Food Lectins in Health and Disease: An Introduction

The name “lectin” is derived from the Latin word *legere*, meaning “to select”. Although they were first discovered more than 110 years ago in plants, they are now known to be present throughout nature. Most lectins are basically non-enzymic in action and non-immune in origin. Lectins occur ubiquitously in nature. They may bind to a soluble carbohydrate or to a carbohydrate moiety which is a part of a glycoprotein or glycolipid. They typically agglutinate certain animal cells and/or precipitate glycoconjugates.

Lectins serve many different biological functions in animals, from the regulation of cell adhesion to glycoprotein synthesis and the control of protein levels in the blood. They may also bind soluble extracellular and intercellular glycoproteins.

Some lectins are found on the surface of mammalian liver cells which specifically recognize galactose residues. It is believed that these cell-surface receptors are responsible for the removal of certain glycoproteins from the circulatory system.

Another lectin is a receptor which recognizes hydrolytic enzymes containing mannose-6-phosphate, and subsequently targets these proteins for delivery to the lysosomes. I-cell disease is one type of defect in this particular system.

Lectins are also known to play important roles in the immune system by recognizing carbohydrates that are found exclusively on pathogens, or that are inaccessible on host cells. Examples are the lectin complement activation pathway and Mannose binding lectin.

The function of lectins in plants is still uncertain. Once thought to be necessary for rhizobia binding, this proposed function was ruled out through lectin-knockout transgene studies.
The large concentration of lectins in plant seeds decreases with growth, and suggests a role in plant germination and perhaps in the seed's survival itself. The binding of glycoproteins on the surface of parasitic cells is also believed to be a function.

**Use in medicine and medical research**

Purified lectins are important in a clinical setting because they are used for blood typing. Some of the glycolipids and glycoproteins on an individual's red blood cells can be identified by lectins.

- A lectin from *Dolichos biflorus* is used to identify cells that belong to the A1 blood group.
- A lectin from *Ulex europaeus* is used to identify the H blood group antigen.
- A lectin from *Vicia graminea* is used to identify the N blood group antigen.

In neuroscience, the anterograde labeling method is used to trace the path of efferent axons with PHA-L, a lectin from the kidney bean.

**Use in studying carbohydrate recognition by proteins**

Lectins from legume plants, such as PHA or concanavalin A, have been widely used as model systems to understand the molecular basis of how proteins recognize carbohydrates, because they are relatively easy to obtain and have a wide variety of sugar specificities. The many crystal structures of legume lectins have led to a detailed insight of the atomic interactions between carbohydrates and proteins.

**Use in biochemical warfare**

One example of the powerful biological attributes of lectins is the biochemical warfare agent ricin. Ricin is isolated from seeds of the castor oil plant and is a protein that comprises two domains,

- One is a lectin that binds cell surface galactosyl residues and enables the protein to enter cells.
• The second domain is an N-glycosidase that cleaves nucleobases from ribosomal RNA resulting in inhibition of protein synthesis and cell death.

In the past two decades we have realised that many lectins are

(a) Toxic, inflammatory, or both;
(b) Resistant to cooking and digestive enzymes;
(c) Present in much of our food.

It is thus no surprise that they sometimes cause “food poisoning.” But the really disturbing finding came with the discovery in 1989 that some food lectins get past the gut wall and deposit themselves in distant organs.

So do they cause real life diseases?

This is no academic question because diet is one part of the environment that is manipulable and because lectins have excellent antidotes, at least in vitro. Because of their precise carbohydrate specificities, lectins can be blocked by simple sugars and oligosaccharides. Wheat lectin, for example, is blocked by the sugar N-acetyl glucosamine and its polymers. These natural compounds are potentially exploitable as drugs should lectin induced diseases be identified.

Wheat gliadin, which causes coeliac disease, contains a lectin like substance that binds to human intestinal mucosa, and this has been debated as the “coeliac disease toxin” for over 20 years. But coeliac disease is already managed by gluten avoidance, so nothing would change were the lectin hypothesis proved. On the other hand, wheat lectin also binds to glomerular capillary walls, mesangial cells, and tubules of human kidney and (in rodents) binds IgA and induces IgA mesangial deposits. This suggests that in humans IgA nephropathy might be caused or aggravated by wheat lectin; indeed a trial of gluten avoidance in children with this disease reported reduced proteinuria and immune complex levels.
Of particular interest is the implication for autoimmune diseases. Lectins stimulate class II HLA antigens on cells that do not normally display them, such as pancreatic islet and thyroid cells. The islet cell determinant to which cytotoxic autoantibodies bind in insulin dependent diabetes mellitus is the disaccharide N-acetyl lactosamine, which must bind tomato lectin if present and probably also the lectins of wheat, potato, and peanuts. This would result in islet cells expressing both class II HLA antigens and foreign antigen together—a sitting duck for autoimmune attack. Certain foods (wheat, soya) are indeed diabetogenic in genetically susceptible mice. Insulin dependent diabetes therefore is another potential lectin disease and could possibly be prevented by prophylactic oligosaccharides.

Another suspect lectin disease is rheumatoid arthritis. The normal human IgG molecule possesses carbohydrate side chains, which terminate with galactose. In rheumatoid arthritis much of the galactose is missing, so that the subterminal sugar—N-acetyl glucosamine—is exposed instead. These deficient IgG molecules feature strongly in the circulating immune complexes that cause fever and symptoms.

In diet responsive rheumatoid arthritis one of the commonest trigger foods is wheat and wheat lectin is specific for N-acetyl glucosamine—the sugar that is normally hidden but exposed in rheumatoid arthritis. This suggests that N-acetyl glucosamine oligomers such as chitotetraose (derived from the chitin that forms crustacean shells) might be an effective treatment for diet associated rheumatoid arthritis. Interestingly, the health food trade has already seized on N-acetyl glucosamine as an antiarthritic supplement.

Among the effects observed in the small intestine of lectin fed rodents is stripping away of the mucous coat to expose naked mucosa and overgrowth of the mucosa by abnormal bacteria and protozoa. Lectins also cause discharge of histamine from gastric mast cells, which stimulates acid secretion. So the three main pathogenic factors for peptic ulcer—acid stimulation, failure of the mucous defence layer,
and abnormal bacterial proliferation (*Helicobacter pylori*) are all theoretically linked to lectins.

If true, blocking these effects by oligosaccharides would represent an attractive and more physiological treatment for peptic ulcer than suppressing stomach acid. The mucus stripping effect of lectins also offers an explanation for the anecdotal finding of many allergists that a “stone age diet,” which eliminates most starchy foods and therefore most lectins, protects against common upper respiratory viral infections: without lectins in the throat the nasopharyngeal mucus lining would be more effective as a barrier to viruses.

But if we all eat lectins, why do not we all get insulin dependent diabetes, rheumatoid arthritis, IgA nephropathy, and peptic ulcers? Partly because of biological variation in the glycoconjugeats that coat our cells and partly because these are protected behind a fine screen of sialic acid molecules, attached to the glycoprotein tips. We should be safe. But the sialic acid molecules can be stripped off by the enzyme neuraminidase, present in several micro-organisms such as influenza viruses and streptococci. This may explain why diabetes and rheumatoid arthritis tend to occur as sequel of infections. This facilitation of lectins by micro-organisms throws a new light on post infectious diseases and makes the folklore cure of fasting during a fever seem sensible.

Alternative medicine popularisers are already publishing articles about dietary lectins, often with more enthusiasm than caution, so patients are starting to ask about them and doctors need to be armed with facts. The same comment applies to entrepreneurs at the opposite end of the commercial spectrum. Many lectins are powerful allergens, and prohevein, the principal allergen of rubber latex, is one. It has been engineered into transgenic tomatoes for its fungistatic properties, so we can expect an outbreak of tomato allergy in the near future among latex sensitive individuals. Dr Arpad Pusztai lost his job for publicising concerns of this type.
In recent years it appears there is a rising epidemic of people suffering from chronic digestive and autoimmune conditions. Food intolerance or sensitivities may lie at the root of the problem. Most people, including doctors, have no clue how foods they eat may be contributing to their chronic illness, fatigue and digestive symptoms. There are, however, a lot of clues in the medical literature and the lay public’s experience about how foods are causing and/or contributing to the current epidemic of chronic illness and autoimmune disease. There are several diets being used by many people with varying success to improve their health despite a general lack of iron clad scientific proof for their effectiveness. One of the clues to the cause and relief of food induced illness may lie in proteins known as lectins that are present in all foods.

Animal and plant sources of food both contain complex proteins known as lectins. These proteins typically have the ability to attach to sugars or carbohydrates on the surface of human cells. Some of these proteins can cause clumping of human red blood cells, a process that is called agglutination. The process of agglutination occurs when someone receives the wrong blood type during a blood transfusion. In fact, red blood cell agglutination specific to each person or groups of people is the basis for testing for blood types.

The attachment or binding of certain food lectins can initiate a variety of cell specific effects. These reactions may mimic hormones or cause changes in cells. This is termed molecular mimicry. Most plants contain lectins, some of which are toxic, inflammatory, or both. Many of these plant and dairy lectin are resistant to cooking and digestive enzymes. Grain lectins, for example, are quite resistant to human digestion but well suited for ruminants like cattle that have multi-chambered stomachs.

Therefore, lectins are present in our food and are often resistant to our digestion and some have been scientifically shown to have significant GI toxicity in humans. Others have been shown to be beneficial and maybe even cancer protecting. Either way, plant and

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animal proteins are foreign proteins to the body and are dealt with by digestion and our immune system in a positive or negative manner. The human digestive system was created to handle a variety of plant and animal proteins through the process of digestion and elimination. Some plant and animal proteins or lectins are severely toxic to humans and cannot be eaten without causing death like those in Castor beans and some mushrooms. Other foods must be prepared before they are safe to be eaten. Preparations may include pealing, prolonged soaking and cooking like kidney beans. Other foods may be poorly tolerated because of a genetic predisposition or underlying pre-existing food allergy or intolerance. Others are tolerated to some degree or quantity but not in large amounts or on a frequent basis. People who are intolerant to the milk sugar lactose, because of inherited or acquired deficiency in lactase enzyme, may tolerate small amounts but may have severe bloating, gas, abdominal pain and cramps with explosive diarrhea when a large amount of lactose containing foods are eaten. Foods can become intolerable to some people after their immune system changes or gut is injured from another cause.

Of the food lectins, grain/cereal lectins; dairy lectins; and legume lectins (especially peanut lectin and soybean lectin) are the most common ones associated with reports of aggravation of inflammatory and digestive diseases in the body and improvement of those diseases and/or symptoms when avoided. Recent research has suggested that these lectins may effectively serve as a “Trojan horse” allowing intact or nearly intact foreign proteins to invade our natural gut defenses and enter behind the lines to cause damage well beyond the gut, commonly in joints, brain, and skin of affected individuals. Once damage occurs to the gut and the defense system is breached the result is what some refer to as a “leaky gut”. Moreover, many people who develop a “leaky gut” not only have gut symptoms such as bloating, gas, diarrhea, and abdominal pain but also other symptoms beyond the gut, or extra-intestinal symptoms. Commonly affected areas are the brain or peripheral nerves, skin, joints, and various body glands. With continued exposure of the gut www.healthoracle.org
by these toxic food lectins a persistent stimulation of the body’s defense mechanism in a dysfunctional manner, occurs, i.e. autoimmune disease.

Wrong types or levels of good and bad bacteria in the gut, or intestinal dysbiosis, may contribute to this process of abnormal stimulation of the immune system. Research supports the strong possibility that such stimulation may be accentuated by interaction of the bacteria with food lectins. It is believed by some that this may further worsen gut injury and autoimmune disease.

This latter concept is gaining acceptance and recognition by doctors in one form as the hygiene theory. It is speculated that our gut bacteria have become altered by increased hygiene and over use of antibiotics and that this phenomenon may be playing a significant role in the rising incidence of autoimmune diseases such as diabetes, arthritis, and chronic intestinal diseases like Crohn’s disease and irritable bowel syndrome.

Lectins as a cause however are largely being ignored in the U.S. though the field of lectinology and lectins role in disease is more accepted internationally. Avoidance of certain food lectins may be helpful in achieving health and healing of chronic gut injury. Healing of a “leaky gut” and avoidance of ongoing abnormal stimulation of the immune system by toxic food lectins and bacteria in the gut is the basis for ongoing research and probable success of several popular diets such as the paleo diet, carbohydrate specific diet and gluten-free/casein-free diet.

More research is needed in this exciting but often neglected area. The Food Doc, LLC features a website www.thefooddoc.com that will provide physician authored information on food intolerance, sensitivity and allergy such as lectin, gluten, casein, and lactose intolerance with dietary guidance that will feature in the near future an online symptom assessment and diet-diary.
Definition

Protein or glycoprotein substances, usually of plant origin, of non-immunoglobulin nature, capable of specific recognition of and reversible binding to, carbohydrate moieties of complex glycoconjugates without altering the covalent structure of any of the recognized glycosyl ligands. This group includes monovalent lectins (i.e. bacterial and plant toxins). These lectins bind to sugar moieties in cell walls or membranes and thereby change the physiology of the membrane to cause agglutination, mitosis, or other biochemical changes in the cell. (Agglutination- clumping; Mitosis-multiplication or division of a cell forming two daughter cells)

Lectins were first described in 1888 by Stillmark working with castor bean extracts. Many members of the lectinic protein family agglutinate (clump together) red blood cells. Research done by Ehrlich, considered to be the father of immunology, has shown that feeding small amounts of lectin containing seeds to rabbits caused partial immunity to the toxicity demonstrating lectins are also antigenic (able to induce antigen antibody reactions).

High levels of lectins (specialized proteins) may be found in grains (also known as cereals or pulses), legumes (that is ‘beans’ including peanuts), dairy and plants in the nightshade family. Many other foods contain lectins but are less well studied and the amounts of lectins present are not thought to be as high or as potentially toxic.

Lectins purified from the germinating seeds of wheat (Triticum spp.); bind to carbohydrate moieties on cell surface glycoproteins and are used to identify certain cell populations and inhibit or promote some immunological or physiological activities.

Lectins purified are used to determine one’s blood type (ABO). Lectins from the castor bean are highly toxic and can kill if ingested in even small amounts. Lectins from kidney beans have been
implicated as cause in an outbreak of ‘food poisoning’ with no known pathogen.

Think of a lectin as a protein containing a key that fits a certain type of lock. This lock is a specific type of carbohydrate. All life forms, plant and animal, insect and fungus have cell membranes that contain carbohydrates that sit within and project from the membrane. If a lectin with the right key comes in contact with one of these ‘locks’ on the gut wall or artery or gland or organ it ‘opens the lock’, that is disrupts the membrane and damages the cell and may initiate a cascade of immune and autoimmune events leading to cell death.

Lectins can be inactivated by specific carbohydrates (technically known as mono and oligosaccarides) which can bind the ‘key’ and prevent the protein from attaching to the carbohydrate ‘lock’ within the cell membrane. Glucosamine is specific for wheat lectin and it is this specificity that may protect the gut and cartilage from cell inflammation and destruction in wheat (or gluten) responsive arthritis.

While various foods and supplements may inactivate some of these toxic lectins it is impossible for such substances to protect the body from them completely. The safest path is avoidance of known toxic lectins.

Many dietary programs are promoted with moderate ‘good’ fats and moderate complex carbohydrates and plenty of fruits and vegetables. For some patients consistently eating enough protein was and is difficult. They (and the culture in which we currently reside) tend to diminish protein’s important contribution to health, both mental and physical. When protein intake is maximized patients have found this moderate, easy to follow, program has aided them in restoring function of body and mind.

The most common potentially 'toxic' lectin containing food groups are:
- grains, especially wheat and wheat germ but also quinoa, rice, buckwheat, oats, rye, barley, millet and corn.
- legumes (all dried beans, including soy and peanuts),
- dairy (perhaps more so when cows are feed grains instead of grass, a speculation based on research showing transference of lectins into breast milk and dairy and potentially more harmful in pasteurized, processed milk because of the reduction of secretory IgA, an immunoglobulin that binds dangerous lectins. The high lectin-binding capacity of human secretory IgA protects nonspecifically mucosae against environmental antigens.).
- nightshade (includes potato, tomato, eggplant and pepper).

Each of these groups has a history of being implicated as allergenic. Also note that we are including all foods made from these substances, (these substances in all forms, milled grains, flours, oils, vinegars), peanut butter, cereal or legume oils (soy, canola, corn), additives, thickeners, grain vinegar and products containing grain vinegar, grain alcohol including grain based vodka, and all beers and ales. The only non-grain based alcohols are 100% Agave tequila and 100% potato vodka. Grape based alcoholic beverages are probably allowed if you know you tolerate them.

There has been some information that lectins may be inactivated by soaking, sprouting, cooking or fermenting. Soaking legumes over night, draining the water, rinsing and draining again does seem to remove or inactivate many of the lectins. Heating seems to remove others in some foods but not all. There is little data to prove that any of these methods remove lectins completely as few foods have been tested and of those that have lectins many seem to remain after processing.

Nachbar and Oppenheim (1980) found 30% of fresh and PROCESSED foods contained active lectins. Lectins from green salads, fruits, spices, seeds, dry cereals and nuts (even after roasting) showed activity of potentially toxic lectins. Some of these lectins

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interact with serum or salivary components and bacteria from the oral cavity.

Another example of the hardiness of lectins is the study by Klurfeld DM and Kritchevsky D Lipids 1987 Sep;22 (9):667-8, Isolation and quantitation of lectins from vegetable oils. Results-Unrefined soy oils contained 858-2983 mcg/kg. After refining the oils contained 24-55 mcg/kg. Both refined and unrefined soya oil contained soya lectins.

Common features of toxic (non-nutritive) effects in lectin-gut interactions

1. High degree of resistance to gut proteolysis.
2. Binding to brush border cells; damage to microvillus membrane; shedding of cells; reduction in the absorptive capacity of the small intestine.
3. Increased endocytosis; induction of hyper-plastic growth of the small intestine; increased turnover of epithelial cells.
4. Interference with the immune system; hypersensitivity reactions.
5. Interference with the microbial ecology of the gut; selective overgrowth.
6. Direct and indirect effects (hormones, etc.) on systemic metabolism.

Especially note #5. The popular Candida Diet is essentially a high protein, low carbohydrate diet which limits starches and sugars and thereby limits lectins. If lectins are a problem for this person (the so-called 'candida' patient) lectin ingestion may be associated with overgrowth of various gut pathogens that may include yeasts and removal of lectins would restore the gut ecology and the gut immune system. If this is true, the diet does not get rid of yeast but relieves the person from symptoms and pathogenic consequences caused by ingestion of lectins to which he or she is intolerant.
Lectins are hardy proteins that do not break down easily. They are resistant to stomach acid and digestive enzymes.

Lectins may bind to the gut wall and damage the gut lining, are not altered by digestive enzymes, and may alter gut permeability and pass through the gut into general circulation.

Lectins can cause alterations in gut function that may be related to colitis, Crohn’s Disease, Celiac-Sprue, IBS and gut permeability.

Lectin damage to the gut wall may allow other non-lectin proteins to cross undigested into general circulation and cause allergic reactions, including anaphylaxis.

Having gained access to general circulation various lectins may bind to surface cell membranes in arteries and vessels, organs and glands, including the thyroid, pancreas, kidney and adrenals, in susceptible animals and humans.

This binding may begin antigen antibody reactions leading to autoimmune disorders and so-called degenerative diseases.

Different lectins have been implicated in different diseases. Dairy lectins have been implicated in juvenile onset type I diabetes. Wheat lectins have been implicated in juvenile nephropathy.

Type or types of lectin and one’s susceptibility (genetic susceptibility) cannot be determined by blood type. Lectin intolerance reactions occur in the gut, general circulation (artery walls and the like), brain, gland or organ as well as red blood cells. Sensitivity of one type of cell does not necessarily determine whether another type cell will or will not react.

Secretory IgA, and other immune factors may, if sufficient in quantity, help protect against some exposure to toxic lectins.

GM (genetically modified foods) is modified by splicing ‘lectins’ from one plant family to another. This is extremely problematic. If you know you react to a particular plant family but that lectin has been put in a plant not of that family you may consume the ‘toxic to you’ lectin, have the reaction/response and not know the cause.

We are or become lectin sensitive because of:
- genetics, our ancestors just did not evolve eating this type of food and our immune system cannot handle it;
- a failure of Secretory IgA barrier protection, genetic or environmentally induced;
- bacterial or virus infection, certain bacteria and virus, including the influenza virus, can damage our cells making them susceptible to lectin antibody/antigen reactions
- Use of NSAIDS (non-steroidal anti-inflammatories) or other drugs which increase gut permeability and allow lectins to enter general circulation.

Historically diagnosis and treatment of Celiac-Sprue related to 'gliadin' (also known as gluten) sensitivity. Gliadin is found in wheat, rye, barley, oats, and foods containing these grains (including beer, grain based alcohols, mayonnaise, grain vinegar, etc). Some Celiacs did not respond to elimination of gluten/gliadin. In 1951 Drs. Sidney V. and Merrill P. Haas published Management of Celiac Disease documenting treatment and cure of celiac and cystic fibrosis of the pancreas with a carbohydrate limiting diet introduced as the ‘Specific Carbohydrate Diet’.

In many cases cited in the book, elimination of certain carbohydrates ‘cured’ diagnosed Celiacs after one year and they were able to return to eating gluten containing foods. In hindsight many of the foods eliminated in this plan are high lectin foods known to be associated with gut and systemic inflammatory reactions. Celiac-Sprue is a genetic disorder treated by elimination of offending foods. The response of some to the specific carbohydrate elimination diet would likely mean that the patients who responded did not have classic gluten intolerance, Celiac-Sprue, which requires life long elimination of gluten/gliadin. It suggests that other lectins may cause similar symptoms and overlapping diagnostic and treatment difficulties.
If all cases of lectin intolerance were genetically based reversal of intolerance would not be possible. There must therefore be a subgroup of IBS, Crohn's, Celiac, colitis that is related to sensitization to food lectins that can be reversed by avoidance of these lectins and a restoration of gut function including Secretory IgA and other immune protectors. Bacteria, virus, or other conditions, drugs or injurious substances acting directly on the gut wall may cause sensitization.

Tests are available to determine SIgA levels, and gut immune reactions to soya, dairy, wheat and egg. These tests do not cover the entire family of lectins, nor would blood or skin tests necessarily show sub-clinical sensitivity reactions. Most of the conditions associated with sub-clinical lectin intolerance appear to be degenerative, often taking extended periods of time to appear and longer to reach life threatening or painful (such as arthritis) stages. Many lectin related conditions may be considered to be 'autoimmune'.

Awareness of genetically based intolerance to one or more lectin groups is important family information. If you or another family member has such intolerance other family members need to be aware and test themselves to prevent problems before they begin.

Infectious or drug related food intolerance responses need to be prevented or reversed. These antigen/antibody responses may be reversible but avoidance of offending lectins should be considered for a minimum of one year before reintroduction to test.

Lectins and their possible involvement in degenerative and autoimmune disease is a relatively new science.

This report, as presented, is hypothesis, not yet fully supported by clinical trials and not yet at a stage where we have any idea of how to connect 'family' with lectin response. What facts can be supported include-

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Proteins institute most allergic and antigenic responses.

Lectins are proteins found in large amounts in the foods as above.

Lectins are not easily removed from foods or rendered harmless to animals and humans.

Lectins from soya, peanut and other beans, wheat germ and wheat, milk, peanut oil (and perhaps other seed oils including soya oil) and nightshades, in a variety of clinical studies have shown various damage to gut lining, joints, kidney, pancreas and brain (even able to cross the blood-brain barrier).

Lectins found in peanut oil have been implicated in atherosclerosis. Leaving open the possibility that other seed oils contain damaging lectins and that polyunsaturation and free radicals may not be the full picture on the dangers of polyunsaturated fats.

You may react to lectin toxicity due to genetics, intensity of exposure, failure of immune factors to protect you, viral infection, bacterial infection or gut permeability induced by medication or infection.

Lectin toxicity (antigen-antibody response) can be ‘sub-clinical’ not showing obvious symptoms for many years.

Lectin intolerance is not an ‘allergy’

A person may be lectin intolerant and not have antibodies to the suspect food when given an allergy test whether blood or skin or saliva. A person may be lectin intolerant and because of the damage done by lectins end up having allergic reactions to a food (that does not contain lectin or may have other lectins), other chemicals or the environment. Lectin intolerance means the inability to deactivate the toxic lectin (prevent its binding to your cells) in the confines of your own body, be it in the gut, artery, organ, gland or brain. The lectin then proceeds to invoke immune responses that damage the cell to which it attaches and possibly surrounding cells. This antigen/antibody response may be the key to many or even most
autoimmune diseases and many degenerative diseases may need to be reclassified as autoimmune.

If you or other family members are suffering from any of the symptoms, conditions or diseases mentioned in this report consider an elimination diet to test for lectin sensitivity. If you have been diagnosed with Celiac-Sprue by blood and biopsy testing you must remove gluten/gliadin for the rest of your life. (visit http://www.csaceliacs.org/). If, however, elimination of this glycoprotein does not resolve your problem consider other lectin families as possible offenders. It is also possible to be gluten intolerant and intolerant to one or more of the other lectin families.

Elimination Diet:

Remove all suspect lectin families (legumes, dairy, etc) for 7 days. Make sure to read labels so that you are not consuming a part of the lectin family hidden in a food. On day 8 reintroduce several of the family members, such as, if testing dairy, milk, cheese and sour cream or legumes, soya, kidney bean and peanut butter. Eat some of the family at each meal. Stop all of the family for the next two days. That is 7 days off, one day on and 2 days off. Check your symptoms on the day of testing and the following 2 days. Look for changes in energy, appetite, bowel function, mood, sleep, skin, and digestion, anything suspicious. Test only one 'family' at a time. You may remove as many groups as you feel are suspect but only reintroduce one family at a time. If you find you must eliminate one or more lectin families retest every six months to see if the intolerance is genetic or induced.

Common groups:

Dairy; legumes (includes soya and peanuts); nuts; seeds; nightshades, includes potato, tomato, eggplant; eggs; grains, esp. gluten grains such as wheat, rye and barley but corn can be an antigen too. Millet, wild rice and plain white rice are usually safe substitutes while testing grains.

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Most persons are aware that there are certain foods they seem to 'react' to. Symptoms could be obvious, such as gas, bloating, diarrhea or constipation (or both, alternating). Less obvious symptoms may include headache, fatigue, and ‘indigestion’, skin problems including hives, psoriasis, swollen joints or water retention. While some symptoms while resolve quickly after eliminating an offending family other symptoms may take 6-12 months. Be patient. If you are genetically intolerant you will never be able to consume that group of foods safely.

Some symptoms may occur chronically and may seem in no way related to a gut/food or lectin intolerance reactions. This group of symptoms includes the so-called degenerative diseases and autoimmune diseases like those mentioned in the list at the beginning of this report including atherosclerosis, hypertension, osteoporosis, senile dementia, osteoarthritis and rheumatoid arthritis, inflammatory joint diseases, fibromyalgia, chronic fatigue, and adult onset diabetes. Obesity has been associated with consumption of ‘enemy’ lectins.

If your condition responds to elimination of one or more of the high lectin groups, consider your intolerance to be at minimum, induced by the environment (infection or medication induced), and continue to restrict your diet for one year before testing a food-lectin group for re-inclusion. If you again react consider your intolerance a probable genetic inheritance and avoid this type of lectin containing food group as completely as you are able.

For severe symptoms or conditions eliminate all of the major suspect groups, all grains, all legumes, and all dairy. Add the nightshades, potato, tomato, eggplant and pepper, to your restricted list if your symptoms are associated with rheumatic or arthritic complaints. If you respond to this elimination diet by a resolution of symptoms keep out the food group/s for a minimum of six months to one year before reintroducing the group/s. If symptoms reappear consider lifelong avoidance. Rarely does a person have to eliminate more than
one or two of the lectin families on a long-term basis. You must let your body decide.

Throughout our history our ancestors had limited exposure to many lectin families depending on location. In our modern world it is common to believe that we can eat any food we like. We can, but the food we eat may not like us. Some persons (a minority) can tolerate all foods. For the rest of us, most will find one or more lectin groups they do not tolerate. Of those who experience antigen responses most will not need to eliminate more than one or two major lectin groups. You have to experiment and see ‘who’ you are and ‘what’ your ideal foods are. It is a process.

Consider the group most likely to be causing a problem.

- Deadly nightshades including tomato, potato and eggplant.
- Glutens found in wheat, rye, barley, malt, and oats.
- Legumes, all beans including soya and its derivatives and peanut.
- Dairy including all milk products, milk, cheese, cottage cheese, yogurt, kefir.
- Eggs

These are the most common lectin families that cause problems. Eliminate the suspect group for 7-10 days. Do not eat any of the group. Check to make sure none of the lectins are contained in other foods you consume. Example: Vinegar is made from grain and contains gluten unless it is apple cider vinegar or wine vinegar. Mayonnaise contains gluten because it is made with grain vinegar.

After abstaining for 7-10 days eat a significant amount of the suspect group over one day. Eat other foods as well. Do not eat any more of the test group for two days after the test day.

Look for symptoms of intolerance: bowel changes sleep changes, mood changes, memory impairment or any other significant changes you can relate to the ingestion of the food group. It may take a day or
so for the symptoms to appear. If you think you have found a lectin incompatibilities avoid the food. You can test again in a few weeks. If every time you avoid the food your symptoms resolve and every time you eat it they return you have found a lectin you should not eat.

**Lectins are**

- important,
- in most foods, with plant foods containing the highest levels of known toxic lectins
- While they have existed as long as life has existed they are not yet well researched or understood.
- Lectins can be extremely toxic, causing rapid death
- may in some situations and in some species be useful in preventing or reversing conditions and illnesses such as cancer.
- Most lectins fall somewhere in between these two extremes allowing the possibility of subclinical conditions which appear over time and which do not appear to be directly related to lectin exposure.

**The high lectin-binding capacity of human secretory IgA protects nonspecifically mucosae against environmental antigens.**

*Davin JC, Senterre J, Mahieu PR*

Department of Pediatrics, State University of Liege, Belgium.

The anti-infectious role of human milk may be, at least partly, ascribed to its content in secretory IgA. As lectins are present in various infectious antigens, the binding of different types of IgA to three lectins (concanavalin A, peanut agglutinin, wheat germ agglutinin) was studied by Elisa. The specificity of those bindings was assessed by inhibitory experiments performed with the corresponding oligosaccharides. The following were found for the three lectins:

1. The lectin-binding capacity of colostrum secretory IgA was markedly greater than that of normal plasma IgA1 (p less than 0.001);

2. The lectin-binding capacity of polymeric IgA1 was greater than that of monomeric IgA1 (p less than 0.001). This property of mucosal IgA may be responsible of a nonimmune opsonization able to prevent the early step of

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some infectious mucosal diseases, i.e. the attachment of bacteria to epithelial cells by lectin-like bonds and also the penetration into the body of some antigens able to favor the development of allergy. Milk mucosal IgA, present in significant amounts in human colostrum and mature milk - but not in infant formulas - may therefore play an important polyvalent protective role in newborns.

IgA antibodies to dietary antigens and lectin-binding IgA in sera from Italian, Australian, and Japanese IgA nephropathy patients.

Coppo R; Amore A; Roccatello D; Gianoglio B; Molino A; Piccoli G; Clarkson AR; Woodroffe AJ; Sakai H; Tomino Y

We studied serum IgA as antibodies to dietary antigens (Ag), as lectin- binding molecules, and as conglutinin-binding immune complexes (IgAIC) in people from geographical areas in which IgA nephropathy (IgAGN) is particularly frequent.

Sera from 63 Italian, 21 Australian, and 25 Japanese patients affected by IgAGN and 24 Italian, 20 Australian, and 40 Japanese healthy controls were studied.

Increased values of IgAIC were detected in 42.8% of Italian patients, while only in 23.8% and 8% of Australian and Japanese patients, respectively. Mean values were significantly increased only in Italian patients (P less than 0.0001). Positive values of IgA antibodies against dietary Ag had variable prevalences, but again Italian patients showed the highest frequency, from 19% to 28.5% versus 0 to 38% in Australians and 0 to 16% in Japanese.

Mean values of these antibodies were not significantly increased in any patient groups in comparison to the corresponding healthy populations. However, patients with elevated values of IgAIC had significantly higher serum concentrations of antibodies to alimentary components and a linear correlation was found between IgAIC and some IgA antibodies to food components. The relationship between these two series of data was particularly evident for Italian and Australian IgAGN patients. Moreover, the patients with positive data tended to have a cluster of increased levels of IgA antibodies against several alimentary Ag at the same time. A linear correlation was evident between values of IgA antibodies to gluten fractions and to heterologous albumins. None of these correlations was evident among healthy controls.

Identification of lectin binding proteins in human tears.

Kuizenga A, van Haeringen NJ, Kijlstra A

Biochemical Laboratory, Netherlands Ophthalmic Research Institute, Amsterdam.

The identity of glycoproteins in stimulated normal human tears was investigated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) of
tears onto minigels, blotting, and subsequent incubation with different biotinylated
lectins (concanavalin A [Con A], peanut agglutinin [PNA], glycine max agglutinin
[SBA], Phaseolus vulgaris agglutinin, wheat germ agglutinin [WGA, native form],
Artocarpus integrifolia agglutinin [Jacalin], and Pisum sativum agglutinin). Control
proteins included purified secretory immunoglobulin A (sIgA) from human
colostrum, human milk lactoferrin, and chicken-egg lysozyme.

All samples were prepared in a denaturing (SDS) buffer under nonreducing and
reducing conditions. The sIgA in tears and IgA (alpha) heavy chain fragments
(reduced sample) were identified with most of the lectins tested. A particular high
molecular weight (greater than 200 kD) protein fraction in tears that just entered
the separation gel on SDS-PAGE was detected with WGA and Jacalin. This
fraction stains poorly with silver.

Tear lactoferrin was identified with all lectins used, although binding was low with
SBA. Purified milk lactoferrin showed a poor reaction with Jacalin, but a protein in
tears of similar mobility bound this lectin (nonreduced samples). Under both
nonreducing and reducing conditions, tear-specific prealbumin in tears did not
bind any of the lectins tested. Tear lysozyme only reacted with lectin after
reduction. The techniques described may provide additional valuable information
in addition to commonly used methods for tear protein analysis and further
knowledge concerning the role of glycoproteins on the ocular surface.