Lectins

Lectins were first discovered in 1888 by Stillmark while investigating the toxic effects of the castor bean. Over the past 30 years lectins have been shown to be relevant in a wide variety of medical problems that are endemic in our Western society including rheumatoid and other auto-immune arthritis, obesity, auto-immune disease, arteriosclerosis and allergy. Research is still in its infancy, and unfortunately research already completed has not been widely accepted despite overwhelming evidence.

Located on the surface of cells of all living things are thousands of different complex sugar molecules (glycoconjugates) projecting outward from their loose anchors on the cell surface like moving antennae. Genetically unique, these molecules comprise a protective coating for the cell and perform many functions including cell recognition and signalling. Lectins are a class of protein molecules capable of using these sugar moieties to bind to the surface of cells. Lectins provide the way for one molecule to stick to another molecule without any immunity reaction to become involved.(5)

Lectins are present in most plants, especially seeds and tubers like cereals, potatoes, and beans. While they already have a medical use as histology and blood transfusion reagents, in the past three decades we have realised that many lectins are (a) toxic, inflammatory, or both; (b) resistant to cooking and digestive enzymes; and (c) present in much of our food. It is thus no surprise that they sometimes cause “food poisoning” symptoms. 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.(1)

The only thing lectins have in common with each other is their ability to bind to sugars. Lectins bind to the terminal sugar, the “glyco,” portion of glycoconjugates found on cell membranes. If the sugars are bound to proteins they are called glycoproteins or bound to fats they are called glycolipids. Collectively they are called glycoconjugates of which 11 percent of the human body is composed.

The important point is that some of the lectins consumed in everyday foods act as chemical messengers that can in fact bind to the sugars of cells in the gut and the blood cells, initiating an inflammatory response. In wheat, gliadin, a component of gluten and an iso-lectin of wheat germ agglutinin (WGA), is capable of activating NF kappa beta proteins which, when up-regulated, are involved in almost every acute and chronic inflammatory disorder including neurodegenerative disease,(5)

Lectins can also cause a broader immune system response as the body’s defences move in to attack the invaders. Symptoms can include skin rashes, joint pain, and general inflammation. Lectin intolerance is not an “allergy.” Allergy tests usually do not detect this intolerance, but because of the damage caused by the lectins end up having allergic reactions to a food, other chemicals or even to the environment. The lectin invokes immune responses that damage the cell to which it is attached, and this may be the key to any or even most auto-immune disease, and potentially many degenerative disease. 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 30 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.

It is not generally known why some individuals become sensitized to food in their diets. In an attempt to clarify this, coeliac disease has been extensively studied, since patients with this disease usually normalize when placed on a gluten free diet. Researchers reported that the mucous membranes of coeliac patients showed sugar residues which were capable of binding to the lectins in wheat germ, which resulted In a cytoxic reaction. Rats treated with Concavallin-A or wheat germ lectin developed a

gut membrane that was paradoxically impermeable to small molecules, but very permeable to large, highly allergenic molecules, a situation which is mimicked in food allergies and coeliac disease.(2)

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.

The fact that lectins appear to aggravate existing inflammatory conditions can be seen in the example of rheumatoid arthritis. The RA antibody is different structurally from a normal antibody in that the side-chain sugar, galactose, is replaced with N-acetyl glucosamine, the sugar for which the wheat germ lectin (WGA) is highly specific. This may point to why patients with rheumatoid arthritis feel better on a wheat-free diet. The defective RA antibody has also been shown to be reactive with the lectin found in the common lentil bean. According to the immunologist David Freed, “Of the various rheumatogenic foods, wheat and other grains top the list. Avoidance of these is frequently the only dietary manoeuvre required, especially in early cases.” He proposed that ingested wheat lectin (and other dietary lectins) enter the bloodstream from the intestine and bind strongly to connective tissues (which contain considerable quantities of glycoprotein) and skin proteoglycans making them stiff. It is a clinical observation that inflammation of the gut is associated with inflammation of the joints. It has been also observed that the pain and inflammation of fibromyalgia may stem from or be contributed to by intolerance to wheat lectins. In fact, lectins are capable of intensifying the effects of autoimmune disorders in general. Nightshade vegetables like potatoes and tomatoes are very high in lectins and are known to trigger the symptoms of arthritis.(5)

Wheat is one of the commonest triggers in diet-responsive rheumatoid arthritis, and wheat lectin is specific for N-acetyl glucosamine—the sugar that is normally hidden but exposed in rheumatoid arthritis. The health food trade has already seized on N-acetyl glucosamine as an anti-arthritic supplement. There is some evidence that the beneficial effect of glucosamine is caused by the blocking of the lectins at the gut wall. As the response is seen very quickly, it is worth trialling in anyone with arthritic pain, especially if you improve with the more effective forms of glucosamine. Usually a week of total restriction of lectins is enough to tell if you will respond. I have been using this restriction now very successfully in other problems as well, such as pancreatitis, Hashimotos thyroiditis and fatty liver often with remarkable success. Even osteoarthritic pain will often reduce in severity when lectin-free.

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 stimulate 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 current traditional method of treatment. 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 don’t we all get insulin dependent diabetes, rheumatoid arthritis, IgA nephropathy, and peptic ulcers? Partly because of biological variation in the glycoconjugates 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 sequelae 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.

My mentor Dr Mervyn Garrett, an Australian pioneer in food intolerance and allergy for years has described the inflammatory cascade for years, where a person may exhibit symptoms of dairy intolerance as a child such as reflux, and after things such as gut infections, stress or trauma, there will develop an increasing level of intolerance. Lectins have the capacity to cause this phenomenon. I suspect the problems may commence in utero.

The effects of dietary lectins only extend for as long as they are in the body, and the effects can be reduced by eating a variety of fruits, vegetables (rather than high amounts of one type) and foods with beneficial bacteria (e.g., fermented foods). The average Australian (developed society) diet is highly grain-based: bread, pasta, rice, cereals, etc. are everywhere, especially in processed foods. Unrefined grains are more nutritious than refined versions because they contain more nutrients. However, they also provide more lectins (and other anti-nutrients). Before the invention of modern agriculture, grains were a minor and seasonal crop. Now we can go to the supermarket market and have a wide range of whole grain pasta, breads, rice, quinoa, kamut, amaranth, oats, barley and chips.

An interesting feature of some lectins is their ability to mimic hormones. As one can imagine, this could contribute a significant impact on metabolism. The hormone insulin stores excess carbohydrates (glucose) as fat. It accomplishes this by attaching itself to the insulin receptor found on the fat cell. Under stimulation from insulin, the fat cell becomes more permeable to glucose, which would otherwise remain in circulation. With mission accomplished, the insulin hormone then disconnects to its receptor. In many people, lectins found in lentils, green peas, corn, potatoes but especially wheat germ agglutinin (WGA), are known to bind to the insulin receptor giving the fat cell the same message that insulin gives, namely to make fat. The lectin, however, due to a lack of feedback inhibition, remains indefinitely attached to the receptor giving the cell a constant message to make fat. This perhaps explains why many weight loss programs that include a moderate-to-high amount of carbohydrate (especially modern grain) fail.[5]

A peculiarity that travellers find is that they may be able to eat bread in Italy or France while react badly to bread in Australia. Is this because the wheat in Australia has been genetically modified so our threat receptors (TLRs) recognize the increased threat? People often feel they are gluten intolerant when they aren't and can tolerate other forms of gluten, eg fresh semolina pasta (which has a high gluten content), so a simple test is to try fresh pasta. Sometimes sourdough bread is tolerated as lectins have been reduced through the preparation process. It is common to start having symptoms after gut infections, probably the most common offender I see being Blastocystis hominus, which can be extremely difficult to eradicate using traditional treatments.

The "Paleo diet" is trendy and while it is not quite right, it can help sort out your intolerances if you are struggling to find food to eat or feed children. Labelling a food as "full of lectins" is quite inaccurate and the common usage of this in "Paleo diet" opens up this diet to valid criticism. Everyone has different lectins they react to more than others, and one fixed diet simply does not fit everyone's needs. It seems likely that even your blood group may determine which ones you may react to.

There does appear to be a genetic component, and it seems likely to me that this dovetails into the MTHFR gene mutation and its own set of problems as the same diseases appear in this mutation. I believe from ongoing research that having the MTHFR mutation causes collagen dysfunction which I expect would contribute to increased potential for the toxic components of the troublesome lectins to enter our body through the gut lining (which would explain the "leaky gut"). As we are learning, and discussed elsewhere, increased vitamin B12 does appear to enhance the function of the defective enzyme that causes the collagen issues, but again further research is needed to confirm this, or whether it will give any relief from symptoms.

After an initial lectin-free trial, which may only require a week, I recommend slowly adding different foods until you can work out your own problems. It needs a lot of fine-tuning, the job for Kelley my

nutritionist, as everyone has different triggers. I urge you to listen carefully to your body as you work through foods, remembering the reaction as you introduce an offending food can vary widely from arthritic-type pain to IBS symptoms, fatigue, memory impairment, FMS symptoms, bladder dysfunction and all the symptoms that may be seen as part of dysautonomia.

Over centuries we have learnt to adapt to lectins. Cooking methods that have been of help have included sprouting, and soaking overnight, then rinsing and draining does appear to remove or inactivate many of the lectins and while fermenting appears to allow beneficial bacteria to convert many of the harmful substances- eg tofu, miso, sourdough bread or beer, and although these do not completely remove the problem, it has made these food sources generally able to be tolerated.

To start, food to eliminate:

1. All grains- wheat, rye, oats, barley etc
2. Dairy- milk, cream, cheese, yoghurt, ice cream, butter, including sheep and goat
3. Nightshades- tomatoes, eggplant, capsicum, chilli, potatoes
4. Legumes, pulses, lentils, soy, peanuts. This includes kidney beans, baked beans etc

Allowed foods:

1. Carbs- sweet potato, pumpkin, quinoa, buckwheat, sprouted breads, small amounts white rice if tolerated (not brown rice)
2. All fruits – unless you have other intolerances eg to citrus
3. Vegetables- all except nightshades
4. Protein- grass fed meats, seafood, eggs, chicken, pork
5. Non-dairy milks- rice (watch quantity), oat, almond, coconut
6. Nut and nut flour- almond, cashew, walnut, macadamia, almond meals, nut butters
7. Seeds- chia, sunflower, pepita, sesame, flaxseed
8. Herbs and spices- all except chilli

Remember this restriction is a trial, and either Kelley or I will free it up by trialling for example roma tomatoes. White rice is generally tolerated better than brown and this is usually safe to add early in the trial period- but in the first week. Generally the responses will be obvious within a fortnight. Some foods, especially sweet potato, appear to protect against the lectin responses.

Diet is one part of the environment that is manipulable. 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.[1] Because lectins are so prevalent in a typical diet, undertaking a supplement regimen to help combat the damaging effects of lectins can help contribute to optimal health, improve the health of the intestinal tract and contribute to weight loss. Certain seaweeds, especially those high in the sugar frucose (Bladderwrack) and mucilaginous vegetables like sweet potato, have the ability to bind to lectins in a way that makes them unavailable to the vulnerable cells of the gut. These foods act as sacrificial decoys and attach to the problematic lectins that would ordinarily attach and bind to gut epithelial cells. I hope that we see some decent research evolve from these early studies, but most importantly that confirmation is rapidly made and with this the understanding that simple diet may control may of these auto-immune diseases.

References, and other sources to read include:

1. Dr David Freed: Do Dietary Lectins Cause Disease: BMJ 1999: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1115436/
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3. Dr Sarah Ballantyne: Are All Lectins Bad? http://www.thepaleomom.com/2014/01/lectins-bad.html
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5. Carolyn Pierini: Lectins- Their Damaging Role in Intestinal Health, Rheumatoid Arthritis and Weight Loss: http://www.vrp.com/digestive-health/digestive-health/lectins-their-damaging-role-in-intestinal-health