* based on each one of the *doshas*, as well as a fourth type (*aghantuja*) that relates to emotional factors such as fear, shock, and anger. Simple measures are undertaken at the outset to address *aruchi*, and among the simplest is to not eat, engaging in a short period of fasting or following a graduated diet until the appetite returns.

The graduated diet, or *sansarjana krama*, is an approach to eating recommended by Ayurveda to restore the digestive fire, feeding it small amounts of food in increasingly larger amounts over a period of three to seven days, until proper digestion is established. The first food given on the first day of the graduated diet is a thin rice soup called *peya*, prepared by cooking basmati or partially-milled red rice in eight times its volume of water. If the *peya* is properly digested, a thicker rice porridge called *vilepi* can be introduced next, prepared by cooking rice in four times its volume of water. If this is well digested, the next meal is *yusha* or *kitchari*, first prepared without spices and salt (*akrita yusha*), followed the use of spices, salt, and fermented vegetables (*krita yusha*). Following this, the last meal during the graduated diet is a meat soup called *mansa rasa*. At this stage, proper digestion should be re-established, and a normal diet can followed, but with an emphasis on soups and stews, prepared with herbs and spices to augment digestion.

Strength of therapy	Peya	Vilepi	Akrita yusha	Krita yusha	Mamsa rasa
mild	Day 1 lunch	Day 1 dinner	Day 2 lunch	Day 2 dinner	Day 3 lunch Day 3 dinner
moderate	Day 1 lunch	Day 2 lunch	Day 3 lunch	Day 4 lunch	Day 5 lunch
	Day 1 dinner	Day 2 dinner	Day 3 dinner	Day 4 dinner	Day 5 dinner
strong	Day 1 lunch	Day 2 dinner	Day 4 lunch	Day 5 dinner	Day 7 lunch
	Day 1 dinner	Day 3 lunch	Day 4 dinner	Day 6 lunch	Day 7 dinner
	Day 2 lunch	Day 3 dinner	Day 5 lunch	Day 6 dinner	

One general formula for lack of appetite used in Ayurveda that is useful for all three *doshas* is <u>Dhanyapanchakam churna</u>, comprised of equal parts dhaniya (*Coriandrum sativum* seed), hriverum (*Pavonia odorata* herb), musta rhizome (*Cyperus rotundus* rhizome/tuber), bilwa (*Aegle marmelos* unripe fruit), and shunthi (*Zingiber officinalis* rhizome), along with smaller amounts of saindhava (pink salt). The standard dose for <u>Dhanyapanchakam churna</u> is 2-3 grams given with warm water, twice daily.

For aruchi caused by emotional factors (*aghantuja aruchi*) it is important to resolve these feelings by creating a pleasant and comforting environment for the patient.

Indigestion

If the causes for poor appetite are not addressed and food continues to be consumed without consideration, the result in Ayurveda is *ajirnam*, or indigestion. There are three basic causes of indigestion in Ayurveda, each of which relates to one of the three *doshas*, called *amaja ajirnam* (*kapha*), *vidagdha ajirnma* (*pitta*), and *vistamba ajirnam* (*vata*).

Amaja ajirnam

The most common form of indigestion is *amaja ajirnam*, caused by the production of *ama* from a weakness of the digestive fire (*mandagni*), caused by *kapha dosha*. Signs and symptoms of *ama* include a lack of appetite, nausea/vomiting, abdominal heaviness, mucus congestion, lethargy, puffiness under the eyes, and frequent burping with the taste of the previous meal. One useful formula in *amaja ajirnam* is <u>Pippalyadi churna</u>, given in doses of 1-2 grams, with warm water, twice daily. <u>Pippalyadi churna</u> is comprised of equal parts:

- pippali (Piper longum fruit)
- haritaki (*Terminalia chebula* fruit)
- musta (*Cyperus rotundus* rhizome)
- dhaniya (*Coriandrum sativum* seed)
- shunthi (*Zingiber officinalis* rhizome)
- vidam lavana (salt made from the fruit of *Phyllanthus emblica*)

In Chinese medicine, *amaja ajirnam* relates to a deficiency of Stomach and Spleen *yang* that allows for the accumulation of dampness causing Food Stagnation. Remedies to warm the middle and dispel dampness are thus called for, such as <u>Li Zhong Wan (Regulate Middle Pill)</u>, <u>Ping Wei San (Calm Stomach Powder)</u>, and <u>Fu Zi Li Zhong Pian (Aconite Regulate Middle Pills)</u>.

In the Western herbal tradition, one classic formula mentioned in Benjamin Colby's *Guide To Health* (1846) for dispelling canker is Samuel Thomson's original <u>Composition Powder</u>, given in doses of 5 mL mixed with 150 mL warm water, 2-3 times daily before meals. The <u>Composition Powder</u> is comprised of equal parts:

- bayberry (*Myrica cerifera* bark)
- ginger (*Zingiber officinalis* rhizome)
- cayenne (*Capsicum annuum* fruit)
- cinnamon (*Cinnamomum cassia* bark)
- prickly ash (*Zanthoxylum americanum* bark)

Vidagdha ajirnam

A lack of appetite may also occur if the liver is blocked or stagnant in its function. In Ayurveda this relates to an increase *pitta*, calling for the use of *pitta*-reducing formulas such as <u>Avipattikara churna</u>. In Chinese medicine this blockage of liver function relates to Liver *qi* stagnation, manifesting as a lack of appetite, a bitter taste, belching, acid reflux, borborygmi,

and loose motions. Among the more important formulas to resolve this issue is <u>Xiao Yao San</u> (<u>Rambling Powder</u>), taken in doses of 6-9 g, traditionally taken along with 6 g wei jiang (*Zingiber officinalis*, baked rhizome) and 3 g of bo he (*Mentha haplocalyx* herb). <u>Xiao Yao San</u> (<u>Rambling Powder</u>) is comprised of:

- 9 g chai hu (*Bupleurum falcatum* root)
- 9 g chao dang gui (Angelica sinensis root, dry fried)
- 9 g bai shao (Paeonia alba root)
- 9 g bai zhu (Atractylodes macrocephala root)
- 9 g fu ling (*Poroa cocos* fruiting body)
- 4.5 g zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)

In the Western Herbal tradition, the Thomsonian <u>Spiced Bitters</u> formula has application when the liver is blocked in its function, prepared as fine powder. The <u>Spiced Bitters</u> formulas can also be used in jaundice, and is suggested in loss of appetite with debility and fatigue (*vidagdha ajirnam*). The dose is 5 mL of the powder given with 125 mL hot water, three times a day, before eating:

- 16 parts poplar bark (*Populus alba* bark)
- 4 parts goldenseal (*Hydrastis canadensis* root/rhizome)
- 6 parts prickly ash bark (*Zanthoxylum americanum* bark)
- 4 parts ginger (*Zingiber officinalis* rhizome)
- 4 parts clove (*Syzygium aromaticum* flower bud)
- 2 parts cinnamon (*Cinnamomum cassia* bark)
- 4 parts balmony (*Chelone glabra* root)
- 3 parts- cayenne (*Capsicum frutescens* fruit)
- 40 parts white sugar

Vistamba ajirnam

If the digestive fire is irregular in its function due to an increase in *vata*, one useful formula is <u>Hingwastak churna</u>, given in doses of 2-3 g bid-tid. Another important formula for *vistamba ajirnam* is <u>Trikatu rasayana vati</u>, given in doses of 2 pills twice daily. In Chinese medicine, the character of *vistamba ajirnam* relates to a deficiency of Stomach and Spleen *qi*, calling for formulas such as <u>Bao He Wan (Preserve Harmony Pill)</u>, <u>Liu Jun Zi Tang (Six Gentlemen</u> <u>Decoction</u>), and <u>Bu Zhong Yi Qi Wan (Tonify Middle Augment Essence Decoction</u>).

In the Western herbal tradition the concept of deficiency isn't as fully developed as it is Ayurveda or Chinese medicine, at least as it relates to the concept of restoring vitality to digestive function. Stimulants, carminatives, and cholagogues make up the primary approach to stimulating weak digestion.

Nausea and vomiting

Nausea relates to a feeling of discomfort in the stomach that sometimes precedes the urge to vomit. It is a nonspecific symptom that has a variety of possible causes including emotional distress, motion sickness, dizziness, migraine, fainting, hypoglycemia, pregnancy, infection, or

poisoning. It is a common side effect of many drugs including antacids (e.g. metoclopramide), anti-inflammatories (e.g. acetaminophen), antibiotics, and chemotherapeutic drugs. Vomiting refers to the involuntary, forceful expulsion of the stomach contents, and in most cases promotes the resolution of nausea, and only needs be treated with supportive means. Like diarrhea or excessive sweating, however, vomiting can result in the loss of electrolytes and lead to a life-threatening dehydration (see Gastroenteritis and diarrhea, page 201).

Apart from supportive measures to restore digestion and prevent dehydration, if nausea and/or vomiting persist beyond a couple days specific treatments can be given to resolve the condition. According to each tradition, this includes:

Ayurveda

- pippali (Piper longum fruit) churna
 - Rx: 1-2 g taken with honey, twice daily
- amla (*Phyllanthus emblica* fruit) fresh juice
 Rx: 10-15 mL taken with honey and pink salt
- equal parts katuka (*Picrorhiza kurroa* rhizome) and chitraka (*Plumbago zeylanica* root) decoction
 - Rx: 30-60 mL twice daily
- equal parts nagara (*Cyperus pertenuis* tuber/rhizome) and dhaniya (*Coriandrum sativum* seed) decoction
 - Rx: 60-90 mL bid
- Dashamula kashaya (Ten Roots Decoction)
 - o one part shalaparni (*Desmodium gangeticum* root)
 - o one part prishnaparni (*Uraria picta* root)
 - one part brihati (Solanum indicum root)
 - o one part kantakari (*Solanum xanthocarpum* root)
 - o one part gokshura (Tribulus terrestris root)
 - one part bilwa (*Aegle marmelos* unripe fruit)
 - o one part agnimantha (Premna integrifolia root)
 - one part shyonaka (Oroxylum indicum root)
 - o one part kashmari (*Gmelia arborea* root)
 - o one part patala (Stereospermum suaveolens root)
 - Rx: 60-90 mL bid

Chinese medicine

- Xiao Ban Xia Tang (Minor Pinellia Decoction)
 - harmonizes Stomach, descends Rebellious *qi*, stops vomiting
 - \circ Rx: 200 mL bid
- <u>Xiang Sha Liu Jun Zi Tang (Six Gentleman Decoction with Saussurea and Amomum)</u>
 - o restores/regulates *qi*, strengthens Spleen, harmonizes Stomach
 - decoction, Rx: 200 mL bid
 - o granules, Rx: 3-4 g bid-tid
- <u>Ding Xiang Shi Di Tang (Clove and Persimmon Calyx Decoction)</u>
 - o augments *qi*, warms middle, descends Rebellious *qi*, relieves hiccup
 - o decoction, Rx: 200 mL bid

- o granules, Rx: 3-4 g bid-tid
- <u>Wu Zhu Yu Tang (Evodia Decoction)</u>
 - o warms/restores Spleen and Stomach, descends Rebellious qi, relieves vomiting
 - o decoction, Rx: 200 mL bid
 - o granules, Rx: 3-4 g bid-tid

Unani

- Jawarish Pudina Wilayti
 - o 22 g barg-e-sudab (*Ruta graveolens* leaf)
 - 56 g boora armani (*Bole armeniac* powder)
 - 230 g zanjabeel (Zingiber officinale rhizome)
 - 185 g zeera safaid (*Cuminum cyminum* seed)
 - o 375 g zeera siyah (Carum carvi seed)
 - 175 g filfil siyah (*Piper nigrum* fruit)
 - 7 g agar hindi (Aquilaria agallocha heartwood)
 - 7 g ilaichi khurd (*Elettaria cardamomum* fruit)
 - 7 g ilaichi kalan (Amomum subulatum fruit)
 - 7 g pudina khushk (*Mentha arvensis* herb)
 - 7 g taj qalmi (*Cinnamomum cassia* bark)
 - 7 g jaiphal (Myristica fragrans seed)
 - 7 g qaranfal (*Syzygium aromaticum* flower bud)
 - 300 g anardana (Punica granatum seed)
 - 300 g tamar hindi (*Tamarindus indicus* fruit pulp)
 - o 300 g maweez munaqqa (Vitis vinifera fruit)
 - \circ 7.3 kg qand safaid (sugar)
 - 450 mL sirka desi (vinegar)
 - 10 g sat pudina (menthol)
 - 1100 mL sharbat zanjabeel (syrup)
 - 500 mL aab-e-leemun (lemon juice)
 - confection, Rx: 5 g ad lib
 - used for zof-e-hazm (indigestion), qai (vomiting), imtela (nausea)

Western herbal

- herbs: chamomile flower, black horehound, peppermint leaf, ginger rhizome, cayenne fruit, prickly ash bark, cinnamon bark, clove flower bud, fennel seed
- <u>Antiemetic Drops</u>
 - 15 g powdered cayenne (*Capsicum annuum* fruit)
 - 2 g salt
 - o 250 mL apple cider vinegar
 - 250 mL water
 - 5 mL ad lib

Gastroesophageal reflux disease

Gastroesophageal reflux disease (GERD) is a somewhat newly recognized pathology that describes a condition previously known as "heartburn." GERD is characterized by a burning sensation behind the sternum, often accompanied by regurgitation of the stomach contents into the mouth or lungs. GERD may also manifest as respiratory symptoms such as cough, wheezing, and hoarseness, and may be confused with other conditions such as chronic bronchitis or asthma. Patients with GERD may also present with diminished salivary secretion and an increased risk of tooth decay and gum recession.

The mechanism of GERD is attributed to a dysfunction of the **lower esophageal sphincter (LES)** that lies between the stomach and esophagus. The LES is composed of smooth muscle, and normally only relaxes upon swallowing. With delayed stomach emptying and an increase in gastric pressure, however, the acidified chyme fails to properly clear the esophagus, blocking the function and eventually weakening the integrity of the LES. The acid itself causes damage to the esophageal epithelium, which lacks the alkaline mucous secreted in the stomach, causing esophageal erosion and ulceration. Chronic inflammation may result in the accumulation of scar tissue and a stricture (narrowing) of the esophagus, and there may be a replacement of the normal squamous epithelium with abnormal **columnar (Barrett's) epithelium,** which is considered to be precancerous.

Most episodes of GERD occur during the day, usually after eating, although some sufferers will also experience reflux during sleep. The nocturnal form of GERD is associated with a higher risk of esophagitis because during sleep the patient produces less saliva and swallows less often, which is required clear out and neutralize the acid. Factors that weaken the integrity of the LES include smoking, caffeine, chocolate, fatty foods, overeating, tight clothing, a hiatal hernia, and certain medications are all associated risk factors for GERD.

Medical Treatment

The medical treatment of GERD consists of lifestyle modifications, drug treatments, and surgery. The patient is encouraged to be aware of which foods or activities tend to make the problem worse, such as smoking, or the consumption of caffeinated foods and beverages, chocolate, and fatty foods. Tight clothing around the torso increases intra-abdominal pressure is to be avoided. Body position is also considered to be an important aspect in managing GERD, and recommendations might be made to maintain an upright posture after eating, ensuring that the ingested food does not reflux back into the esophagus. Some patients may be counselled to insert a wedge under their back at night to keep the esophagus above the stomach while sleeping. Similarly, patients are counselled to avoid exertion after a meal, such as bending or lifting, as this contracts the abdominal muscles and forces food back up through the weakened LES. Patients are also recommended to eat in a relaxed manner, and eat smaller meals. Patients that are obese are often a greater risk of GERD because of the excess abdominal fat that puts pressure on the stomach. Similarly, pregnant women often complain of heartburn, simply because of the pressure placed upon the stomach from the growing fetus, but also because hormonal fluctuations tend to make the esophageal and gastric mucosa more sensitive and therefore more reactive.

Gastric impairment leading to GERD can also be found in diabetic patients suffering from gastroparesis, as well as in neurological disorders such as Parkinson's disease. In some cases gastric impairment is a symptom associated with system pathologies such as scleroderma in which the dysfunction of the LES is attributed to autoimmune-induced fibrosis. Commonly used oral medications linked to GERD and gastric disease, include acetyl salicylic acid (ASA) and ibuprofen, which are directly toxic to the gastric mucosa, and well as potassium supplements, and the antibiotic tetracycline that often promote burning sensations in the esophagus.

The classical medical approach to GERD relies use of over-the-counter (OTC) antacid medications such as calcium carbonate that neutralize stomach acid. Although recommended for only occasional use many patients are encouraged or end up using them on a chronic basis, which has a negative effect upon gastric secretion and weakens stomach function. Another similar regimen is the use of bismuth subsalicylate that acts to coat the lining of the stomach and suppress acid secretion. Like other salicylates, however, it is likely that bismuth subsalicylate is also toxic to the gastric mucosa.

Prescription medications include promotility agents, histamine H2-receptor antagonists (H2 blockers), and proton pump inhibitors (PPIs). Promotility drugs such as the drug cisapride, metoclopramide and bethanechol are used to promote gastric motility. Cisapride in particular has since been recognized to have some dangerous effects including ventricular tachycardia and ventricular fibrillation, as well as diarrhea, gastric pain, headaches, and constipation. More commonly, H2 blockers (e.g. cimetidine, famotidine, nizatidine, ranitidine) and PPIs (e.g. omeprazole, lansoprazole, rabeprazole and pantoprazole) are prescribed to reduce the amount of acid produced in the stomach. The theory behind the use of H2 inhibitors and PPIs is that an excess secretion of stomach acid underlies GERD, even though most patients with this condition usually have lower than normal gastric acid levels. Although the use of acid-suppressing agents can give the esophageal epithelium time to heal, they often promote the underlying issue because they weaken gastric function further.

PPIs are among the top-selling drugs in the world, despite the fact that they come with some serious risks. Used long term PPIs can increase the risk of *Clostridium difficile* infection, pneumonia, and interfere with mineral absorption such as magnesium, promoting the risk of muscle spasm, heart palpitation, and convulsion. The general diminishment in nutrient absorption when taking PPIs increases the risk of bone fractures, and also inhibits the production of intrinsic factor, impairing the absorption of vitamin B12 and increasing the risk of pernicious anemia. More recently, the use of PPIs has been linked to an increased risk of dementia, kidney disease, and heart attack.⁴¹

Surgery is an alternative to prescription drugs when treatment is unsuccessful, or when certain complications arise. Negative effects of surgery can occur in up to 20% of patients, such as difficulty swallowing or the inability to belch or vomit. Frequently the benefits are limited, and patients will often require the continued use antacid drugs post-surgery to control symptoms.

⁴¹ Goodman B. *Research Evaluates Possible Link to PPI Risks*. WebMd. June 8, 2016. Available from: http://www.webmd.com/heartburn-gerd/news/20160608/proton-pump-inhibitor-health-risks

Holistic Treatment

From a Western herbal perspective GERD is viewed as an upper GI tract digestive deficiency. Impaired secretion and poor motility leads to a commensurate weakening of the LES, causing the gastric contents to reflux back into the esophagus. Thus to resolve GERD treatment must be directed towards restoring gastric secretion and motility. Nonetheless, temporary measures are often required to neutralize the refluxed acids and promote healing of the esophagus. Generally this is best undertaken by eating small meals of starchy foods with only small amounts of protein, and very little fat (see the graduated diet, p. 182). Flour products, however, which have a glue-like consistency and impair gastric motility should be avoided. Likewise, the basic principles of food combining should also be followed, such as avoiding the consumption of animal proteins with carbohydrates, or eating fruit after meals.

In Ayurveda, GERD is resembles a condition called *amlapitta* ('sour bile'), which describes the reflux of stomach acid (amla) into the esophagus commensurate with an impairment in biliary excretion (pitta). The underlying factor, however, relates to mandagni or weak digestion. Likewise in Chinese medicine, most cases of GERD are caused by Cold and Damp affecting the Spleen, along with Liver qi stagnation, causing Stomach Fire and Stomach yin deficiency. In both Ayurveda and Chinese medicine the goal is to clear the heat, restore gastric digestion, and promote healing. At the outset of treatment this can be achieved by using bitter-tasting herbs that have a mild laxative effective. Bitter-taste in particular has a number of interesting effects on its own, promoting the secretion of gastrin by stimulating chemoreceptors on the tongue. This in turn stimulates the secretion of gastric juices, the closure of the LES, and the opening of the ileo-cecal sphincter, promoting proper motility. Bitter herbs also appear to modulate the secretion of cholecystokinin (CCK), which allows for proper gastric churning and the secretion of bile and pancreatic juices in anticipation of the chyme moving into the duodenum. In Ayurveda, however, too much of the bitter taste tends to inhibit digestion, so bitters need to be used in judicious doses, and are often combined with carminatives and stimulants to offset this effect. Likewise in both Chinese medicine and Ayurveda, herbs with a sweet flavour are used to reduce heat and promote healing, as well as carminative and digestive stimulants to restore proper function to the stomach

The following is a review of the holistic strategy used to address GERD:

Reduce esophageal inflammation

- demulcents: shatavari (*Asparagus racemosa* root), mai men dong (*Ophiopogon japonicus* root), tian men dong (*Asparagus cochinchinensis* root), slippery elm (*Ulmus fulva inner* bark), marshmallow (*Althaea officinalis* root), comfrey (*Symphytum officinalis* leaf/root)
 - prepared as cold infusion or as powders taken with honey
- de-glycyrrhized licorice (DGL): 2–3 tablets, chewed ad lib
- fresh aloe (Aloe vera leaf juice): 15-25 mL bid-qid
- banana, as ripened fruit, banana chips, or leaf powder, ad lib
- alkaline remedies: shankha bhasma (*Turbinella pyrum* calcinated shell ash), shukti bhasma (*Ostrea gigas* calcinated shell ash), pravala bhasma (calcinated coral ash)

- laxative botanicals to purge excess heat, used for a short period during the initial stages of treatment, e.g. turkey rhubarb (*Rheum palmatum* root), cascara sagrada (*Rhamnus purshianus* wood), trivrit (*Operculina turpethum* root)
 - use with carminatives e.g. ginger (*Zingiber officinalis* rhizome), fennel (*Foeniculum vulgare* seed), ajwain (*Trachyspermum ammi* seed)
- astringents, to promote muscular tone of the LES, check bleeding and heal ulcerations, e.g. cranesbill geranium (*Geranium maculatum* root), oak (*Quercus alba* bark), avens (*Geum urbanum* leaf), goldenseal (*Hydrastis canadensis* root/rhizome), bayberry (*Myrica cerifera* bark), goldenrod (*Solidago canadensis* herb), fir (*Abies grandis* bark)
- flavonoids and flavonoid-containing botanicals to limit acid production via inhibiting histamine release, e.g. nettle (*Urtica dioica* leaf), calendula (*Calendula officinalis* flower), amalaki (*Phyllanthus emblica* fruit), meadowsweet (*Spiraea ulmaria* herb), chamomile (*Matricaria chamomilla* flower), huang qian (*Scutellaria baicalensis* root), chai hu (*Bupleurum falcatum* root), green tea extract, quercitin

Promote proper gastric digestion and motility

- bitters, taken in small doses before meals, e.g. barberry (*Berberis vulgaris* root), gentian (*Gentiana lutea* root), centaury (*Erythraea centaurium* root), buckbean (*Menyanthes trifoliata* root), goldenseal (*Hydrastis canadensis* root/rhizome)
 - use of bitters are often avoided at the outset of treatment because the initial stimulation of acid production may worsen any esophageal ulceration
- carminatives and stimulants, to counter the effects of bitter-tasting herbs, and to enhance digestion, e.g. calamus (*Acorus calamus* rhizome), fennel (*Foeniculum vulgare* seed), chamomile (*Matricaria chamomilla* flower), aniseed (*Pimpinella anisum* seed)
 - use caution with pungent botanicals such as ginger and cayenne, and "upward moving" aromatics such as mint (*Mentha* spp.), caraway (*Carum carvi* seed), lavender (*Lavandula angustifolia* flower)
- digestive enzymes, full spectrum (i.e. HCl, pancreatic enzymes, ox bile): 2 3 caps with meals
 - \circ used primarily in the older patients; ideally only short term
- dietary factors
 - food combining: avoid mixing animal proteins with starchy food, fruit should only be consumed on an empty stomach
 - avoid dairy and flour products, which due to their sticky and heavy properties impair gastric motility
 - avoid overeating
 - avoid eating within three hours of bedtime
 - avoid alcohol
 - avoid tobacco
 - \circ avoid deep-fried foods, e.g. French fries, potato chips, etc.
- weight loss, to reduce intra-abdominal pressure

Promote healing of epithelium

• avoid Factors that promote intestinal permeability, p. 166

- vulneraries, e.g. calendula (*Calendula officinalis* flower), plantain (*Plantago spp.* leaf), selfheal (*Prunella vulgaris* leaf), St. John's wort (*Hypericum perforatum* flower), licorice (*Glycyrrhiza glabra* root), chickweed (*Stellaria media* herb)
- bone broth
- nutrients
 - o vitamin A: 25,000-50,000 IU daily
 - vitamin C: 1 2 g bid tid, to bowel tolerance
 - o vitamin E: 800-1200 IU daily
 - zinc citrate: 50 mg daily
 - o methylsulfonylmethane (MSM): 2 3 g, bid tid

Formulations - Ayurveda

- Dhanyapanchakam churna
 - as a general digestive aid with balanced effects
 - o 1-2 grams given with warm water twice daily
- <u>Avipattikara churna</u>, 1-2 grams given with warm water twice daily
 - for *pitta* symptoms
 - 1-2 grams given with warm water twice daily

Formulations – Chinese medicine

- Ban Xia Xie Xin Tang (Pinellia Decoction to Drain the Epigastrum)
 - harmonizes Stomach, directs Rebellious *qi* downwards, disperses distension
 - o granules, Rx: 3-4 g bid-tid
- Mai Men Dong Tang (Ophiopogon Decoction)
 - o benefits Stomach, nourishes *yin*, descends *qi*
 - o decoction, Rx: 200 mL qid
 - o granules, Rx: 2-4 g bid-tid
- <u>Xiao Yao San (Rambling Powder)</u>
 - spreads Liver *qi*, strengthens Spleen
 - powder, Rx: 6-9 g bid-tid with 6 g wei jiang (*Zingiber officinalis*, baked rhizome) and 3 g of bo he (*Mentha haplocalyx* herb)
 - o granules, Rx: 2-4 g bid-tid

Formulations – Unani

- Jawarish-E-Kamooni
 - \circ 70 g zeera siyah (*Carum carvi seed*)
 - 70 g barg-e-sudab (*Ruta graveolens* leaf)
 - 70 g filfil siyah (Piper nigrum fruit)
 - 70 g zanjabeel (*Zingiber officinale* rhizome)
 - 20 g bura-e-armani (silicates of alumina and iron oxide)
 - \circ 1 kg qand safaid (sugar)
 - used for *humuzat-e-meda* (hyperacidity), *fuwaq* (hiccough), *nafkh-e-shikam* (flatulence in the stomach), qabz (constipation)
 - confection, Rx: 10-15 g bid-tid

Formulations – Western herbal

- <u>Robert's Formula</u>
 - o stimulates GI motility, decreases inflammation, decongests mucosa
 - 20-40 gtt bid-tid before meals

Hiatus hernia

The term **hiatus hernia** refers to a protrusion of the stomach above the diaphragm. Technically speaking, the usage of the term "hernia" is incorrect, as a hernia specifically refers to the protrusion of an organ through an *abnormal* opening. In the case of a hiatal hernia it is the protrusion of the fundus of the stomach through the *normal* opening of the esophageal hiatus, a hole that pierces the diaphragm and allows the esophagus to connect to the stomach.

The conventional medical perspective states that the etiology of hiatus hernia is often idiopathic, but in a minority of cases is related to a congenital abnormality or is secondary to trauma, such as a tear in the diaphragm. More recently it has been suggested that a hiatus hernia is caused by the elongation or rupture of the **phrenoesophageal ligament** (PEL), by which the esophagus is attached to the diaphragm, and allowing for the independent movement of the diaphragm and esophagus during respiration and swallowing. This displacement of the PEL allows a portion of the stomach to rise up above the diaphragm. It is thought that the PEL becomes displaced because of an increase in intra-abdominal pressure, which pushes the stomach upwards. Thus anything that increases intra-abdominal pressure, such as bending or lifting, especially after eating, puts an upward pressure upon the stomach, forcing a portion of it through the esophageal hiatus. In a **sliding hiatus hernia** the gastroesophageal junction and a portion of the stomach are pushed up above the diaphragm. In **paraesophageal hiatus hernia** the gastroesophageal junction is in the normal location but a portion of the stomach is adjacent to the esophagus rises above the diaphragm.

Approximately 40% of the population suffers from hiatus hernia, although in many cases these patients are asymptomatic or only occasionally affected (i.e. by overeating, or eating certain foods such as flour products, spicy foods, etc.). A paraesophageal hiatus hernia is generally asymptomatic but unlike a sliding hiatus hernia, may become occluded and strangulate. Small amounts of blood or even a massive hemorrhage may occur with either type of hiatus hernia.

Medical Treatment

The medical treatment of hiatus hernia is in large part similar to the treatment of GERD, relying upon the usage of antacids, acid-secretion inhibitors, and surgery. In the latter case surgical options have a very limited rate of success.

Holistic Treatment

From holistic perspective the etiology of hiatus hernia is similar to that of GERD, and thus all approaches indicated under GERD are applied here as well. As mentioned, the term "hernia" confuses the diagnosis and therefore the treatment options. Fundamentally there are two basic components to hiatus hernia: a structural weakness and misalignment of the muscles in the mediastinum, and either an increase in the intra-abdominal pressure.

The primary cause of the structural problems in hiatus hernia, apart from developmental or constitutional weaknesses, is by poor gastric motility (e.g. constipation) and a chronic increase in intra-abdominal pressure (e.g. obesity). An increase in abdominal pressure can also be caused by over-eating, eating too much fatty food, poor food combinations, and eating hard to digest foods. Poor digestion can lead to alterations in the gastrointestinal microbiome that promote fermentation and gas, distending the stomach and causing it to rise above the esophageal hiatus. Another cause of an increase in intra-abdominal pressure is chronic stress, which causes the diaphragm to be chronically contracted, and abdominal obesity.

To resolve hiatus hernia, measures are taken to ensure proper gastric motility through medication and diet, reducing sympathetic stress, implementing a weight loss regimen, and taking active measures to reduce intra-abdominal pressure. Although medication and herbs can be highly effective in treating hiatus hernia, there are some additional physical techniques that can be employed to push the stomach back down below the esophageal hiatus.

One physical technique is to have the patient drink 750 – 1000 mL of water upon awakening, and then standing erect on their toes, allow all the weight of their body to come down upon the heels. This is repeated ten times, every morning, over several weeks. The idea here is to use the weight of the water to pull the protrusion down and slip the stomach back under the diaphragm. Drinking this much water as well helps to purify the gastrointestinal tract and promote proper motility.

Another physical technique to resolve hiatus hernia is massage, working on the surface of the surrounding tissues with the fingers, and gradually working deeper, softening any muscular tension, ensuring that the patient is able to release any muscle tension in thorax. Care must be taken not to place pressure upon the xyphoid process or the floating ribs during massage. Once the patient has relaxed sufficiently the practitioner can stand behind the patient a place a tennis ball under the ribs, and roll it back and forth along the edge along the diaphragm, pushing into and down against the stomach, once again being careful not to push directly on the xyphoid process. This technique often brings about immediately relief.

Gastritis, and gastric and duodenal ulcers

Gastritis refers to inflammation of the gastric mucosa. This inflammation can be classified as either **acute** or **chronic**, depending on the case history; and either **erosive** or **nonerosive**, based on endoscopic examination or from a histological analysis of the inflammatory cells.

Acute gastritis is a serious and life-threatening condition that usually occurs in very ill patients, typically experienced as a vague abdominal discomfort. Life-threatening clinical manifestations include hemorrhage from the mouth or nose (e.g. "coffee-grounds"), and symptoms of gastric perforation. Histological examination reveals an infiltration of polymorphonuclear leukocytes in the gastric mucosa. The most common cause of **acute erosive gastritis** are NSAIDs (e.g. ibuprofen, naproxen) and other anti-inflammatory drugs such acetyl salicylic acid, as well as alcohol consumption, and acute emotional stress. If the patient has a history of chronic NSAID use they may also present with anemia, which suggests

chronic gastric bleeding. Less common but important causes of acute gastritis include radiation burns, viral infections (e.g. cytomegalovirus), vascular injury, and direct trauma (e.g. nasogastric tubes). In very ill patients the overall mortality can be between 40-50%.

Patients with **chronic erosive gastritis** typically present with a vague dyspepsia, with epigastric pain and nausea, and may even be asymptomatic. Endoscopic examination reveals the presence of multiple punctate or apthous ulcers, while further histological examination will show some degree of atrophy or metaplasia. If it involves the antrum of the stomach there will be a loss of G cells and thus decreased gastrin secretion, whereas if it involves the corpus, there may be a loss of the fundic glands, with reduced HCl, pepsin, and intrinsic factor secretion. The causes of chronic gastritis are usually the same as for its acute forms.

Nonerosive gastritis is typically associated with *Helicobacter pylori*, a spiral-shaped, gramnegative bacteria that thrives in the high acid environment of the stomach. First identified by medical researchers in 1982, it is thought that infection with *H. pylori* invariably leads to gastric mucosal inflammation, which in turn alters gastric secretion and leaves the mucosa susceptible to damage by stomach acid. Normally the low pH of the stomach prevents microbial invasion, but *H. pylori* uses its flagella to burrow deep into the mucosal lining of the stomach to reach the epithelial cells underneath where the pH is more neutral. *H. pylori* can also secrete the enzyme urease, which breaks down urea present in the stomach into CO_2 and NH4⁺ (ammonia), which serves as a base to neutralize stomach acid. The highest concentrations of *H. pylori* are typically found in the antrum of the stomach. Beyond its association with gastritis, individuals infected with *H. pylori* have a 10–20% increased risk of developing peptic ulcers, and a 1–2% increased risk of stomach cancer.

H. pylori infection appears to be exceptionally common, and despite being fingered as a pathogen, research shows that over 80% of individuals infected with the bacteria are asymptomatic, suggesting that *H. pylori* is a commensal and only becomes pathogenic in certain cases. Another explanation is that different strains of *H. pylori* have different degrees of virulence. Although the exact mode of transmission is unclear, the organism has been cultured from stool, saliva, and dental plaque, suggesting an oral-oral or fecal-oral transmission. Infections tend to predominate in families and in hospital workers, frequently found in nurses and gastroenterologists (Carroll 2005; Sepulveda 2005b).

Autoimmune factors can also promote chronic nonerosive gastritis. Auto-antibodies are produced against the parietal cells and intrinsic factor, leading to a reduction or complete inhibition of gastric secretion. Further investigation usually reveals increased serum gastrin (due to G-cell hyperplasia in the antrum) and gastric enterochromaffin-like hyperplasia due to gastrin stimulation (Sepulveda 2005b).

The mildest form of nonerosive gastritis is **superficial gastritis**, which can yield very mild symptoms. The inflammatory process can involve all parts of the stomach but is not accompanied by atrophic or metaplastic changes within the mucosa. Upon histological examination the infiltrating cells are usually found to be lymphocytes, plasma cells, and neutrophils.

Atrophic gastritis results from chronic, superficial gastritis, typically presenting as a definite but nonetheless mild dyspepsia. Histological examination yields lymphocytes and plasma cells often penetrating the mucosa all the way to the muscularis layer of the stomach. This condition also involves degenerative changes in HCl and pepsin secreting cells leading to a loss of gastric secretion including intrinsic factor, which in turn, promotes a vitamin B_{12} deficiency (Sepulveda 2005a).

Gastric metaplasia is common in chronic nonerosive gastritis, typically occurring with the severe atrophy of the gastric glands, which are progressively replaced by mucous glands, typically in the antral mucosa. The gastric mucosa may resemble the mucosa of the small intestine, with goblet cells, endocrine (enterochromaffin or enterochromaffin-like) cells, and rudimentary villi, and may even assume functional (absorptive) characteristics. In complete metaplasia, gastric mucosa is completely transformed into small intestinal mucosa, with the ability to absorb nutrients and secrete peptides. In incomplete metaplasia, the epithelium assumes a histologic appearance closer to that of the large intestine and frequently exhibits dysplasia. Both cell types are associated with an increased risk of stomach cancer (Sepulveda 2005b).

Peptic ulcer disease refers to an excoriated segment of the gastrointestinal mucosa, either in the stomach (gastric ulcer) or the duodenum (duodenal ulcer), that can progress to penetrate through the muscularis layer of the stomach. The ulcers may be one or more, and may range in size from several millimeters to several centimeters. The etiology of peptic ulcer disease was at one time considered to be due to excess gastric acid secretion but this theory has given way to factors that disrupt normal mucosal defense mechanisms, including *H. pylori* infection and NSAID use, which makes the mucosa more susceptible to the effects of the stomach acid. Gastric ulcers typically arise from chronic nonerosive gastritis, and in most cases patients secrete less acid than healthy individuals.

As previously discussed, the mechanism by which *H. pylori* promotes damage to the gastric mucosa relates to the synthesis of the enzyme urease that breaks down urea into ammonia, which then serves to erode the mucous membrane. *H. pylori* has also been shown to produce cytotoxins that promote epithelial damage, as well as mucolytic enzymes such as protease and lipase, inhibiting the mucosal barrier. In turn, this causes the damaged gastric mucosa to release cytokines that promote further damage and ulceration as part of a vicious cycle.

NSAIDs promote damage to the gastric mucosa by exerting a directly toxic effect as well as indirectly by modifying the host response. When ingested NSAIDs diffuse into gastric epithelial cells and promote the release of H^+ ions, promoting mucosal injury. NSAIDs also inhibit the cyclooxygenase pathway and the synthesis of prostaglandins that maintain gastric integrity. The interruption of this metabolic pathway results in significant changes to the gastric mucosa, inhibiting blood flow, reducing mucus and HCO_3 secretion, and inhibiting cellular repair mechanisms.

The signs and symptoms of peptic ulcer disease can vary. While in some cases, such as in the elderly, the pain can be minimal or even absent, others can experience a burning pain in the epigastrum that refers to the back. In some cases the pain is relieved by eating, whereas for

others eating makes little difference. Very often the symptoms come and go. Gastric ulcers are often associated with symptoms of obstruction such as bloating, nausea, and vomiting. In duodenal ulcers the pain tends to be consistent, frequently absent when the patient awakens but appearing later in midmorning. The pain of duodenal ulcers is often relieved by food but recurs within a few after eating.

The most common complication of peptic ulcer disease is hemorrhage, and occurs in up to 20% of patients. Symptoms include the vomiting of blood ("coffee grounds"), occult blood in the stools, and iron-deficiency anemia. The penetration of the gastric or duodenal wall by an ulcer results in stomach acids leaking into the abdominal cavity, causing peritonitis, which can attack the pancreas or liver. The overall mortality rate for perforated ulcers is 10-40% with gastric ulcers, and 5-13% with duodenal ulcers.

Medical Treatment

The medical treatment of gastritis and peptic ulcer disease doesn't differ all that much from GERD or hiatus hernia, with a reliance upon suppressing acid secretion through the use of antacids, bismuth subsalicylate, H2 antagonists, and proton pump inhibitors. This is despite the fact that patients with gastritis and ulcers have been shown to have lower gastric acid levels than normal. With the attribution of *H. pylori* as the etiology for gastric and peptic ulcer disease, however, it is now common for these drugs to be used along with one or more antibiotics such as metronidazole, tetracycline, clarithromycin, or amoxicillin, referred to as "triple therapy." The claim that this treatment has an 80-90% cure rate must be regarded with some skepticism. In 1997 a follow-up study of patients that had received antibiotic therapy for *H. pylori* over a two year period indicated an overall treatment failure rate of 23%, with *H. pylori* showing high levels of resistance to clarithromycin (30%), metronidazole (66%), or to both of these antibiotics (23%) (McMahon, et al. 2002). Patients that have undergone successful treatment for *H. pylori* may find that the initially positive results are replaced by a gradual onset of recurring symptoms. As for the usage of antibiotics, treatment will almost certainly result in the destruction of beneficial bacterial strains and in some cases, may provoke esophageal disease or gastric cancer of the cardia (Hunt et al 2001). The larger issue of antibiotic resistance, which is increasingly shown to be an important and vital issue, must also be taken into account.

Holistic treatment

The treatment of gastritis and peptic ulcer disease is similar to that of GERD and hiatus hernia although the severity of symptoms are usually worse, along with the risk that this condition can be life-threatening, as is the case of a perforating ulcer. Another important factor to take into account is the loss of intrinsic factor and chronic bleeding, which leads to the development of anemia.

In Ayurveda gastric and duodenal ulcers are classified as *amlapitta*, and are caused by eating incompatible foods or foods that promote burning sensations, as well as general factors that lead to the increase and vitiation of *pitta*. Likewise in Chinese medicine, irregular eating and the consumption of excessively greasy foods and alcohol can result in Stomach Fire. Stagnation of the Liver *qi*, often caused by emotions such as anger, depression and anxiety, can also obstruct the Stomach *qi* and result in Stomach Fire.

The following is a review of the holistic strategy used to address gastritis, gastric ulcer, and duodenal ulcer:

Reduce gastric inflammation

- demulcents: shatavari (*Asparagus racemosa* root), mai men dong (*Ophiopogon japonicus* root), tian men dong (*Asparagus cochinchinensis* root), slippery elm (*Ulmus fulva inner* bark), marshmallow (*Althaea officinalis* root), comfrey (*Symphytum officinalis* leaf/root)
 - prepared as cold infusion or as powders taken with honey
- de-glycyrrhized licorice (DGL): 2–3 tablets, chewed ad lib
- fresh aloe (*Aloe vera* leaf juice): 15-25 mL bid-qid
- banana, as ripened fruit, banana chips, or leaf powder, ad lib
- alkaline remedies: shankha bhasma (calcinated *Turbinella pyrum* shell ash), shukti bhasma (calcinated *Ostrea gigas* shell ash), pravala bhasma (calcinated coral ash)
- laxative botanicals to purge excess heat, used for a short period during the initial stages of treatment, e.g. turkey rhubarb (*Rheum palmatum* root), cascara sagrada (*Rhamnus purshianus* wood), trivrit (*Operculina turpethum* root)
 - use with carminatives e.g. ginger (*Zingiber officinalis* rhizome), fennel (*Foeniculum vulgare* seed), ajwain (*Trachyspermum ammi* seed)
- astringents, to check bleeding and heal ulcerations, e.g. cranesbill geranium (*Geranium maculatum* root), oak (*Quercus alba* bark), avens (*Geum urbanum* leaf), goldenseal (*Hydrastis canadensis* root/rhizome), bayberry (*Myrica cerifera* bark), goldenrod (*Solidago canadensis* herb), fir (*Abies grandis* bark)
- flavonoids and flavonoid-containing botanicals to limit acid production via inhibiting histamine release, e.g. nettle (*Urtica dioica* leaf), calendula (*Calendula officinalis* flower), amalaki (*Phyllanthus emblica* fruit), meadowsweet (*Spiraea ulmaria* herb), chamomile (*Matricaria chamomilla* flower), huang qian (*Scutellaria baicalensis* root), chai hu (*Bupleurum falcatum* root), green tea extract, quercitin

Promote proper gastric digestion

- stimulants, to enhance local blood flow and healing, e.g. cayenne (*Capsicum annuum* pod), shunthi (*Zingiber officinalis* rhizome), pippali (*Piper longum* fruit), maricha (*Piper nigrum* fruit), tumburu (*Zanthoxylum alatum* pericarp/bark)
- bitters, taken in small doses before meals, e.g. barberry (*Berberis vulgaris* root), gentian (*Gentiana lutea* root), centaury (*Erythraea centaurium* root), buckbean (*Menyanthes trifoliata* root), goldenseal (*Hydrastis canadensis* root/rhizome)
 - use of bitters are often avoided at the outset of treatment because the initial stimulation of acid production may worsen ulceration
- antispasmodics, used in chronic conditions to ease spasm, pain and cramping, e.g. calamus (*Acorus calamus* rhizome), fennel (*Foeniculum vulgare* seed), chamomile (*Matricaria chamomilla* flower), aniseed (*Pimpinella anisum* seed), wild yam (*Dioscorea villosa* root), crampbark (*Viburnum opulus* bark), kava (*Piper methysticum* root bark), lobelia (*Lobelia inflata* herb), hops (*Humulus lupus* strobiles)
 - o care must be taken if acid reflux is a part of the clinical presentation
- dietary factors

- food combining: avoid mixing animal proteins with starchy food, fruit should only be consumed on an empty stomach
- avoid dairy and flour products, which due to their sticky and heavy properties impair gastric motility
- avoid overeating
- avoid eating within three hours of bedtime
- avoid alcohol
- \circ avoid tobacco
- $\circ~$ avoid deep-fried foods, e.g. French fries, potato chips, etc.
- avoid overeating, do not eat within three hours of bedtime
- promote weight loss, to reduce intra-abdominal pressure

Reduce insult to gastric and duodenal epithelium

- avoid Factors that promote intestinal permeability, p. 166
- reduce exposure to xenobiotics such as pesticides, insecticides and herbicides, emphasizing organically grown vegetables and free range, hormone/antibiotic-free animal produce
- increase intake of high fiber foods and foods rich in antioxidant phytochemicals such as broccoli, cabbage, cauliflower, beets, carrots and onions
- avoid NSAIDs
 - o increase intake of n-3 fatty acids
 - foods such as wild salmon, halibut, arctic char, smelt, herring, mackerel
 - supplement with EPA/DHA (triglyceride source, 60:40), 2-3 g daily
 - simultaneously reduce consumption of n-6 fatty acids, i.e. most seed oils
- ensure a balance of macro and trace minerals to provide for the synthesis of detoxification enzymes and antioxidants, e.g. seaweed, bone broth

Promote healing of epithelium

- vulneraries, e.g. calendula (*Calendula officinalis* flower), plantain (*Plantago spp.* leaf), selfheal (*Prunella vulgaris* leaf), St. John's wort (*Hypericum perforatum* flower), licorice (*Glycyrhiza glabra* root), chickweed (*Stelaria media* herb)
- bone broth
- nutrients
 - o vitamin A: 25,000-50,000 IU daily
 - \circ vitamin C: 1 2 g bid tid, to bowel tolerance
 - o vitamin E: 800-1200 IU daily
 - zinc citrate: 50 mg daily
 - methylsulfonylmethane (MSM): 2 3 g, bid tid

Address the underlying anemia

• herbs that nourish the blood, e.g. shilajit, amalaki (*Phyllanthus emblica* fruit), shatavari (*Asparagus racemosa* root), ashwagandha (*Withania somnifera* root), shu di huang (*Rehmannia glutinosa* root, stir-fried in wine), dang gui shen (*Angelica sinensis* root, stir-fried in wine), bai shao (*Paeonia lactiflora* root), da zao (*Ziziphus jujuba* fruit), gou qi zi (*Lycium barbarum* fruit), yellowdock (*Rumex crispus* root), nettle (*Urtica dioica* leaf)

- lauha bhasma (calcinated iron oxide ash): 125-250 mg bid, taken with honey, ghee, or <u>Trikatu churna</u>, <u>Triphala churna</u>, or haridra (*Curcuma longa* rhizome juice)
- foods: red meat, egg yolk, liver, prunes, figs, leafy greens, garlic
- vitamin C: 2 3 g daily
- vitamin B complex: 100 mg daily
- avoid elemental iron supplementation as this tends to promote intestinal infection and constipation

Restore the gastrointestinal ecology

- probiotics: Lactobacillus, Bifidobacterium, live culture foods
- prebiotics: FODMAP foods

Formulations - Ayurveda

- <u>Punarnavasava</u>
 - for *amlapitta* (gastric ulcer)
 - Rx: 12 24 mL bid-tid
- <u>Mahatiktaka ghrita</u>
 - for *amlapitta* (gastric ulcer)
 - Rx: 3-6 g bid-tid
- Avipattikaryadi churna
 - o 10 parts <u>Avipattikara churna</u>
 - 4 parts shankha bhasma (calcinated *Turbinella pyrum* shell ash)
 - 2 parts pravala bhasma (calcinated coral ash)
 - o 2 parts shukti bhasma (calcinated *Ostrea gigas* shell ash)
 - 1 part churnahh bhasma (calcinated limestone ash)
 - for *amlapitta* (gastric ulcer)
 - Rx: 12 g bid with milk or water
- <u>Samudradya churna</u>
 - 1 part samudra lavana (sea salt)
 - 1 part saindhava (rock salt)
 - 1 part yavakshara (*Hordeum vulgare*, purified ash of grass)
 - o 1 part swarjikshara (sodium bicarbonate)
 - 1 part sauvarchala (black salt)
 - o 1 part romaka lavana (sambhar salt)
 - 1 part vida lavana (salt made from *Phyllanthus emblica* fruit)
 - 1 part danti (Baliospermum montanum root)
 - 1 part lauha bhasma (calcinated iron oxide ash)
 - 1 part mandura bhasma (calcinated iron)
 - 1 part trivrit (*Operculina turpethum* root)
 - 1 part suranaka (Amorphophallus campanulatus stem tuber)
 - 1 part dadhi (yogurt)
 - 1 part gomutra (cow's urine)
 - 1 part godugdha (cow's milk)
 - for *parinama shula* (duodenal ulcer)
 - Rx: 1-2 g bid

- <u>Shulanirmulanarasa vati</u>
 - <u>Trikatu</u> (*Piper longum* fruit, *Piper nigrum* fruit, *Zingiber officinalis* rhizome)
 - o gandhaka (purified sulfur)
 - o shankha bhasma (calcinated *Turbinella pyrum* shell ash)
 - saindhava (mineral salt)
 - rasasindura (red crystal of mercury sulphide)
 - o jiraka (*Cuminum cyminum* seed)
 - o amlavetasa (*Rheum emodi* root)
 - o kuchila (*Strychnos nuxvomica* seed ground with *Zingiber officinalis* rhizome juice)
 - for parinama shula (duodenal ulcer)
 - Rx: 3 pills bid

Formulations - Chinese medicine

- Ban Xia Xie Xin Tang (Pinellia Decoction to Drain the Epigastrum)
 - harmonizes Stomach, directs Rebellious *qi* downwards, disperses distension
 - o granules, Rx: 3-4 g bid-tid
- Mai Men Dong Tang (Ophiopogon Decoction)
 - o nourishes *yin*, directs Rebellious *qi* downwards
 - o decoction, Rx: 200 mL bid-tid
 - o granules, Rx: 3-4 g bid-tid
- <u>Wei Te Ling (Stomach Special Remedy)</u>
 - harmonizes Stomach, clears Stomach Fire, stops bleeding and pain
 - Rx: 4-6 tablets bid-tid
- <u>Yunnan Baiyao</u>
 - for ulceration with bleeding
 - Rx: 2 capsules tid qid

Formulations - Unani

- Jawarish-E-Kamooni
 - o confection, Rx: 10-15 g bid-tid
- <u>Habb-E-Raal</u>
 - o 100 g raal (Vateria indica resin)
 - 100 g samagh-e-arabi (*Acacia arabica* gum)
 - used for qurooh-e-meda (gastric ulcer), qurooh-e-isnaashri (duodenal ulcer), ishal (diarrhoea)
 - pill (500 mg), Rx: 1-2 pills bid, after meals

Formulations – Western herbal

- <u>Neutralizing cordial</u>
 - 80 mL *Rheum officinalis* root tincture
 - 64 mL Cinnamomum cassia bark tincture
 - 40 mL *Hydrastis canadensis* root/rhizome tincture
 - 20 gtt peppermint E.O.
 - \circ 16 g potassium carbonate (KCO₃)
 - o 250 mL sugar syrup

- 550 mL diluted alcohol (50%)
 - Rx: 1 tsp bid-qid
- <u>Robert's Formula</u>
 - Rx: 20 gtt tid in water, before meals
- Dr. Chistopher's <u>Ulcer Formula</u>
 - one part bayberry
 - one part chickweed
 - o one part slippery elm
 - o one part mullein
 - powder, Rx: 3-5 g taken with an infusion of chamomile (*Matricaria chamomilla* flower) or hops (*Humulus lupus* strobiles)

Gastroenteritis and diarrhea

Gastroenteritis refers to the inflammation of the lining of the stomach and intestines, manifested by symptoms such as anorexia, nausea, vomiting, as well as diarrhea and abdominal pain. Gastroenteritis is a general term for these symptoms, and the cause in modern medicine is denoted by a descriptive term such as 'bacterial,' or 'viral.' In most cases diarrhea is a relatively benign and self-limiting condition, but in debilitated patients and children it can be fatal due the loss of electrolytes and the resultant dehydration.

There are a number of pathological mechanisms involved in **bacterial gastroenteritis**, including organisms such as *Vibrio cholerae* (cholera) and *Escherichia coli* that synthesize and release **enterotoxins** that impair intestinal absorption and stimulate the secretion of electrolytes and water into the lumen of the bowel. Other bacteria such as *Shigella* (shigellosis) and *Salmonella* (salmonellosis) actually penetrate into the mucosa of the intestine to cause ulceration and bleeding, resulting in the secretion of pus, electrolytes, and water.

Campylobacter jejuni is a common bacterial cause of gastroenteritis in North America, often acquired from domesticated animals and improperly prepared food. Person-to-person transmission is especially common with gastroenteritis caused by *Shigella* and *Escherichia coli*. Likewise *Salmonella* infection may be acquired through contaminated food or water, but also from contact with reptiles (e.g. frogs, turtles) or from insects (e.g. cockroaches).

Clostridium difficile is a spore-forming bacterium that causes diarrhea, fever, nausea, and abdominal pain. It is spread via the oral-fecal route, and can result in severe colonic inflammation that may lead to perforation and sepsis. It is often acquired by older patients in a hospital environment, in the immunocompromised, and in those taking PPIs. "C diff" is resistant to most antibiotics and forms long-lived, heat-resistant spores that are killed neither by alcohol nor routine surface cleaning.

Causes of **viral gastroenteritis** include the novovirus, rotaviruses, adenoviruses, astroviruses, and caliciviruses. The most common viral pathogen is the Norwalk virus, the only species of norovirus, thought to be responsibe for 90% of epidemic nonbacterial outbreaks of gastroenteritis around the world, and up to 50% of all foodborne outbreaks of gastroenteritis.

Epidemics of viral diarrhea are typically spread via contaminated water or food or via the fecal-oral route. Norwalk virus infections occur year-round, whereas during the winter in temperate climates rotaviruses are the major cause of viral gastroenteritis.

A number of **intestinal parasites** can also cause gastroenteritis by invading the bowel mucosa. *Giardia lamblia* is a protozoan that infects the crypts of the intestinal villi, causing symptoms that range from mild colic and poorly formed stools, to severe cramping, diarrhea, and fever. Infection can persist for a number of years, causing chronic diarrhea, flatulence, malabsorption, and weight loss. Both humans and animals are reservoirs for *Giardia*, spread by the fecal-oral route. **Giardiasis** or 'beaver fever' is common in the colder climates of North America, Europe and Asia, and is typically acquired by drinking water from contaminated creeks and streams.

Another protozoal infection is **cryptosporidiosis**, caused by *Cryptosporidium parvum*. It is usually acquired by drinking contaminated water, and promotes a watery diarrhea sometimes accompanied by abdominal cramps, nausea, and vomiting. It is usually mild and self-limiting in healthy individuals, but in the immunocompromised, as well as young children and the elderly, the infection can be severe causing significant electrolyte and fluid loss.

Entamoeba is a group of amoebas that cause **amoebiasis**, also known **amoebic dysentery**. Infection causes symptoms such as bloody diarrhea that can result in perforation of the bowel and life-threatening peritonitis. In chronic conditions the patient often develops anemia due to a loss of blood. It is usually transmitted via the fecal-oral route, found more often in areas of poor sanitation, particularly in warm climates.

Non-infectious causes of gastroenteritis include the ingestion of toxins found in fungi and plants (e.g. poisonous mushrooms, potato leaves), heavy-metals (e.g. arsenic, lead, mercury and cadmium), and antibiotics, the latter of which promote GI dysfunction by altering the GI microbiome.

The signs and symptoms of gastroenteritis vary to a large degree, depending on host resistance, the virulence or toxic nature of the etiological agent, and the duration of its activity. Symptoms of gastroenteritis often have a sudden onset, and can include anorexia, nausea, vomiting, borborygmi, colic, and diarrhea with or without blood and mucus. Other symptoms may include fatigue and lethargy, muscle aches, and fever. With either vomiting or diarrhea excessive fluid loss can occur, promoting symptoms of dehydration including muscular spasm, nervous irritability, shock, vascular collapse, and renal failure.

Medical treatment

The treatment of simple diarrhea is usually addressed by the use of anti-diarrheals such as loperamide hydrochloride that inhibits peristaltic activity. Other similar interventions include bismuth subsalicylate, diphenoxylate, codeine phosphate, camphorated opium tincture, atropine, propantheline, psyllium, kaolin, pectin, and activated attapulgite (a clay mixture containing silicon, aluminum and iron oxides). As diarrhea is a symptom of gastrointestinal infection, however, the primary focus of treatment is to inhibit the pathogenic agent, and even more importantly, control electrolyte loss and prevent dehydration.
A number of antimicrobials are used in the treatment of gastroenteritis, including tetracycline, metronidazole, and ciprofloxacin, although their benefit is sometimes quite limited, and may even prolong certain infections such as salmonellosis.

With regard to dehydration, the World Health Organization (WHO) has developed an oral rehydration therapy (ORT) formula to restore electrolytes, comprised of:

- 2.6 g sodium chloride
- 2.9 g trisodium citrate dihydrate
- 1.5 g potassium bicarbonate
- 13.5 g glucose
- 1 liter purified water

While ORT can be life-saving, an earlier version of this formula was criticized for containing too much sodium which may cause hypernatremia. The WHO ORT formula also requires that the water is purified to avoid re-infection, which can be a challenge in certain situations, and especially among the poor. Seeking inspiration from traditional remedies used to treat diarrhea, researchers have found that rice starch is highly effective in lessening the severity of diarrhea and to prevent dehydration, and in many cases is more effective than the standard WHO formula (Hossain et al 2003; Sharma et al 1998; Bhattacharya et al 1998). Specifically, it appears that the rice starch blocks the chloride channel and inhibits the flow of water into the lumen of the bowel (Matthews et al 1999; Goldberg et al 1996).

Increasingly there has been a recognition among medical professionals with respect to the importance of the gut microbiome, and a growing body of evidence that demonstrates the clinical benefits of probiotics in the treatment of gastroenteritis, and in particular, bacterial species such as *Lactobacillus rhamnosus* and *Saccharomyces boulardii* (Szajewska et al. 2014). Likewise, it is very clear that a reliance upon antibiotics and chemical hygiene in modern times has dramatically increased the prevalence of pathogens such as *Clostridium difficile*, underscoring the importance of maintaining a healthy gut microbiome.

Holistic Treatment

The holistic treatment of gastroenteritis is dependent upon the nature and quality of the signs and symptoms. Beyond the issue of diarrhea and risk of dehydration, patients will often exhibit a particular pattern of signs and symptoms. In Ayurveda there are three basic forms of gastroenteritis, each relating to one of the three *doshas* (and combinations thereof), as well as emotional factors such as fear and shock. In Chinese medicine the diagnosis and treatment of gastroenteritis can be differentiated into four basic patterns, between hot/cold, and excess/deficiency.

In both systems gastroenteritis relates to an underlying digestive weakness, related to the concept of *ama* in Ayurveda and Dampness in Chinese medicine. In this sense, the disease itself is a natural response to this encumbrance, and measures should be taken to support the process, inasmuch as it serves as a kind of 'reset' for good digestive health. In this regard, active measures to inhibit diarrhea are not employed at the outset of treatment. To inhibit

diarrhea too early is the cause of several disorders in Ayurveda, most prominent of which are chronic digestive disorders. In most cases the diarrhea will gradually resolve as long as proper hydration is maintained. In acute cases, however, marked by severe inflammation with copious diarrhea, blood and/or pus, agents that are both antimicrobial and astringent may be indicated at the outset of treatment.

The following is a review of the holistic strategy used to address gastroenteritis:

Restore electrolytes and prevent dehydration

- lightly salted rice soup (i.e. kanjika or congee, see p. 182) prepared at a 1:8 ratio
- WHO ORT
 - o alternate is an infusion of potassium-rich dandelion leaf (one liter)
 - ½ tsp sea salt
 - 1 tsp lemon or lime juice
 - 6 tsp table sugar

Ease spasm and griping

- carminatives, e.g. ajwain (*Trachyspermum ammi* seed), aniseed (*Pimpinella anisum* seed), fennel (*Foeniculum vulgare* seed), caraway (*Carum carvi* seed), calamus (*Acorus calamus* rhizome), ginger (*Zingiber officinalis* rhizome), prickly ash (*Zanthoxylum spp.* bark/seed), cardamom (*Elettaria cardamomum* seed), nutmeg (*Myristica fragrans* seed)
- antispasmodics, e.g. bilva (*Aegle marmelos* unripe fruit), hingu (*Ferula narthex* gum), musta (*Cyperus rotundus* rhizome/tuber), bai shao (*Paeonia lactiflora* root), chen pi (*Citrus reticulata* pericarp), shan za (*Crataegus pinnatifida* fruit), wild yam (*Dioscorea villosa* root), crampbark (*Viburnum opulus* bark), poppy (*Papaver somniferum* capsule), cannabis (*Cannabis indica* flower), hops (*Humulus lupus* strobiles)

Restore balance to the gut microbiome

- berberine and related isoquinoline alkaloid-containing botanicals, e.g. Oregon grape (*Mahonia spp.* root), barberry (*Berberis spp.* root), goldenseal (*Hydrastis canadensis* root/rhizome), coptis (*Coptis spp.* rhizome/root)
- other antimicrobial agents, e.g. kutaja (Holarrhena antidysenterica bark), haritaki (*Terminalia chebula* fruit decoction), garlic (*Allium sativum* bulb), sweet annie (*Artemisia annua* herb), black walnut (*Juglans nigra* green hull), nimba (*Azadirachta indica* leaf/bark), quassia (*Quassia amara* wood), pau d'arco (*Tabebuia spp.* bark)
- probiotics, introduced after appetite has improved and symptoms diminished
 - o live culture foods, e.g. yogurt, buttermilk (*takra*), lacto-fermented pickle brine
 - o supplements: Lactobacillus, Bifidobacterium
 - Saccharomyces boulardii in Clostridium difficile
- avoid yeasted foods, e.g. fruit, bread, wine, beer, kombucha/tibicos, etc.
- avoid all refined sweeteners and sweet/dried fruit

Decrease bowel movement frequency

• botanicals to astringe the mucosa, promote tissue healing and reduce the volume of the stool, e.g. bilva (*Aegle marmelos* unripe fruit), dadima (*Punica granatum* rind),

raspberry/blackberry (*Rubus spp.* root), cinnamon (*Cinnamomum cassia* bark), goldenseal (*Hydrastis canadensis* root/rhizome), haritaki (*Terminalia chebula* fruit decoction), bayberry (*Myrica cerifera* bark), agrimony (*Agrimonia eupatoria* herb), cranesbill geranium (*Geranium maculatum* root), oak (*Quercus alba* bark)

- such remedies should only be used 3-4 days after the onset of symptoms if not resolved
- hydrophilic compounds, e.g. psyllium, pectin, activated charcoal, bentonite clay

Formulations for acute gastroenteritis - Ayurveda

- bilva (*Aegle marmelos* unripe fruit) powder with 10% shankha bhasma (calcinated *Turbinella pyrum* shell ash)
 - 1-2 g twice daily
- amra (Magnifera indica seed, roasted in a fire) powder
 - o one seed, taken in divided doses, 2-3 times per day
- <u>Yavanyadi churna</u>
 - o 1 part yavani (*Trachyspermum ammi* seed)
 - 1 part pippalimula (*Piper longum* root)
 - 1 part tvak (Cinnamomum verum bark)
 - 1 part ela (*Elettaria cardamomum* seed)
 - 1 part patra (Cinnamomum tamala leaf)
 - 1 part nagakeshara (*Messua ferrea* flower)
 - 1 part maricha (Piper nigrum fruit)
 - 1 part chitraka (Plumbago zeylanica root)
 - 1 part hrivera (Pavonia odorata herb)
 - 1 part shwetajiraka (Cuminum cyminum seed)
 - 1 part dhaniya (*Coriandrum sativum* seed)
 - 3 parts sauvarchala (black salt)
 - 3 parts vrikshamla (*Garcinia indica* fruit pulp)
 - 3 parts dhataki (Woodfordia fruticosa flower)
 - 3 parts pippali (Piper longum root)
 - 3 parts dadima (Punica granatum pericarp)
 - 3 parts ajamoda (*Trachyspermum roxburghianum* seed)
 - 6 parts sita (sugar)
 - 8 parts kapittha (Feronia limonia pericarp)
 - Rx: 2-4 g bid-tid, with buttermilk
- <u>Kutaja arishta</u>
 - 4.8 kg kutaja (Holarrhena antidysenterica root bark)
 - 2.4 kg draksha (Vitis vinifera dried fruit)
 - 480 g madhuka (*Madhuca indica* flower)
 - 480 g kashmari (*Gmelia arborea* root)
 - o 960 g dhataki (Woodfordia fruticosa flower)
 - 4.8 kg sugar
 - 12 -24 mL twice daily

Formulations for acute gastroenteritis - Chinese medicine

<u>Bai Tou Weng Tang (Pulsatilla Decoction)</u>

- clears Heat, alleviates dysentery
- decoction, Rx: 200 mL bid-tid
- granules, Rx: 3-5 g bid-tid
- Huang Qin Tang (Baikal Skullcap Decoction)
 - clears Heat, alleviates dysentery
 - decoction, Rx: 200 mL bid-tid
 - granules, Rx: 3-4 g bid-tid

In cases of chronic diarrhea the approach taken is similar, but milder, with a focus on the underlying issue of *ama* or Dampness. In this case there is a greater emphasis placed upon carminatives, digestive stimulants, digestive restoratives, and hepatics to restore digestion, along with antispasmodics to alleviate pain and discomfort.

Formulations for chronic gastroenteritis - Ayurveda

- <u>Musta arishta</u>
 - 9.6 kg musta (*Cyperus rotundus* tuber/rhizome)
 - o 768 g dhataki Woodfordia fruticosa –
 - o 768 g yavani (*Trachyspermum ammi* seed)
 - 96 g shunthi (Zingiber officinalis rhizome)
 - 96 g maricha (*Piper nigrum* seed)
 - 96 g lavanga (*Syzygium aromaticum* flower bud)
 - 96 g methika (*Trigonella foenum-graceum* seed)
 - 96 g chitraka (Plumbago zeylanica root)
 - o 96 g krishna jiraka (*Nigella sativa* seed)
 - \circ 14.4 kg guda (sugar)
 - Rx: 12-24 mL bid-tid
- <u>Shambhukadi vati</u>
 - o shankha bhasma (calcinated *Turbinella pyrum* shell ash)
 - maricha (*Piper nigrum* seed)
 - dhaniya (Coriandrum sativum seed)
 - o pippali (*Piper longum* fruit)
 - o shunthi (Zingiber officinalis rhizome)
 - o jiraka (*Cuminum cyminum* seed)
 - o amalaki (Phyllanthus emblica fruit)
 - hingu (Ferula narthex gum)
 - saindhava (mineral salt)
 - vida (salt made from amalaki fruit)
 - yavakshara alkaline ash (calcinated *Hordeum vulgare* grass)
 - Rx: 2 pills bid-tid
 - usually given with <u>Chandraprabha vati</u>

Formulations for chronic gastroenteritis - Chinese medicine

- Zhen Ren yang Zang Tang (True Man's Decoction Nourish Organs)
 - \circ 3-6 g ren shen (*Panax ginseng* root)
 - o 9-12 g chao bai zhu (*Atractylodes macrocephala*, dry-fried root)
 - o 3-4.5 g rou gui (Cinnamomum cassia twig)

- 9-15 g wei rou dou kou (*Myristica fragrans*, roasted seed)
- o 6-15 g he zi (Terminalia chebula fruit)
- 6-20 g mi zhi ying su ke (Papaver somniferum pericarp)
- 9-15 g bai shao (Paeonia lactiflora root)
- 6-12 g dang gui (Angelica sinensis root)
- 6-9 g mu xiang (Saussurea lappa root)
- o 6-9 g zhi gan gao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - warms Spleen, restores deficiency, stops diarrhea
 - Rx: 200 mL bid-tid
- <u>Si Shen Wan (Four Miracle Pill)</u>
 - 4 parts bu gu zhi (*Psoralea corylifolia* seed)
 - 1 part chao wu zhu yu (*Evodia rutaecarpa* dry-fried seed)
 - 2 parts rou dou kou (*Myristica fragrans* seed)
 - 2 parts wu wei zi (Schisandra chinensis fruit)
 - warms Spleen, binds up intestines, stops diarrhea
 - Rx: 200 mL bid-tid

Formulations – Unani

- <u>Habb-E-Raal</u>
 - o pill (500 mg), Rx: 1-2 pills bid, after meals

Formulations – Western herbal

- Dr. Christopher's Diarrhea Formula
 - 6 parts bistort (polygonum bistorta root)
 - 6 parts raspberry (Rubus idaeus leaf)
 - 1 part <u>Composition Powder</u>
 - decoction, 30-90 mL very few hours

Small intestine bacterial overgrowth (SIBO)

Small intestine bacterial overgrowth (SIBO) is a digestive disorder caused by the infiltration and accumulation of colonic bacteria within the small intestine. Normally the small intestine is relatively sterile, as the digestive secretions of the stomach and small intestine exert a powerful antimicrobial effect to limit bacterial growth. As the chyme passes from the small intestine to the colon through the ileocecal sphincter it is inoculated by bacteria within the cecum, after which colonic bacteria ferment the chyme to form the feces. In SIBO, however, there is a retrograde flow of bacteria into the small intestine through the ileocecal sphincter, where they take up residence and interfere with the process of digestion and absorption. Excessive bacterial concentrations in the small intestine cause direct inflammation to the epithelial cells leading to diarrhea, whereas improperly digested nutrients such lipids, proteins, and carbohydrates that pass into the colon induce diarrhea by osmosis.

Patients with SIBO typically develop signs and symptoms associated with **irritable bowel syndrome (IBS)**, including nausea, bloating, vomiting, diarrhea and/or constipation, malnutrition, weight loss, and malabsorption. Considered to some extent a "waste-basket"

diagnosis, recent research has demonstrated that up to 78% of IBS patients likely suffer from SIBO.⁴² Some patients may lose weight and children with SIBO may fail to thrive. Impaired fat digestion evidenced by steatorrhea may also occur, leading to deficiencies of fat-soluble nutrients and vitamins. Likewise, SIBO may impair the absorption of iron and vitamin B12, and lead to chronic anemia.

There are a number of factors that cause or promote SIBO, including immunosuppression from the use of immunosuppressant medications, as well as both acquired and inherited immunodeficiency conditions. Patients with chronic pancreatitis are also at risk due to an impairment in the secretion of digestive enzymes (Trespi and Ferrieri 1999). Patients that have undergone surgery for Crohn's disease in which the ileum is damaged or removed are at increased risk of SIBO (Kholoussy al 1986). Medications that impair gastric acid secretion including PPIs are associated with an increased risk of developing SIBO (Lo and Chan 2013). More broadly, SIBO appears to be related the development of intestinal permeability, and is linked to autoimmune conditions such as fibromyalgia and roseacea (Lykova et al 2005, Goebel et al 2008, Parodi et al 2008).

The gold standard for the diagnosis of SIBO relies upon an aspirate obtained from the jejunum by endoscopy that is cultured for bacteria. The most common bacteria isolated from the small intestine of patients with SIBO are *Escherichia coli, Streptococcus, Lactobacillus, Bacteroides,* and *Enterococcus* species. A positive diagnosis of SIBO is obtained if the bacterial load is greater than 105 bacteria per millilitre, but a count as low as 103 may still suggest SIBO if the flora is predominately colonic type bacteria. The results of culturing, however, are not always representative, and false positives may occur due to contamination from the oral flora. As such, with reproducibility rates as low as 38% the reliability of this diagnostic technique has been questioned (Quigley and Quera 2006).

A non-invasive alternative to culturing jejunal aspirations are breath tests that detect the bacterial metabolism of carbohydrates to hydrogen and/or methane. This test requires that the patient fasts for a minimum of 12 hours before drinking a solution containing glucose or lactulose, and then measuring expired hydrogen and methane concentrations over a 2–3 hour period. The hydrogen breath test has been criticized, however, as it depends on the presence of hydrogen producing bacteria, and does not measure the proportion of non-hydrogen producing bacteria (Simrén M, Stotzer PO. 2006). Other similar breath tests measure the bacterial metabolism of D-xylose and glycocholic acid.

Medical treatment

SIBO is usually treated with a rotating course of antibiotics including tetracycline, amoxicillin, fluoroquinolones, metronidazole, neomycin, and rifaximin. More recently, some medical practitioners have recommended probiotics as a first line of therapy, including supplementation with *Lactobacillus casei*, *L. plantarum*, *L. plantarum*, *L. rhamnosus*, and *L. acidophilus*, although some probiotic species such as *L. fermentum* and *Saccharomyces boulardii* have been found to be ineffective (Quigley and Quera 2006). Another alternative to antibiotic

⁴² Ghoshal UC, Shukla S, Ghoshal U. 2017. Small Intestinal Bacterial Overgrowth and Irritable Bowel Syndrome: A Bridge between Functional Organic Dichotomy. *Gut Liver.* 11(2): 196–208.

therapy is a restricted diet low in fiber to limit the fuel for bacterial fermentation, called the FODMAP diet.

Holistic treatment

SIBO relates to a fundamental weakness of digestion, typically caused by dietary and lifestyle factors such as improper food combinations, eating raw or improperly prepared food, eating too much food, eating at irregular times, and chronic mental/emotional stress. To resolve SIBO dietary modification is often suggested, and while this can be helpful, without proper treatment the diet may become limited as the patient becomes increasingly intolerant to a wide variety of foods, risking not just convenience but also nutrition and health. A good example of this is the supposed connection between SIBO and "histamine intolerance," the latter of which has long and diverse list of histamine-containing foods that must be avoided. Another frequent recommendation are probiotics and lacto-fermented foods to restore the gut microbiome, but such interventions should not be given before digestion has begun to improve, introduced to the diet in only small amounts. While dietary factors and probiotics are obviously important to restore gut health, applying just these factors alone will typically fail to resolve the condition.

A key feature of SIBO is a failure of the ileocecal sphincter to properly close and prevent the retrograde flow of bacteria into the ileum from the cecum. The ileocecal sphincter can be compromised by a number of factors, including low-grade to moderate inflammation of the cecum and appendix caused by an impairment of the colonic microbiome. A diet too low in fiber increases the risk of diverticular disease, such as cecal diverticulitis that can impair the function of the ileocecal sphincter. Likewise, a diet too high in fiber – usually commensurate with poor digestion – causes the ileocecal sphincter to remain open too often, facilitating the retrograde flow of bacteria. Apart from stretch receptors in the ileum that stimulate its opening, the tonicity of the ileocecal sphincter is regulated by the secretion of digestive hormones including gastrin (secreted in the stomach) and CCK (secreted in the duodenum). Thus, an impairment of the stomach and duodenum, as well as the biliary system that plays a role in the feedback regulation of CCK, all serve to promote a dysegulation of the ileocecal sphincter.

In Ayurveda SIBO is related to the same factors as indigestion (*ajirna*) and the accumulation of *ama*, but specifically resembles a disorder called *grahani*, a condition that often arises due to the improper treatment of gastroenteritis. The disorder relates to a relative increase in stomach function, often causing an increase in appetite, but typically a feeling of discomfort a few hours after eating due to an impairment in the alkaline secretions of the duodenum (*grahani*). There are four basic causes of *grahani*: three related to a vitiation of each one of *doshas*, and a fourth relating to all three *doshas* in combination (*sannipata*). Mental and emotional factors such as *chinta* (worry), *bhaya* (fear), and *krodha* (anger) also play an important role in *grahani*, each of which relates to a pariclar *dosha*, i.e. worry (*kapha*), fear (*vata*), and anger (*pitta*).

In Chinese medicine, SIBO typically relates to an underlying impairment of the Spleen *qi* that is typically commensurate with Liver *qi* stagnation. In a state of health, the Liver regulates the function of the Spleen, but if the Liver *qi* stagnates and the Spleen *qi* is weak, the Liver

overwhelms the Spleen. This pattern of "Liver attacking Spleen" typically results in an alternating pattern of Liver *qi* stagnation (e.g. constipation, abdominal distention, borborygmi, and colic), with a pattern of Spleen *qi* deficiency (e.g. weak digestion, fatigue, loose motions). Stomach function may also be impaired, including Stomach *yang* deficiency and Stomach *qi* deficiency (Food Stagnation). Liver *qi* stagnation can also cause Stomach Fire and promote symptoms such as epigastric discomfort and gastric reflux. A key formula used to harmonize the function of the Liver and Spleen, indicated in a diarrhea-dominant pattern, is Tong Xie Yao Fang (Formula for Painful Diarrhea):

- 9-12 g chao bai zhu (*Atractylodes macrocephala* dry-fried root)
- 6-24 g chao bai shao (*Paeonia alba* dry-fried root)
- 4.5-9 g chao chen pi (*Citrus reticulata* pericarp)
- 3-6 g fang feng (*Ledebouriella divaricata* root)

This formula is based on an older version called <u>Si Ni San (Frigid Extremities Powder)</u>, which is better indicated in a constipation-dominant pattern:

- 9-12 g chai hu (*Bupleurum falcatum* root)
- 6-24 g chao zhi shi (*Citrus aurantium* dry-fried immature fruit)
- 4.5-9 g bai shao (Paeonia alba root)
- 3-6 g gan cao (*Glycyrrhiza uralensis* root)

Both formulas contain bai shao (*Paeonia alba* root), a key herb to harmonize the Liver and Spleen, but contain different herbs with similar and opposing functions. <u>Tong Xie Yao Fang</u> contains fang feng instead of chai hu to spread the Liver *qi*, but compared to the latter has more of a warming, upward-moving activity to oppose diarrhea. Likewise, both formulas contain bai zhu and gan cao to nourish the Spleen *qi*, but again bai zhu has more of an upward-moving quality. The primary difference between these formulas comes from the action of chao zhi shi in <u>Si Ni San</u>, the roasted immature fruit of the bitter orange. Chao zhi shi directs the *qi* downwards and unblocks the bowels, whereas chen pi is more directed to the underlying issue of Food Stagnation.

Both <u>Tong Xie Yao Fang</u> and <u>Si Ni San</u> can be modified based on additional patterns. For Damp-Heat symptoms, herbs such as huang lian (*Coptis chinensis* root/rhizome), huang qin (*Scutellaria baicalensis* root), and huang bai (*Phellodendron amurense* bark) can be added, whereas for Cold patterns warming herbs are indicated, such as including rou gui (*Cinnamomum cassia* bark), gan jiang (*Zingiber officinalis* rhizome), and zhi fu zi (*Aconitum carmichaeli*, processed lateral roots). To help resolve the diarrhea specifically, astringing herbs that enhance digestion are used, such as fu ling (*Poria cocos* fruiting body), he zi (*Terminalia chebula* fruit), and shi liu pi (*Punica granatum* pericarp).

The following is a review of the basic elements required in holistic medicine to resolve SIBO:

Enkindle the digestive fire

• ensure regular meal times; do not eat if not hungry; avoid over-eating

- periodic fasting on warm water, or eating simple foods
 - e.g. kitchari, soup, stew (see graduated diet, p. 182)
- temporarily limit/avoid high fiber (FODMAP) foods
- avoid raw, cold, dry, or excessively greasy or sweet foods
- digestive stimulants, e.g. shunthi (*Zingiber officinalis* rhizome), pippali (*Piper longum* fruit), maricha (*Piper nigrum* fruit), ela (*Elettaria cardamomum* fruit), hingu (*Ferula narthex* gum), tumburu (*Zanthoxylum alatum* pericarp/bark), cayenne (*Capsicum annuum* pod)
- carminatives, e.g. yavani (*Trachyspermum ammi* fruit), ajwain (*Trachyspermum ammi* seed), aniseed (*Pimpinella anisum* seed), fennel (*Foeniculum vulgare* seed), caraway (*Carum carvi* seed), chamomile (*Matricaria chamomilla* flower), calamus (*Acorus calamus* rhizome)
- trophorestoratives, e.g. Ashwagandha (*Withania somnifera* root), ren shen (*Panax ginseng* root), dang shen (*Codonopsis pilosula* root), huang qi (*Astragalus membranaceus* root), bone broth (fat free), vegetable peel/seaweed soup

Decrease intestinal irritation, inflammation, and spasm

- alkaline remedies, to be used with carminatives and digestive stimulants
 - e.g. shankha bhasma (calcinated *Turbinella pyrum* shell ash), shukti bhasma (calcinated *Ostrea gigas* shell ash), pravala bhasma (calcinated coral ash)
- antispasmodics, e.g. bilva (*Aegle marmelos* unripe fruit), hingu (*Ferula narthex* gum), musta (*Cyperus rotundus* rhizome/tuber), bai shao (*Paeonia lactiflora* root), chen pi (*Citrus reticulata* pericarp), shan za (*Crataegus pinnatifida* fruit), wild yam (*Dioscorea villosa* root), crampbark (*Viburnum opulus* bark), hops (*Humulus lupus* strobiles), cannabis (*Cannabis indica* flower), belladonna (*Atropa belladonna* root)
- vulneraries, e.g. calendula (*Calendula officinalis* flower), plantain (*Plantago spp.* leaf), selfheal (*Prunella vulgaris* leaf), St. John's wort (*Hypericum perforatum* flower), licorice (*Glycyrhiza glabra* root), chickweed (*Stelaria media* herb)
- demulcents, use judiciously as mucopolysaccharides may be too high in fiber, e.g. shatavari (*Asparagus racemosa* root), mai men dong (*Ophiopogon japonicus* root), tian men dong (*Asparagus cochinchinensis* root), slippery elm (*Ulmus fulva inner bark*), marshmallow (*Althaea officinalis* root)
- avoid Factors that promote intestinal permeability, p. 166

Inhibit diarrhea and prevent dehydration

- astringents, e.g. bilva (*Aegle marmelos* unripe fruit), dadima (*Punica granatum* rind), raspberry/blackberry (*Rubus spp.* root), cinnamon (*Cinnamomum cassia* bark), goldenseal (*Hydrastis canadensis* root/rhizome), haritaki (*Terminalia chebula* fruit decoction), bayberry (*Myrica cerifera* bark), agrimony (*Agrimonia eupatoria* herb), cranesbill geranium (*Geranium maculatum* root), oak (*Quercus alba* bark)
- lightly salted rice soup (i.e. kanjika or congee, see p. 182) prepared at a 1:8 ratio
- WHO ORT

Support bile synthesis and excretion

• cholagogues, e.g. chai hu (Bupleurum falcatum), dandelion (Taraxacum officinalis root), turmeric (Curcuma longa rhizome), gentian (Gentiana lutea root), yellowdock (Rumex

crispus root), barberry (*Berberis/Mahonia spp.* root), chiretta (*Swertia chiretta* herb), fringetree (*Chionanthus virginica* root bark)

Restore balance to the gut microbiome

- antimicrobials, e.g. barberry (*Mahoia/Berberis spp.* root), goldenseal (*Hydrastis canadensis* root/rhizome), coptis (*Coptis spp.* rhizome/root), kutaja (*Holarrhena antidysenterica* bark), haritaki (*Terminalia chebula* fruit decoction), garlic (*Allium sativum* bulb), hing (*Ferula narthex* gum), guggul (*Commiphora wightii* resin, myrrh (*C. myrrha* resin), sweet annie (*Artemisia annua* herb), black walnut (*Juglans nigra* green hull), nimba (*Azadirachta indica leaf*/bark), quassia (*Quassia amara* wood), pau d'arco (*Tabebuia spp.* bark)
- probiotics, introduced after appetite has improved and symptoms diminished
 o live culture foods, e.g. yogurt, buttermilk (*takra*), lacto-fermented pickle brine
- supplements: Lactobacillus, Bifidobacterium, Saccharomyces boulardii
- avoid yeasted foods, e.g. fruit, bread, wine, beer, kombucha/tibicos, etc.
- avoid all refined sweeteners and sweet/dried fruit

Formulations – Ayurveda

- <u>Musta-bilwadi yoga</u>
 - 4 parts musta (*Cyperus rotundus* rhizome/tuber)
 - 4 parts bilwa (Aegle marmelos unripe fruit)
 - 4 parts dhaniya (*Coriandrum sativum* seed)
 - 4 parts hriverum (Pavonia odorata herb)
 - 4 parts shunthi (*Zingiber officinalis* rhizome)
 - 4 parts twak (*Cinnamomum verum* bark)
 - 4 parts shatapushpa (*Pimpinella anisum* seed)
 - 2 parts saindhava (pink salt)
 - 1 part shankha bhasma (calcinated *Turbinella pyrum* shell ash)
 - a *grahi* remedy for diarrhea
 - 3-5 g bid-tid
- <u>Chandraprabha vati</u>
 - 2 pills bid-tid
- <u>Shambhukadi vati</u>
 - \circ 2 pills bid-tid

Formulations - Chinese medicine

- Bu Zhong Yi Qi Wan (Tonify Middle Augment Essence Decoction)
 - for Spleen *qi* deficiency
 - decoction, Rx: 200 mL bid-tid
 - tea pills, Rx: 8 pills bid-tid
 - o granules, Rx: 2-4 g bid-tid
- Xiao Yao San (Rambling Powder)
 - spreads Liver *qi*, strengthens Spleen
 - o powder, Rx: 6-9 g bid-tid
 - o granules, Rx: 2-4 g bid-tid
- Ban Xia Xie Xin Tang (Pinellia Decoction to Drain the Epigastrum)
 - o harmonizes Stomach, directs Rebellious *qi* downwards, disperses distension

- o decoction, Rx: 200 mL bid-tid
- o granules, Rx: 3-4 g bid-tid
- <u>Tong Xie Yao Fang (Formula for Painful Diarrhea)</u>
 - o for diarrhea-dominant conditions
 - o decoction, Rx: 200 mL bid-tid
 - o restores Spleen, harmonizes Spleen and Liver, expels dampness
 - o granules, Rx: 3-5 g bid-tid
- <u>Si Ni San (Frigid Extremities Powder)</u>
 - for constipation-dominant conditions
 - o restores Spleen, harmonizes Spleen and Liver, expels dampness
 - o decoction, Rx: 200 mL bid-tid
 - powder, Rx: 6-9 g bid-tid
 - o granules, Rx: 2-4 g bid-tid

Formulations - Unani

- <u>Habb-E-Raal</u>
 - o pill (500 mg), Rx: 1-2 pills bid, after meals
- Jawarish-E-Jalinoos
 - o 25 g mastagi (Pistacia lentiscus resin)
 - o 10 g sumbul-ut-teeb (*Nardostachys jatamansi* resin)
 - 10 g heel khurd (*Elettaria cardamomum* resin)
 - 10 g saleekha (*Cinnamomum cassia* resin)
 - o 10 g darchini (*Cinnamomum zeylanicum* resin)
 - o 10 g khulanjan (Alpinia galanga resin)
 - 10 g qaranfal (*Syzygium aromaticum* resin)
 - 10 g sad kufi (*Cyperus rotundus* resin)
 - 10 g zanjabeel (Zingiber officinale resin)
 - 10 g filfil daraz (*Piper longum* resin)
 - 10 g filfil siyah (*Piper nigrum* resin)
 - 10 g qust shireen (Saussurea lappa resin)
 - o 10 g ood-e-balsan (*Commiphora gileadensis* resin)
 - 10 g asaroon (Asarum europaeum resin)
 - 10 g habb-ul-aa (*Myrtus communis* resin)
 - 10 g chiraita shireen (Swertia chirata resin)
 - 10 g zafran (*Crocus sativus* stigma/style)
 - \circ 600 g qand safaid (sugar)
 - used in *zof-e-meda* (weakness of the stomach), *nafkh-e-shikam* (flatulence in the stomach), and as a *muqawwi-e-kabid* (liver restorative)
 - confection, Rx: 5-15 g bid

Constipation

Constipation refers to the difficult or infrequent passage of feces as well as a subjective feeling of incomplete evacuation. There is no general agreement in modern medicine as to the proper frequency for bowel movements, which in different patients can fluctuate anywhere

between two to three bowel movements per day, to one bowel movement two to three times per week. This wide variation in bowel habits that is accepted as "normal" in modern medicine is contrary to the perspective held in traditional medicine, which suggests that every person should evacuate the bowels at least one to two times on a daily basis. Nonetheless, an occasional alteration in normal bowel habits is not necessarily pathogenic, and care should be taken not to worry the patient if this happens. In Ayurveda, the colon is the seat (sthana) of *vata dosha*, and hence there is an inherent relationship between the state of the nervous system and the function of the bowel. As such, any disruption to the normal rhythm of daily living, such as a change in dietary or lifestyle routines, as well as travel, a lack of exercise, or emotional and mental stress can alter bowel habits. Recalling the traditional Chinese perspective, the function of the Large Intestine relates to the notion of "letting go." Thus, excessively worrying about constipation can be a part of of vicious cycle relationship that can end up making things worse. Further, an excessive reliance upon laxatives and enemas can disturb the delicate bowel ecology and contribute to a loss of the colonic haustra. Worry might also be incurred when patients who habitually use anthraquinone laxatives such as turkey rhubarb (Rheum palmatum root) or cascara sagrada (Rhamnus purshianus wood) exhibit melanosis coli, which are deposits of brown pigment in the mucosa that are seen upon endoscopy or colonic biopsy. While this temporary pigmentation has never been proven to be harmful in any way, a gastroenterologist may indicate to their patient that it is due to a preexisting bias against herbs, causing further worry and upset.

A proper study of constipation begins by comprehending the dynamics of feces formation and what constitutes its make-up. In a healthy individual, the bulk of the feces is formed by the bacterial fermentation of indigestible fibers. As the chyme is extracted of its available nutrients, the ileum of the small intestine passes a relatively small volme of whatever is left over to the cecum of the colon, through the ileocecal sphincter. These indigestible materials are then inoculated by resident bacteria in the cecum, which also houses the appendix, a tube-like pocket of the cecum that is dense with lymphatic tissues, and serves as a reservoir of colonic bacteria. As the bacteria ferment this waste, agitated and enhanced by haustral chrning, they literally "grow" the feces, such that the total volume of the feces is upwards of 80% by bacteria. Thus the bowel ecology is of prime importance when discussing the issue of constipation, the role of diet, and the use of medications such as antibiotics that adversely affect the microbiome.

The conventional biomedical approach recognizes a few distinct causes of constipation, the most common of which is usually ascribed to a diet low in fiber diet, dehydration, and a lack of exercise. The elderly are particularly susceptible to constipation due to an overall weakening and diminishment of digestive secretions, with a loss of colonic reflexes. Other major causes of constipation include pregnancy, inflammatory conditions such as peritonitis and diverticulitis, bowel infection, neurological trauma (e.g. head injuries, spinal fractures, spinal cord injury), hypothyroidism, Parkinson's disease, cerebral thrombosis, cancer, and ascities.

The most serious manifestation of chronic constipation is **fecal impaction**. This occurs when a large amount of feces accumulates in a region of the colon, most often the rectum, causing either complete or partial obstruction, and in the latter causing fecal incontinence (e.g. "overflow" diarrhea, or encopresis). The patient often complains of rectal pain and spasm, and

when after a period of time finally evacuates the bowels, the consistency of the feces is hard, dry and marble-like, often evacuated with watery mucus. Sometimes the bowel movement may vary in color and shape, the first portion appearing as a dark hard mass that causes much pain to evacuate, but then followed by a watery diarrhea. It is more common in the elderly and in the bedridden, as well as in spinal cord injury, Parkinson's, diabetes, chronic kidney failure, and cancer. Other diseases associated with fecal impaction include SIBO, autoimmune disease (e.g. inflammatory bowel disease, lupus), and hypothyroidism.

Given the relative unimportance of constipation from a conventional biomedical perspective, it perhaps isn't surprising that fecal impaction is a common side effect of different procedures and medications, such as barium enema, antibiotics, and opioids. Other medications that promote fecal impaction include aluminum hydroxide, bismuth salts, iron salts, cholestyramine, anticholinergics, ganglionic blockers, tranquilizers, sedatives, and general anesthesia. Fecal impaction can also be caused by the use of hydrophilic bowel laxatives, such as psyllium, in which the patient doesn't drink enough water, rendering the fiber into a sticky mass that is difficult to evacuate.

Medical Treatment

While laxatives such as milk of magnesia and polyethylene glycol were at one time popular OTC remedies for constipation, with a recognition of the importance of dietary fiber, the current medical treatment of uncomplicated constipation relies upon the usage of vegetable fibers, usually in the form of hydrophilic bulk laxatives such as psyllium, cereal brans (e.g. wheat, oat), or methylcellulose. Occasionally other laxatives will be recommended such as docusate, bisacodyl, mineral oil, and castor oil. In chronic constipation and fecal impaction warm water or oil enemas may also be indicated.

Some laxatives such as milk of magnesia (magnesium hydroxide) are comprised of poorly absorbed polyvalent ions that draw water into the intestine by osmosis, increasing the intraluminal contents and pressure. Other laxatives such as mineral oil and castor oil may help to soften the fecal matter but because they pass through the digestive tract unabsorbed, may interfere with the absorption of fat-soluble vitamins. Detergent laxatives such as docusate are used both internally and as a suppository to soften stools, breaking down surface barriers and allowing water to enter the fecal mass to soften and increase its bulk. To one extent or another, all laxatives irritate the intestinal mucosa and inhibit the absorption of water, often promoting dependence, and when used long term can cause serious fluid and electrolyte disturbances.

Holistic Treatment

Constipation is an exceptionally common problem in the West, and for the most part, is a symptom of an improper diet and a lack of exercise. The most common dietary relates to the consumption foods that are low in fiber, and in particular, have a sticky, adherent, greasy, and heavy property, such as flour products, cheese, sticky rice, potato, banana, deep-fried foods, and fatty meat. Apart from serving as a kind of glue (e.g. papier mâché) that poses serious mechanical issues for the gut, the consumption of cereals as a homogenous, acellular product

has shown been to promote alterations in the gastrointestinal microbiome, increasing GI inflammation and contributing to metabolic disorders such as obesity.⁴³

Apart from the health of the gut microbiome, another frequent cause of constipation is impaired biliary secretion. In Chinese medicine, Liver *qi* stagnation is a major cause of constipation, which in Ayurveda relates to an increase in *pitta-vata*. Here the liver is understimulated or blocked in its function, and as the bile becomes dry and stagnant (*vata*) it leads to an increase heat and inflammation (*pitta*). Bile is a hot, greasy, oleaginous substance mostly comprised of cholesterol that serves to lubricate the gut and exert a stimulant effect upon peristalsis. Without the regular and proper secretion of bile digestion becomes erratic and weak, and eventually leads to problems such as constipation. Apart from the consumption of fatty foods, much of the stimulus for biliary excretion relates to the consumption of bittertasting foods, including both culinary and medicinal herbs.

Whether or not to make use of herbal laxatives in a given case of constipation is an important consideration. While such medications can be used with short-term symptomatic success, chronic use will tend to create dependence. The same problems seen for some of the conventional medical laxatives are also seen with herbs that have a laxative activity, as they essentially function in the same manner by interfering with the reabsorption of water. Simply prescribing a laxative in constipation is to be avoided, especially without taking into consideration the underlying causes. Both Ayurveda and Chinese medicine account for the fact that constipation is often caused by too much dryness, and because most herbal laxatives have a drying property, should only be given so as not to make the underlying issue worse. Especially in elderly patients that often suffer from dryness (*vata*), natural products such as bulk laxatives and especially herbs that lubricate the colon are indicated.

The following is an overview of the holistic measure used in chronic constipation:

Promote proper digestion

- pungent-tasting stimulants to enhance enteric blood flow and remove mucoid accumulations, e.g. shunthi (*Zingiber officinalis* rhizome), pippali (*Piper longum* fruit), maricha (*Piper nigrum* fruit), ela (*Elettaria cardamomum* fruit), hingu (*Ferula narthex* gum), tumburu (*Zanthoxylum alatum* pericarp/bark), cayenne (*Capsicum annuum* pod), <u>Trikatu churna, Hingvastak churna</u>
- aromatic carminatives, to enkindle proper digestion and relieve flatulence and spasm, e.g. chen pi (*Citrus reticulata* pericarp), yavani (*Trachyspermum ammi* fruit), ajwain (*Trachyspermum ammi* seed), aniseed (*Pimpinella anisum* seed), fennel (*Foeniculum vulgare* seed), caraway (*Carum carvi* seed), chamomile (*Matricaria chamomilla* flower), calamus (*Acorus calamus* rhizome), mint (*Mentha spp.* herb)
- rejuvenatives, for use in cases of digestive weakness and fatigue (Spleen *qi-ojas* deficiency), e.g. ren shen (*Panax ginseng* root), huang qi (*Astragalus membranaceus* root), dang shen (*Codonopsis pilosula* root)

⁴³ Spreadbury I. 2012. Comparison with ancestral diets suggests dense acellular carbohydrates promote an inflammatory microbiota, and may be the primary dietary cause of leptin resistance and obesity. *Diabetes Metab Syndr Obes*. 5: 175–189.

• in elderly patients with hypochlorhydria or liver/gall-bladder disease, use full spectrum digestive enzymes (with oxbile and lipase)

Support liver function and biliary excretion

- cholagogues, taken before meals e.g. dandelion (*Taraxacum officinalis* root), turmeric (*Curcuma longa* rhizome), gentian (*Gentiana lutea* root), yellowdock (*Rumex crispus* root), barberry (*Berberis/Mahonia spp.* root), chiretta (*Swertia chiretta* herb), fringetree (*Chionanthus virginica* root bark)
- hepatic trophorestoratives, to support hepatic function, e.g. bai shao (*Paeonia lactiflora* root), milk thistle (*Silybum marianum* seed), wu wei zi (*Schizandra chinensis* fruit), guduchi (*Tinospora cordifolia* stem), amalaki (*Phyllanthus emblica* fruit), chai hu (*Bupleurum falcatum* root)
- avoid tobacco, alcohol, acetaminophen, solvents, etc. that impair liver function

Restore the bowel ecology

- live culture foods, e.g. fermented vegetables, kefir
- probiotics, e.g. *Lactobacillus, Bifidobacterium*, live culture foods
- prebiotics, e.g. FODMAP-containing foods, chicory root, beet root, fructooligosaccharides (e.g. inulin)
- avoid foods that has a heavy, sticky, and greasy property, e.g. flour, dairy,
- avoid simple sugars, sweet fruits, dried fruits, and yeasted foods (e.g. wine, beer, kombucha)
 - o most yeasts (e.g. Candida albicans) are antagonistic to the "friendly bacteria

Restore bowel tone and peristalsic function

- herbal laxatives, e.g. zhi shi (*Citrus aurantium* unripe seed), da huang (*Rheum palmatum* root), cascara sagrada (*Rhamnus purshianus* wood), trivrit (*Operculina turpethum* root), senna (*Cassia angustifolia* pods)
 - \circ $\;$ always use with carminatives to avoid griping $\;$
- bulk laxatives, e.g. psyllium husk, flax seed, chia seed
- magnesium oxide

Lubricate the colon and restore the intestinal mucosa

- consume 500-750 mL water upon rising; drink around one liter of water during the remainder of the day
- generally, increase oil consumption in the diet, e.g. ghee, olive, sesame, coconut
 o ensure proper fat digestion
- matra vasti karma (oil rentention enema), 30-90 mL injected before bed
- demulcents, e.g slippery elm (*Ulmus fulva* inner bark), huo ma ren (*Cannabis spp.* seed), marshmallow (*Althaea officinalis* root), yu li ren (*Prunus japonica* seed), tila (*Sesamum indicum* seed), hu tao ren (*Juglans regia* nut), bai zi ren (*Biota orientalis* seed)
- colon restoratives, e.g. bala (Sida cordifolia root), we shou wu (Polygonum multiflorum root), ashwagandha (Withania somnifera root), sang shen (Morus alba fruit), shatavari

(Asparagus racemosa root), shu di huang (Rehmannia glutinosa, root stir-fried in wine), mai men dong (Ophiopogon japonicus root), licorice (Glycyrrhiza glabra root)

Formulations – Ayurveda

- <u>Triphala churna</u>
 - o powder, Rx: 2-5 g bid
 - mix with an equal part trivrit (*Operculina turpethum* root) for recalcitrant cases
- Gandharvahastadi kashaya
 - o one part gandharvahasta (eranda) (*Ricinus communis* root)
 - o one part chirabilva (Holoptelea integrifolia bark)
 - o one part hutasha (chitraka) (*Plumbago zeylanica* root)
 - o one part vishwa (shunthi) (*Zingiber officinalis* rhizome)
 - o one part pathya (Terminalia chebula fruit)
 - o one part punarnava (Boerhaavia diffusa root)
 - o one part yavasa (Alhagi pseudalhagi herb)
 - one part bhumitala (musali) (*Chlorophytum borivilianum* root)
 - decoction, Rx: 100 mL bid
 - kashayam tablets, Rx: 2-4 tab bid
- <u>Chandraprabha vati</u>
 - Rx: 2 pills 2-3 times daily

Formulations - Chinese medicine

- Run Chang Wan (Moisten Intestines Pill)
 - o for Large Intestine Dryness; moistens/unblocks bowels
 - Rx: 15 g mixed with honey, in divided doses
- Ji Chuan Jian (Benefit River Decoction)
 - o for Large Intestine Cold; warms the Kidneys, moistens/unblocks bowels
 - o decoction, Rx: 200 mL bid-tid
 - o granules, Rx: 3-4 g bid-tid
- <u>Xiao Yao San (Rambling Powder)</u>
 - spreads Liver *qi*, strengthens Spleen
 - powder, Rx: 6-9 g bid-tid
 - o granules, Rx: 2-4 g bid-tid

Formulations - Unani

- 10 g burge sana (*Cassia angustifolia* pods) with 5 g kamun halu (*Pimpinella anisum* seed) boiled in 250 mL water, taken with jaggery
- gulqand (*Rosa spp.* petals prepared with jaggery), 40 g taken with boiled milk
- 6 g halela zard (*Terminalia chebula* pericarp powder), taken with pink salt and warm water
- <u>Safoof-E-Sana</u>
 - 1 part burge sana (Cassia angustifolia leaf)
 - 1 part zanjabeel (*Zingiber officinalis* rhizome)
 - 1 part halela kabuli (*Terminalia chebula* fruit)
 - 1 part namak-e-siyah (black salt)

• powder, Rx: 2-3 g bid-tid

Formulations - Western herbal

- Dr. Christophers <u>Lower Bowel Formula</u>
 - o 1 part barberry (Berberis vulgaris root bark)
 - o 2 parts cascara sagrada (Rhamnus purshianus wood)
 - 1 part cayenne (*Capsicum annuum* pods)
 - 1 part ginger (Zingiber officinalis rhizome)
 - 1 part lobelia (Lobelia inflata leaf)
 - o 1 part raspberry (Rbus idaeus leaf)
 - 1 part turkey rhubarb (*Rheum palmatum* root)
 - 1 part fennel (Foeniculum vulgare seed)
 - o 1 part goldenseal (*Hydrastis canadensis* root/rhizome)
 - powder, Rx: 3-5 g bid-tid

Hemorrhoids

Hemorrhoids are traditionally viewed as a varicosity of the hemorrhoidal plexus, a specialized arteriovenous shunt that is filled with oxygenated blood, and the reason why the blood of bleeding hemorrhoids is bright red. **External hemorrhoids** are located below the anal sphincter, which is lined with squamous epithelium, whereas **internal hemorrhoids** are located above the sphincter and are covered in rectal mucosa. External hemorrhoids drain through the inferior rectal vein into the inferior vena cava, whereas internal hemorrhoids drain through the superior rectal vein into the portal system.

Although referred to as a varicosity, hemorrhoids are clusters of vascular tissue, smooth muscle and connective tissue that are a normal part of human physiology. The problem arises when these tissues become swollen and inflammed. Most hemorrhoidal issues relate to enlarged internal hemorrhoids, and hence problems with the drainage of the portal vein. The dilatation and engorgement of the arteriovenous plexuses causes the prolapse of rectal tissue into the anal canal, which is easily injured by the passage of stool. This triggers an inflammatory response that includes itching, mucus discharge, and a sensation of incomplete evacuation following defection.

In many cases hemorrhoids are asymptomatic but they can protrude, bleed, and cause pain. Any kind of rectal bleeding should only be attributed to hemorrhoids after other, more serious conditions are excluded, such as diverticulosis or colorectal cancer. The volume of blood that is discharged following defecation is usually self-limiting, and rarely leads to complications such anemia or hemorrhage, but in chronic bleeding can be a cause of iron deficiency anemia. Over the course of the condition both external and internal hemorrhoids can protrude and then regress, and in most cases can be reduced temporarily by manually pushing them back inside the rectum. In some cases however they cannot be reduced manually and can become strangulated, ulcerated or thrombotic, causing severe pain. External hemorrhoids often present some difficulty in properly cleansing the anal region, which can lead to irritation and itching.

There a number of causes of hemorrhoids, the most important of which is any factor that causes trauma, impairs venous return or causes blood to pool in the pelvic cavity. This includes sitting for long periods (especially on the toilet), constipation (particularly with straining), pregnancy, aging, poor posture, a lack of exercise, liver disease, and obesity. Hemorrhoids that arise from trauma include chronic diarrhea, inflammatory bowel disease, and anal intercourse.

Medical Treatment

The medical treatment of hemorrhoids consists primarily of stool softeners (e.g. docusate, mineral oil) or bulking agents (e.g. psyllium) that can help correct constipation and prevent straining. A thrombosed hemorrhoid is treated with warm water sitz baths, anesthetic ointments, and NSAIDs. Bleeding hemorrhoids are sometimes treated by injection sclerotherapy which causes the vein to harden and die, leaving a scar on the wall of the anal canal. Larger internal hemorrhoids that fail to respond to injection sclerotherapy may be treated by rubber band ligation, in which the internal hemorrhoid is withdrawn through the band, resulting in necrosis and a sloughing of the dead tissue. Despite the risk of pain during treatment rubber band ligation has a high recurrence rate, and thus surgical hemorrhoidectomy is a better alternative in most cases of severe hemorrhoids.

Holistic treatment

In Ayurveda hemorrhoids (*arsha*) are grouped into six categories: four relating to the *doshas* (*vata, pitta, kapha, sannipata*); one caused by blood stagnation (*raktaja*); and one that is congenital (*sahaja*) in nature. *Vataja* hemorrhoids are caused by factors that increase *vata,* including activities that deplete *ojas* such as excessive sexual activity and exercise, manifesting with a dull, aching pain with small bluish colored swellings. *Pittaja* hemorrhoids involve severe inflammation of the anus, with a sharp burning pain and reddish-colored swellings, caused by general factors that increase *pitta. Kaphaja* hemorrhoids are characterized by edema, swelling, a whitish discoloration, and itchiness, caused by general factors that increase *kapha. Sannipataja* and *sahaja* hemorrhoids are incurable, but can be maintained with symptomatic treatment. *Raktaja* hemorrhoids are treated with the same principle of hemorrhoid with the character of *pitta.*

Whatever the causal factor, the basic approach used in the treatment of hemorrhoids in Ayurveda is to support digestion with *dipana-pachana* remedies, along with measures to decongest the portal vein, and the use of mild laxative herbs (except in cases of diarrhea). Specific treatments are given according to the *dosha* indicated. If the hemorrhoids are bleeding, this should not be immediately inhibited with astringent remedies as this is thought to promote complications including anemia (*pandu*) and liver problems (*kamala*).

In Chinese medicine, according to the third chapter of the *Huang Di Nei Jing Su Wen* (Yellow *Emperor's Classical on Internal Medicine*), hemorrhoids are caused by "too much food intake", which "causes injury and flaccidity of vessels." In particular, this is related to the excess consumption of foods that are roasted, fatty/greasy, cold, raw, or spicy, as well as alcohol, and

improper food intake. Other factors that are recognized in Chinese medicine include excessive sexual activity and excessive physical labor.

There are five specific patterns that can lead to hemorrhoids include Dryness, Damp/Heat, or Wind of the Large Intestine, usually commensurate Spleen *qi* deficiency and Blood Stasis. Large Intestine Wind relates to bleeding before or after defecation, whereas Large Intestine Dryness is associated with the dry forms of chronic constipation, typically seen in the elderly. Large Intestine Damp/Heat relates to inflammation, pain, and swelling of the hemorrhoidal tissues, usually accompanied by systemic signs of Heat. A Spleen *qi* deficiency relates to a sinking of the *qi*, causing the blood to pool in the hemorrhoidal plexus along with a failure of the Spleen to contain the blood within the vessels resulting in bleeding. Blood Stasis relates to severe pain and swelling in the form of an irreducible thrombus.

The following is a review of the holistic principles underlying the treatment of hemorrhoids.

External measures to relieve pain and bleeding

- for heat symptoms (pitta): cold compress, cold water sitz baths, application of ice
- for cold symptoms (*vata/kapha*): warm compress, warm water sitz baths
- application of a paste made from haridra (*Curcuma longa* rhizome) with the latex of arka (*Calotropis gigantea*)
- application of <u>Narayana taila</u> mixed with a pinch of sphutikarika (purified alum)
- application of cold fresh dadhi (yogurt) with cotton
- astringents for bleeding and prolapse, e.g. san qi (*Panax notoginseng* root), shepherd's purse (*Capsella bursa-pastoris* herb), trillium (*Trillium spp.* rhizome), witch hazel (*Hamamelis virginiana* bark), yarrow (*Achillea millefolium* herb), figwort (*Srophularia nodosa* herb), black walnut (*Juglans nigra* leaves)
 - prepared as a poultice, sitz bath (decoction, infusion), paste, etc.
- vulneraries, to promote resolution e.g. calendula (*Calendula officinalis* flower), plantain (*Plantago spp.* leaf), selfheal (*Prunella vulgaris* leaf), St. John's wort (*Hypericum perforatum* flower), licorice (*Glycyrhiza glabra* root), balm of gilead (*Populus trichocarpa* leaf bud resin), chickweed (*Stelaria media* herb)
 - o prepared as a poultice, sitz bath (decoction, infusion), paste, etc.
- Dr. Christopher's <u>Pile Ointment:</u>
 - 1 part bistort (*polygonum bistorta* root)
 - 1 part cranesbill gernanium (*Geranium maculatum* root)
 - o 1 part hemlock (*Tsuga canadensis* inner bark)
 - 6 parts beeswax
 - 4 parts muttonsuet
 - \circ 8 parts lard
 - \circ 2 parts olive oil
 - simmer ingredients (except olive oil) for one hour; after straining add the olive oil and allow to cool
- Dr. Christopher's <u>Herbal Suppository</u>:
 - 2 parts bayberry (*Myrica cerifera* bark, finely powdered)
 - 1 part hemlock (*Tsuga heterophylla* inner bark, finely powdered)

- o 1 part goldenseal (*Hydrastis canadensis* rhizome/root, finely powdered)
- 1 part slippery elm (*Ulmus fulva* inner bark, finely powdered)
 - mix with sufficient glycerine to form a suppository

Promote proper digestion

- pungent-tasting stimulants to enhance enteric blood flow and remove mucoid accumulations, e.g. shurana (*Amorphophallus campanulatus* tuber), shunthi (*Zingiber officinalis* rhizome), krishnajiraka (*Nigella sativa* seed), pippali (*Piper longum* fruit), maricha (*Piper nigrum* fruit), ela (*Elettaria cardamomum* fruit), hingu (*Ferula narthex* gum), tumburu (*Zanthoxylum alatum* pericarp/bark), cayenne (*Capsicum annuum* pod), <u>Trikatu churna, Hingwastak churna</u>
- aromatic carminatives, to enkindle proper digestion and relieve flatulence and spasm, e.g. nutmeg (*Myristica fragrans seed*), chen pi (*Citrus reticulata pericarp*), yavani (*Trachyspermum ammi fruit*), ajwain (*Trachyspermum ammi seed*), aniseed (*Pimpinella anisum seed*), fennel (*Foeniculum vulgare seed*), caraway (*Carum carvi seed*)
- digestive astringents (*grahi*), to gently astringe diarrhea/bleeding and strengthen digestion, e.g. bilwa (*Aegle marmelos* unripe fruit), kutaja (*Holarrhena antidysenterica* bark/seed), dadima (*Punica granatum* pericarp), musta (*Cyperus rotundus* rhizome/tuber), haritaki (*Terminalia chebula* fruit decoction), bayberry (*Myrica cerifera* bark)
- rejuvenatives, for use in cases of digestive weakness and fatigue (Spleen *qi-ojas* deficiency), e.g. ren shen (*Panax ginseng* root), huang qi (*Astragalus membranaceus* root), dang shen (*Codonopsis pilosula* root), bai zhu (*Atractylodes macrocephala* root), etc.

Support liver function and biliary excretion

- cholagogues, taken before meals e.g. daruharidra (*Berberis nepalensis* root bark), dandelion (*Taraxacum officinalis* root), balmony (*Chelone glabra* herb), turmeric (*Curcuma longa* rhizome), gentian (*Gentiana lutea* root), yellowdock (*Rumex crispus* root), barberry (*Berberis/Mahonia spp.* root), chiretta (*Swertia chiretta* herb), fringetree (*Chionanthus virginica* root bark), figwort (*Srophularia nodosa* herb), chapparal (*Larrea tridentata* herb)
- hepatic trophorestoratives, to support hepatic function, e.g. bai shao (*Paeonia lactiflora* root), milk thistle (*Silybum marianum* seed), wu wei zi (*Schizandra chinensis* fruit), guduchi (*Tinospora cordifolia* stem), amalaki (*Phyllanthus emblica* fruit), chai hu (*Bupleurum falcatum* root)
- avoid tobacco, alcohol, acetaminophen, solvents, etc. that impair liver function

Restore bowel tone and peristalsic function

- herbal laxatives, e.g. butternut (Juglans cinerea root bark), zhi shi (Citrus aurantium unripe seed), poke (Phytolacca decandra root), da huang (Rheum palmatum root), cascara sagrada (Rhamnus purshianus wood), trivrit (Operculina turpethum root), senna (Cassia angustifolia pods)
 - always use with carminatives to avoid griping
- bulk laxatives, e.g. psyllium husk, flax seed, chia seed
- magnesium oxide

Tone and strengthen the hemorrhoidal vasculature

- circulatory astringents, e.g. yarrow (Achillea millefolium herb), mullein (Verbascum thapsus herb), stoneroot (Collinsonia canadensis root), horsechestnut (Aesculus hippocastanum leaf/bark), cranesbill geranium (Geranium maculatum root), avens (Geum urbanum leaf), bayberry (Myrica cerifera bark), pilewort (Ficaria verna)
- to promote healing of anal mucosa, for 2-4 weeks
 - o vitamin A: 10,000 IU daily
 - vitamin C: 2-3 g daily, or to bowel tolerance
 - o vitamin E: 400 IU daily
 - o citrus bioflavonoids: 3-5 g daily
 - zinc citrate: 30-50 mg daily

Restore the bowel ecology

- live culture foods, e.g. fermented vegetables, kefir
- probiotics, e.g. *Lactobacillus, Bifidobacterium,* live culture foods
- prebiotics, e.g. FODMAP-containing foods, chickory root, beet root, fructooligosaccharides (e.g. inulin)
- avoid foods that has a heavy, sticky, and greasy property, e.g. flour, dairy,
- avoid simple sugars, sweet fruits, dried fruits, and yeasted foods (e.g. wine, beer, kombucha)
 - most yeasts (e.g. *Candida albicans*) are antagonistic to the "friendly bacteria

Formulations - Ayurveda

- <u>Gandharvahastadi kashaya</u>
 - decoction, Rx: 100 mL bid
 - o kashayam tablets, Rx: 2-4 tab bid
- <u>Avipattikara churna</u>
 - 2-3 g twice daily
- <u>Chandraprabha vati</u>
 - 2 pills twice daily

Formulations - Chinese medicine

- <u>Yi Zi Tang (Decoction 'B')</u>
 - for Large Intestine Damp-Heat and Wind
 - o decoction, Rx: 200 mL bid-tid
 - o granules, Rx: 3-4 g bid-tid
- Run Chang Wan (Moisten Intestines Pill)
 - for Large Intestine Dryness
 - Rx: 15 g mixed with honey, in divided doses
- Bu Zhong Yi Qi Wan (Tonify Middle Augment Essence Decoction)
 - for Spleen *qi* deficiency
 - o decoction, Rx: 200 mL bid-tid
 - tea pills, Rx: 8 pills bid-tid
 - o granules, Rx: 2-4 g bid-tid
- <u>Tao He Cheng Qi Tang (Peach Pit Order Essence Decoction)</u>

- 12 15 g tao ren (*Prunus persica* seed)
- 12 g da huang (*Rheum palmatum* root)
- 6 g gui zhi (*Cinnamomum cassia* twig)
- 6 g mang xiao (sodium sulphate)
- 6 g zhi gan gao (*Glycyrrhiza uralensis* root, stir-fried in honey)
 - for Blood Stasis
 - decoction, Rx: 200 mL bid-tid
 - granules, Rx: 2-4 g bid-tid

Formulations - Unani

- <u>Habb-e-Rasaut</u>
 - o 1 part halela zard (*Terminalia chebula* yellow fruit)
 - o 1 part gugal (*Commiphora wightii* resin)
 - 1 part rasaut (Berberis aristata root bark)
 - pills (180 mg each), Rx: 2 pills bid-tid
- <u>Habb-e-Muqil</u>
 - 84 g turbud safaid (*Ipomoea turpethum* root bark)
 - o 10 g khardal (Brassica nigra seed)
 - 42 g sakbeenaj (*Ferula persica* resin)
 - o 124 g muqil (*Commiphora mukul resin*)
 - o 124 g halela siyah (*Terminalia chebula* black fruit)
 - 124 g halela zard (*Terminalia chebula* yellow fruit)
 - pills (150 mg each), Rx: 1-2 pills daily

Formulations – Western herbal

- Dr. Christophers Prolapse Formula
 - 6 parts oak (Quercus alba bark)
 - 3 parts mullein (Verbascum thapsus leaf)
 - 4 parts yellowdock (*Rumex crispus* root)
 - 3 parts walnut (Juglans nigra bark/leaves)
 - 6 parts comfrey (*Symphytum officinalis* root)
 - 0 1 part lobelia (Lobelia inflata leaf)
 - 3 parts marshmallow (Althaea officinalis root)
 - decoction, Rx: 60-100 mL bid-tid
 - can also be used as a retention enema
- Dr. Christophers Lower Bowel Formula
 - o powder, Rx: 3-5 g bid-tid