

# Autoimmune disease

An autoimmune disease is a disorder in which the immune system produces auto-antibodies to an endogenous antigen, with the subsequent injury to the tissues of the body that display this antigen. Although the connective tissues like skin, muscles and joints are susceptible to wear and tear, some people have a tendency for these tissues to undergo degenerative changes that are initiated by inflammatory and immunological mechanisms. Inflammation of the skin and joints, such as systemic lupus erythematosus and rheumatoid arthritis, respectively, are among the most familiar forms of proinflammatory autoimmune disorders, but other types of conditions have been found to have an autoimmune component, including Hashimoto's thyroiditis, myasthenia gravis, diabetes mellitus, and vitiligo. The modern medical approach of autoimmune disorders rests primarily upon the use of nonsteroidal antiinflammatory drugs (NSAIDs) and corticosteroids. While effective initially, it appears that many of these conditions respond poorly to these drugs over time, and with increased dosages there is an increased risk of iatrogenic effects.

## Intestinal Permeability: Leaky Gut syndrome

There is an increasing amount of evidence that damage to the gut wall plays a role in autoimmune disease. A significantly high number of patients with ankylosing spondylitis, a rheumatoid-like condition of the axial skeleton and large joints, have been shown to have histological indications of chronic gastrointestinal inflammation and damage. It appears that in many of these patients the remission of the condition occurs in tandem with the remission of digestive inflammation, and vice versa (Mills and Bone 1999, 144-4).

In Ayurvedic medicine, most treatable disease arises from the presence of *ama*, a toxic by-product of poor digestion. One of the features of *ama* is that once it enters the *dhatu* cycle it impairs the nutrition and function of the *dhatu*s. *Ama* has a particular affinity for tissues that are weak and accumulates in these locations. Once it is firmly wedged in these locations, the *doshas* become vitiated, first *Kapha*, with an increase in congestion, followed by

### Conditions linked with Intestinal Permeability

Food allergies and sensitivities  
Atopic conditions  
Arthritis  
Rheumatoid Arthritis  
Asthma  
Chronic fatigue  
Chronic urticaria  
Crohn's disease  
Diabetes mellitus  
Ulcerative colitis  
Irritable bowel  
Thyroiditis  
Raynaud's syndrome  
Acute gastroenteritis  
Cystic fibrosis  
Multiple sclerosis  
Spondyloarthropathies  
Acne  
Eczema  
Dermatitis herpetiformis  
Psoriasis  
AIDS, HIV  
Neoplasia treated with cytotoxic drugs  
Autism  
ADD/ADHD  
Environmental illness  
Exocrine pancreatic defects  
Poor digestion  
Iron deficiency  
Bowel cancers.  
Vitiligo  
Vaginitis  
(Galland 1995)

*Pitta* which sets up a cycle of inflammation, and then *Vata*, which is responsible for degenerative changes.

The modern theory of Intestinal Permeability is that some agent or combination of agents initiates an inflammatory response in the digestive tract. Persistent GI inflammation eventually disrupts the integrity of the mucosal lining of the gut, and tiny perforations allow for molecules larger than usual to pass across this barrier, including molecules from dietary protein and fats, bacteria, parasites and fungi. In response to this infiltration, an immune response is initiated and the body begins to manufacture specific antibodies to these antigens. Unfortunately, human tissues have antigenic sites almost identical to those substances that pass across a permeable intestinal wall. These antibodies then circulate throughout the body and “look” for more antigens. When an antigen is found, such as a tissue that has similar markers to an exogenous antigen, the antibody initiates an immune response and the tissue begins to be destroyed. Factors that directly or indirectly promote gut irritation and inflammation include antibiotics, alcohol, caffeine, parasites, pathogenic bacteria, peroxidized fats, some food preservatives and food additives, enzyme deficiencies (e.g. celiac disease, lactose intolerance), NSAIDs, corticosteroids, refined carbohydrates, oral contraceptives and mycotoxins (from stored grains and dried fruit).

Chronic gut inflammation damages the protective coating of IgA, the antibody that functions to maintain the integrity of the mucous membranes. As a result, the inhibition of a localized immune response allows for a decline of non-specific resistance, and an increased risk of viral, bacterial, fungal and parasite infection. The toxins that are produced by these microorganisms can inhibit liver function, and promote a heightened sensitivity to antigenic triggers such as cigarette smoke and strong perfumes. Thus many environmental sensitivities, from such as hayfever, could be caused, in part, by intestinal permeability.

Chronic gut inflammation also damages the absorptive capacity of the epithelium, and thus plays an important role in nutrient deficiency. Antibody-mediated destruction of transport proteins will also inhibit the absorption of these nutrients. Thus, conditions such as osteoporosis and fibromyalgia that often display a deficiency of certain minerals such as zinc, calcium and magnesium, can be reasonably linked with increased intestinal permeability. Beyond the impaired absorption of minerals, deficiencies of vitamins, amino acids and essential fatty acids can lead the development of many conditions, and such generalized complaints as fatigue, irritability, poor concentration and headaches.

Achlorhydria (absence of stomach acid) and hypochlorhydria (deficiency of stomach acid) are increasingly recognized as a factor in leaky gut syndrome and autoimmune disorders, as well as many nutrient deficiencies such as Vitamin B12 (Kelly 1997). As we age, the parietal cells that secrete hydrochloric acid (HCl) begin to atrophy, and the levels of HCl drop. HCl is absolutely necessary for the activation of an enzyme called intrinsic factor, which then assists in the absorption of vitamin B12, or extrinsic factor, necessary for proper nervous function and the formation of red blood cells. Other common nutrient deficiencies in hypochlorhydria include protein deficiencies, as well as mineral deficiencies such as chromium, copper, iron, magnesium, manganese,

molybdenum, selenium and zinc (Bergner 1997, 292). Apart from aging, the causes of low stomach acid include hereditary factors, chronic stress, and most commonly, the use of antacids and antiulcer medications such as cimetidine and ranitidine. Low stomach acid is difficult to diagnose, and many different techniques exist that can provide some indication. Hypochlorhydria can be inferred by the presence of pernicious anemia, poor gastric motility and poor digestion, seen in the iris as a darkish area around the pupil of the eye, and determined from stool samples in which animal proteins will be poorly digested. Low stomach acid is best treated by dealing with chronic stress issues, using bitter herbs such as Gentian (*Gentiana luteum*) and Barberry (*Berberis vulgaris*), and broad spectrum digestive enzymes that include HCl, papain, bromelain and pancreatin. Additional measure include supplementing with the B complex vitamins, vitamin C, and taking apple cider vinegar regularly (but not in *Candida*).

Another factor in the equation is the presence of beneficial bacteria such as *Lactobacillus acidophilus* and *Bifidobacterium bifidum*. Research has shown that these friendly bacteria enhance the capacity of the immune cells in the gastrointestinal tract to defend against foreign pathogens. These immune cells maintain an important link to other cells in the immune system, and appear to have a modulating role in immune function. Clinical research has shown that the oral supplementation of friendly bacteria enhances the phagocytic activity of the immune system, and provides sustained immunological protection, both within and without the gastrointestinal tract. Additionally, these bacterium compete with other pathogenic organisms such as *Candida albicans* and the diarrhea-causing *Clostridium difficile*.

## Treatment of Autoimmune disorders

The treatment of autoimmune disorders is strengthened by an understanding of the underlying energetic principle of treatment. Using an Ayurvedic approach, autoimmune conditions with a *Kapha*-type variant would benefit from warm, dry and stimulating therapies, to remove congestion, enhance circulation and promote digestion. *Pitta*-type variants of autoimmune disorders benefit from cooling, moistening and grounding therapies. *Vata*-type variants of autoimmune disorders require warming, moistening and rejuvenating therapies.

Generally, the treatment of autoimmune conditions involves several steps:

### **1. Reduce and extinguish the inflammatory cascade.**

This is the primary treatment of autoimmune disorders by modern medicine. Unfortunately, patients with autoimmune disorders are in a weakened state, and the additional toxic burden of NSAIDs (which promote liver damage and irritate the gut wall) and corticosteroids (which depress immune function) is often too much. Botanicals with antiinflammatory properties include: Black Cohosh (*Cimicifuga racemosa*), Turmeric (*Curcuma longa*), Wild Yam (*Dioscorea villosa*), Amla berry (*Emblica officinalis*), Ash (*Fraxinus excelsior*), Licorice root (*Glycyrrhiza glabra*), Lignum vitae (*Guaiacum officinalis*), Devil's Claw

(*Harpagophytum procumbens*), Buckbean (*Menyanthes trifolata*), Trembling Aspen (*Populus tremuloides*), Willow (*Salix spp.*), Huang Qin (*Scutellaria baicalensis*), Sarsaparilla (*Smilax spp.*), Feverfew (*Tanacetum parthenium*), and Ashwagandha (*Withania somnifera*).

Where mucous membranes are concerned, the initial use of demulcents followed by astringents is often the best course. Demulcents are helpful to reestablish the integrity of the epithelial lining of the gut, and are better for constipative conditions. Astringents tone the mucous membranes and firm the musculature of the bowel, and are more appropriate for diarrheal conditions. Antispasmodics relieve abdominal pain and hyperperistalsis, slowing the frequency of loose motions. Of special note is the potentially toxic Belladonna (*Atropa belladonna*), which through the antimuscarinic activity of its tropane alkaloids inhibits peristalsis and gastrointestinal hypersecretion.

Demulcents: e.g. Kumari (*Aloe vera*), Marshmallow root (*Althaea officinalis*), Slippery Elm (*Ulmus fulva*), Licorice root (*Glycyrrhiza glabra*)

Antispasmodics: e.g. Black Cohosh (*Cimicifuga racemosa*), Wild Yam (*Dioscorea villosa*), Belladonna (*Atropa belladonna*- TOXIC)

Astringents: e.g. Goldenseal (*Hydrastis canadensis*), Witch Hazel (*Hamamelis virginiana*), Bayberry (*Myrcia cerifera*), White Oak (*Quercus alba*)

Additionally, some herbs are useful for relieving musculoskeletal pain, such as Arnica (*Arnica montana*), Wild Lettuce (*Lactuca virosa*), Jamaican Dogwood (*Piscidia erythrina*), Nutmeg (*Myristica fragrans*) and California Poppy (*Eschscholzia californica*).

## **2. Support liver function and enhance detoxification.**

This is not an area that is given much consideration in modern medicine, probably because the rationale for such an approach is derived from older ideas of autotoxicity. The importance of the liver has in maintaining health cannot be argued however, and with increasing exposure to environmental contaminants, mutagens and xenobiotics, liver detoxification pathways can become quickly overwhelmed. If mercury amalgams are present they should be removed carefully and with the use of intravenous chelating agents to prevent toxicity.

There are many botanicals that display hepatoprotective and hepatoregenerative properties. In any protocol for autoimmune conditions, such herbs should be considered: Barberry (*Berberis spp.*), Turmeric (*Curcuma longa*), Licorice (*Glycyrrhiza glabra*), Boldo (*Peumus boldo*), Bhumyamalaki (*Phyllanthus amarus*), Katuka (*Picrorrhiza kuroa*), Dan Shen (*Salvia miltiorrhiza*), Wu Wei Zi (*Schizandra chinensis*), Huang Qin (*Scutellaria baicalensis*), Milk Thistle (*Silybum marianum*), and Guduchi (*Tinospora cordifolia*).

Additional botanicals that have an affinity for removing congestion and eliminating wastes from the body include: Celery seed (*Apium graveolens*), Burdock (*Arctium lappa*), Cleavers (*Galium aparine*), Red Clover (*Trifolium pratense*) and Nettle (*Urtica dioica*).

### **3. Support immune function and enhance non-specific resistance.**

This includes the use of antivirals, antimicrobials, and botanicals that either stimulate or modulate immune function. The use of herbs with an immunostimulant activity in autoimmune conditions is something of a controversy among practitioners, because if an autoimmune condition is an expression of an immune system gone out of control, stimulation could exacerbate the condition. Thus the use of directly immunostimulant botanicals such as *Echinacea* should be approached with caution in autoimmune disorders. Another category of botanicals, called immunomodulants, appear to be more appropriate to autoimmune conditions, although where one herb ceases being immunostimulant and becomes immunomodulant is a gray area, and is likely highly individual.

Antivirals: Garlic (*Allium sativum*), St. John's Wort (*Hypericum perforatum*), Ban Lan Gen (*Isatis tinctoria*), Biscuit root (*Lomatium dissectum*), Osha (*Ligusticum spp.*), Lemon Balm (*Melissa officinalis*), Cubeb (*Piper cubeba*), and Western Red Cedar (*Thuja plicata*).

Antibacterials: Wild Indigo (*Baptisia tinctoria*), Guggulu (*Commiphora mukul*), Purple Coneflower (*Echinacea angustifolia*), Huang Lian (*Coptis chinense*), Lian Qiao (*Forsythia suspens*), Jin Yin Hua (*Lonicera japonica*), Ban Lan Gen (*Isatis tinctoria*), Huang Qin (*Scutellaria baicalensis*), and Goldenseal (*Hydrastis canadensis*).

Paracidals: Garlic (*Allium sativum*), Sweet Annie (*Artemisia annua*), Nimba (*Azadirachta indica*), Malefern (*Dryopteris felix-mas*), and Kutaj (*Holarrhena antidysenterica*)

Fungicidals: Sweet Annie (*Artemisia annua*), Nimba (*Azadirachta indica*), Huang Lian (*Coptis chinense*), Bhringaraj (*Eclipta alba*), Toothache plant (*Spilanthes acmella*), Pau d'Arco (*Tabebuia spp.*), Haritaki (*Terminalia chebula*), and Bibhitaki (*Terminalia bellerica*).

Immunostimulants: Red root (*Ceanothus spp.*), Purple Coneflower (*Echinacea angustifolia*), Biscuit root (*Lomatium dissectum*), and Western Red Cedar (*Thuja plicata*)

Immunomodulants: Huang Qi (*Astragalus membranaceus*), Amla berry (*Emblica officinalis*), Ling Zhi (*Ganoderma spp.*), Siberian Ginseng (*Eleuthrococcus senticosus.*), Wu Wei Zi (*Schizandra chinense*), and Ashwagandha (*Withania somnifera*).

### **4. Adjust flora of the bowel.**

Following a regimen that inhibits the growth of pathogenic microbes in the intestine, it must be followed by introducing and supporting healthy gut flora. The obvious choice here is a non-dairy derived *Lactobacillus acidophilus* and *Bifidobacterium bifidum* supplement, preferably encapsulated or in tablet form. The dosage is between 6 and 18 billion bacteria, thrice daily. Additionally, recent research has suggested that polysaccharides produced by the mucosal secretions of the body may act as a first line of defense by binding with the carbohydrate-

binding proteins on the cell membranes of pathogenic bacteria. The “decoy” activity of such carbohydrates disables bacteria from adhering to the mucosa of the gut wall. This suggests that similar polysaccharides from exogenous sources may exhibit the same activity. Further, some polysaccharides such as inulin appear to be almost exclusively fermented by bifidobacteria, the so-called “friendly” bacteria (Roberfroid 1993). This leads to an overall increase in fecal bacterial biomass, a decrease in ceco-colonic pH, and a positive effect upon lipid metabolism. Many medicinal plants contain inulin, such as Burdock (*Arctium lappa*) and Dandelion root (*Taraxacum officinale*).

### **5. Eliminate or reduce the insult to the gut barrier.**

Diet is an all important factor in autoimmune disease, and care must be taken to eliminate those foods which initiate an immune response. To determine which foods may be a factor in the disorder, an eliminative diet is best undertaken, removing the most common of food allergens from the diet for a period of two weeks, and then gradually introducing them back into the diet, one at a time over a period of one week, and noting any symptoms. Other important strategies to enhance gut function include:

- Limiting the use of antibiotics and NSAIDS as they causes gastrointestinal irritation.
- Reducing exposure to xenobiotics such as pesticides, insecticides and herbicides, emphasizing organically grown vegetables and free range, hormone/antibiotic-free animal produce.
- Increasing intake of high fiber foods and foods rich in antioxidant phytochemicals such as broccoli, cabbage, cauliflower, beets, carrots and onions.
- Increasing intake of omega 3 fatty acids, such as salmon, halibut and arctic char or supplementing with seeds high in omega 3 fats, such as flax and hemp oil, to inhibit the inflammatory cascade.
- Supplementing with a chelated multimineral and trace mineral supplement to provide for the manufacture of detoxification enzymes and antioxidants.
- Supplementing with digestive enzymes (e.g. bromelain, HCl, pancreatin) to counter hypochlorhydria and to improve digestion. Bromelain and pancreatin also inhibit antiinflammatory prostaglandins and leukotrienes.
- Supplementing with glucosamine to improve bowel wall integrity
- Supplementing with antioxidants such as N-acetyl cysteine to limit oxidative stress, but only after protozoal infections have been dealt with.

To accurately assess for intestinal permeability, some diagnostic laboratories offer a simple test that measures the ability of two non-metabolized sugar molecules, mannitol and lactulose, to permeate the intestinal mucosa. Mannitol is easily absorbed and serves as a marker of transcellular uptake, while lactulose is only slightly absorbed and serves as a marker for mucosal integrity. To perform the test, the patient mixes premeasured

amounts of lactulose and mannitol and drinks the challenge substance. The test measures the amount of lactulose and mannitol recovered in a urine sample over the next 6 hours.