What Are Lectins?

Lectins are proteins that bind to specific carbohydrates. They are found in most plants, as well as in humans, microorganisms, animals, and fish. Therefore, they are found in many of the foods we eat. The most concentrated forms appear to be in edible seeds such as those found in cereal grains and legumes. The edible seeds of the legume plant (Leguminosae) may be referred to as pulses, legumes, dried beans, or beans. The term "bean" is used most often by consumers and will be used in this review. Peanuts and soybeans are in the Leguminosae family as well but will be referred to separately.

Lectin levels in plants and food crops can vary significantly depending on environmental conditions such as drought and salinity. Lectin compounds may have evolved in plants as a survival mechanism, reducing the chance the they will be consumed in large amounts.

Potential functions of lectins:

- Carbohydrate transport
- Specific cellular recognition
- Embryonic development
- Cohesion
- Binding of carbohydrates

Lectins initially drew the attention of researchers because of their ability to bind or “agglutinate” red blood cells and were defined as “hemagglutinins” though they are capable of binding a variety of cells. Researchers noted that certain lectins would bind to one type of red blood cell (e.g. type A, B, AB, or O) but not another depending on the saccharide associated with the specific blood type.

In one study of 88 different foods, 29 foods were found to agglutinate either human erythrocytes or

Glycobiology

Glycobiology is the comprehensive study of carbohydrates/saccharides and the specific proteins such as lectins that recognize/bind them.

For example,

- Wheat germ agglutinin (WGA) binds to N-acetylglucosamine and its glycosides
- Concanavalin A from jack beans binds to mannose, glucose, glycosides of mannose & glucose
- Peanut agglutinin binds to galactose and galactosides
- Red kidney bean lectin binds to N-acetylglucosamine

The Latin word legere, meaning to choose or select, is the basis for the word lectin.

The term lectin in general refers to “all sugar-specific agglutinins of nonimmune origin, irrespective of source and blood type specificity.”

Lectins may bind several cells and create “clumps” or masses of cells that become stuck together.

Lectins are considered the most widely studied molecules in glycobiology. They are capable of reversibly binding monosaccharides, oligosaccharides, and polysaccharides (glycans), including glycans found on cell walls or membranes. These interactions can be as specific as the binding of antibodies to antigens, or enzymes to target substrates. They are capable of binding multiple molecules/cells, creating a clump or mass. Due to their biological activity, some lectins may be detrimental (occurring as toxins in raw foods while others may have anti-cancer effects or act as mediators for targeted drug delivery.

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bacteria commonly found on or in humans (e.g. S. mutans, S. sanguis, S. aureus). The clinical significance of these activities has yet been well defined.\textsuperscript{18} Another study points out that greater than one-third of beans tested was found to display hemagglutinating activity. Of those displaying activity, ten percent exhibited blood type specificity.\textsuperscript{19}

Dr. Peter D'Adamo’s Eat Right 4 Your Type ®, also known as the “blood type diet” is based on the principle that certain lectins bind to certain types of red blood cells.\textsuperscript{20} The premise of this eating plan is still considered controversial due to lack of clinical research (as of April 2018) though some individuals may benefit from following such a plan.\textsuperscript{21, 22}

Cardiologist Steven Gundry, MD, FACS, FACC addresses dietary lectins in his 2017 book The Plant Paradox: The Hidden Dangers in “Healthy” Foods That Cause Disease and Weight Gain\textsuperscript{23}, linking them to a number of disorders including obesity, cardiovascular disease, allergies, autoimmune disease, and cancer. The book provides specific lists of foods to include or avoid along with recipes, meal plans, and modifications for ketogenic, vegetarian, and vegan plans.\textsuperscript{24} Dr. Gundry has been conducting research into the effects of a low-lectin diet (combined with other nutrition interventions) on risk of cardiometabolic disease, autoimmunity, and other disorders.\textsuperscript{25, 26, 27, 28, 29} Practitioners should watch closely for confirmation and conclusions that may come from that research.

A paleolithic or “paleo” eating plan also advocates eliminating lectin-containing foods.\textsuperscript{30}

At this time, some nutrition experts suggest that the benefits of eating healthy foods that contain lectins outweigh potential negative effects as long as adequate soaking, cooking, or processing is utilized to deactivate lectins.\textsuperscript{31, 32}

Bean concentrates such as Phaseolus vulgaris from white kidney bean have been commercially marketed as “starch-blockers” and weight loss aides as they essentially induce “starch malabsorption.”\textsuperscript{33, 34} While some research suggests they may have a role in weight management, a 2011 meta-analysis finds the research available on “starch blockers” to be inconclusive.\textsuperscript{35}

Ricin has been in the news when used in its purified form to poison people. Ricin is a toxic lectin derived from castor seeds but is not considered a dietary source of lectins.\textsuperscript{36} The CDC emphasizes that “castor beans are processed throughout the world to make castor oil...it would take a deliberate act to make ricin and use it to poison people.”\textsuperscript{37}
Sources of Plant Lectins

Lectins are found in most plants including wheat, corn, tomato, peanut, kidney bean, banana, pea, lentil, soybean, mushroom, rice, and potato. However, lectins are found in the greatest concentration in raw beans and grains (especially wheat), followed by dairy, seafood, and plants in the nightshade family (e.g. tomato, potato, eggplant, bell pepper). Wheat germ agglutinin (WGA) is considered the most common food lectin consumed.

Lectins exist primarily in the raw forms of foods. Heating and cooking appears to degrade most lectins, although pre-soaking beans is most effective at eliminating lectin activity. Dry heat or roasting may not be as effective at deactivating lectins.

Lectins found in various parts of the plant have distinct biological activities. Some lectins have strong anti-viral, anti-fungal, and anti-bacterial properties.

<table>
<thead>
<tr>
<th>Part</th>
<th>Biological Activities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seeds</td>
<td>Anticoagulant and antiplatelet properties; coagulant, mitogenic, antibacterial, antifungal, and antitumor activities</td>
</tr>
<tr>
<td>Bark</td>
<td>Antifungal and insecticidal activities</td>
</tr>
<tr>
<td>Heartwood</td>
<td>Termiticidal activity</td>
</tr>
<tr>
<td>Stem</td>
<td>Antiviral and apoptosis-inducing activities</td>
</tr>
<tr>
<td>Leaves</td>
<td>Antiviral, antibacterial and antifungal activities</td>
</tr>
<tr>
<td>Fruits</td>
<td>Mitogenic and antiviral activities</td>
</tr>
<tr>
<td>Roots</td>
<td>Antifungal and termicidal activities</td>
</tr>
<tr>
<td>Tubers</td>
<td>Insecticidal and antitumor activities</td>
</tr>
<tr>
<td>Bulbs</td>
<td>Proteolytic activities</td>
</tr>
<tr>
<td>Rhizomes</td>
<td>Antiproliferative, immuno-stimulatory, antiviral, antifungal, antitumor and apoptosis-inducing activities</td>
</tr>
</tbody>
</table>

Reducing Lectin Content

Lectins may be deactivated using different methods including dry roasting, toasting, autoclaving, microwaving, and infrared heat. However, lectins in some foods such as beans may be heat stable and are best deactivated via aqueous heat. This method requires initial soaking in water for at least 8 hours followed by cooking in water at high heat (212’ Fahrenheit/100’ Celsius) for at least 10 minutes or at 203’F/95’ Celsius for 60 minutes. This method may not be effective with other lectins such as wheat germ agglutinin, gluten-associated lectins, and taro tuber lectins and further prolonged heating may be required. Lectins may also be resistant to breakdown by digestive enzymes though appropriate heat treatment may denature them enough to increase their digestibility.

Slow cooking may not get hot enough and may not deactivate kidney bean lectins. It is best to soak kidney beans (and other legumes) for at least 5 hours, discard soaking water, and boil the beans for at least 30 minutes.

Sprouting or fermenting foods that are high in lectins in their raw state may also effectively reduce lectin activity. Lectin content may be reduced by 59% by sprouting (e.g. soybeans) and may be reduced by up to 95% by fermenting as is the case with tempeh, a fermented soybean food. History suggests that traditionally prepared grains were first fermented or sprouted, in effect reducing their lectin content.
Recommend aqueous heat to deactivate or destroy legume bean lectins:

- Soak in water for at least 8 hours, up to 16 hours at 68°F/20°C, discard water
- Cook in water at high heat (212° Fahrenheit/100° Celsius) for at least 10 minutes or at 203°F/95° Celsius for 60 minutes.
- Discard any beans that remain hard as lectins may not be adequately destroyed

Wheat germ agglutinin, gluten-associated lectins, and taro tuber lectins require prolonged heating beyond that used for beans.  

Cooking and processing appears to minimize or eliminate lectin content in wheat-based products as well. Some products such as whole wheat pasta may be exposed to adequate heat during process which may eliminate lectins completely.

Lectins can be blocked by certain carbohydrates such as simple sugars or oligosaccharides such as N-acetylglucosamine (blocks wheat lectin). One animal study using the endogenous lectin galectin-1 downregulated the autoimmune response to beta cells and even reverse hyperglycemia in mice with type 1 diabetes.

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### Amount of active WGA in wheat-derived products.

<table>
<thead>
<tr>
<th>Wheat Derived Products</th>
<th>WGA µg/g (±SD)</th>
<th>Reference Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheat germ</td>
<td>300 (±35)</td>
<td>Vincenzi et al., 2000 [42]</td>
</tr>
<tr>
<td>Wheat germ</td>
<td>100–500</td>
<td>Peumans and Van Damme, 1996 [39]</td>
</tr>
<tr>
<td>Semolina a</td>
<td>4.0 (±1.0)–10.7 (±1.5)</td>
<td>Matucci et al., 2004 [43]</td>
</tr>
<tr>
<td>Flour a</td>
<td>4.3 (±0.7)–4.4 (±1.0)</td>
<td></td>
</tr>
<tr>
<td>Wholemeal flour a</td>
<td>29.5 (±2.5)–50 (±5.5)</td>
<td></td>
</tr>
<tr>
<td>Pasta a</td>
<td>≤0.4 (±0.2)–3.2 (±0.2)</td>
<td></td>
</tr>
<tr>
<td>Pasta cooked a</td>
<td>≤0.3 (±0.2)</td>
<td></td>
</tr>
<tr>
<td>Wholemeal pasta (enriched with wheat germ)</td>
<td>40 (±2.7)</td>
<td></td>
</tr>
<tr>
<td>Wholemeal pasta cooked (enriched with wheat germ)</td>
<td>Not detectable</td>
<td></td>
</tr>
<tr>
<td>Wholemeal pasta a</td>
<td>0–5.7 (±0.2)</td>
<td></td>
</tr>
<tr>
<td>Wholemeal pasta cooked a</td>
<td>Not detectable</td>
<td></td>
</tr>
<tr>
<td>Breakfast cereals a</td>
<td>13–53</td>
<td>Ortega-Barría et al., 1994 [41]</td>
</tr>
</tbody>
</table>

*a Values are obtained from more than one product and from different manufacturers.

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Fig. 26-1. 
[https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3705319/](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3705319/)  
Creative Commons license [https://creativecommons.org/licenses/by/3.0/](https://creativecommons.org/licenses/by/3.0/)
Interesting animal research demonstrated that co-administration of sucrose with raw kidney bean lectin significantly reduced the lectin’s toxic effects including increased intestinal permeability and bacterial translocation.\textsuperscript{58} Ongoing research is exploring the potential benefit of administering certain sugars and oligosaccharides concurrently with lectin-containing foods to help deactivate the lectin component.

Supplementation with specific carbohydrates may affect lectin activity as well. Commercial dietary supplements have been formulated with a variety of ingredients designed to either block lectin binding, enhance digestion, or help support gastrointestinal microbiota.

- N-Acetyl Glucosamine
- Bladderwrack (Fucus vesiculosus)
- Okra fruit
- D-Mannose
- Mucin
- Sodium Alginate
- Pepsin
- Larch Arabinogalactans
- Methylsulfonylmethane (MSM)
- Vegetable Peptase

Researchers focused on genetic engineering have proposed transferring lectin genes into food crops due to their insecticidal properties. However, many scientists warn against this practice due to potential negative health effects on humans.\textsuperscript{59, 60, 61, 62} Unfortunately, scientists expressing concerns associated with engineering lectins into foods have been professionally silenced in the past.\textsuperscript{63}

Lectins are not always deactivated or degraded by heat or digestion.

Lectins that appeared to resist proteolytic digestion included those in wheat germ (wheat germ agglutinin or WGA), tomatoes, and navy beans although these foods weren’t found specifically to bind human erythrocytes. Lectins in wheat germ, peanuts, and dry cereals do appear to possess hemagglutinating activity.\textsuperscript{64}

One study revealed that peanut lectin was detected in blood samples after individuals consumed either raw or roasted peanuts, suggesting that digestive activity and heat by roasting do not always destroy lectins, particularly those in peanuts.\textsuperscript{65} Cell research hypothesizes that circulating peanut agglutinin (PNA) may have the ability to mimic endogenous galactin-3 and promote cancer cell metastasis. However, considering the study used high doses of PNA applied directly to cancer cells, the research may not be relevant to humans, though the premise does warrant further monitoring.\textsuperscript{66}

On the other hand, peanut agglutinin may play a role in detecting cancer cells through its binding capabilities. One study points out that PNA was found to bind to colon mucins (glycoprotein constituents of mucus) in cancerous but not normal cancer cells.\textsuperscript{67} It appears the tendency of PNA to bind GI cancer cells is being employed in the area of cancer diagnostics.\textsuperscript{68} Lectin histochemistry may also be used in a diagnostic capacity to help reveal dysplastic changes in gastric mucosa.\textsuperscript{69}

Most research suggests that lectins can be “deactivated” or degraded by adequately cooking and/or soaking and cooking foods high in lectins.\textsuperscript{70} Plant lectins are found to be more resistant to breakdown by heat and digestion than animal-derived lectins.\textsuperscript{71} Concerns have been raised regarding ingestion of lectins because of their ability to bind biological molecules that contain carbohydrate moieties (e.g. red blood cells, mucosal cells). Because some lectins are resistant to breakdown, some researchers recommend minimizing their intake all together.\textsuperscript{72}
What health benefits or concerns are associated with lectins?

Lectins in their active state have the potential to disrupt cell physiology and function and may be referred to as “anti-nutrients.” They must be deactivated prior to consumption or degraded by digestion to be consumed safely. Lectins may be endocytosed (engulfed) by gut epithelial cells which may trigger detrimental effects due to their binding to the wall of the intestine. According to a toxicology review in the early 2000s, lectins in their active, binding state have the potential to

- Bind to membrane glycosyl groups of cells lining the digestive tract
- Affect turnover and loss of gut epithelial cells
- Damage the luminal membranes of the epithelium
- Interfere with nutrient digestion and absorption
- Stimulate shifts in GI bacterial flora
- Modulate the immune state of the digestive tract
- Systematically disrupt lipid, carbohydrate, and protein metabolism
- Promote enlargement and/or atrophy of key internal organs and tissues
- Alter hormonal and immunological status
- May threaten growth and health of animals consuming them in large amounts
- Are detrimental to numerous insect pests of crop plants

Remember these detrimental effects refer to active lectins that are not broken down or degraded.

Adverse Reactions to Lectins

It is still not clear exactly what the health risks of lectins in the food supply are or what their exact effects on human cells may be.

Acute reactions to lectin consumption (e.g. undercooked kidney beans) include nausea, vomiting, diarrhea, and general gastroenteritis.

Researchers propose that lectins interfere with repair mechanisms at the level of the gut epithelial cells, leading to observed gastrointestinal symptoms.

Potential gut-specific adverse effects of lectins if eaten in large quantities

- Affect turnover and loss of epithelial cells
- Damage luminal membranes of the epithelium
- Interfere with digestive/absorptive activities
- Acute nausea, vomiting, diarrhea
- Stimulate shifts in bacterial flora
- Modulate the immune state of the digestive tract
- Interfere with gut hormone secretion
- Serve as potent growth factors for the small intestine (e.g. hyperplasia, hypertrophy)
- Some effects may be beneficial though human research is scarce

Some suspect that reported adverse reactions to foods in the nightshade family (e.g. tomatoes, potatoes, eggplant, bell pepper) may be due to the presence of lectins.

Some research has extrapolated lectins’ affinity for specific carbohydrates to corresponding tissue, suggesting the potential for related adverse reactions.
### Lectins/Agglutinins with affinity to specific tissues.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Wheat Germ Agglutinin</th>
<th>Soybean Agglutinin</th>
<th>Peanut Agglutinin</th>
<th>Lentil Lectin</th>
<th>Pea Lectin</th>
<th>Bean Agglutinins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Buccal mucosa</td>
<td>*</td>
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<td>*</td>
<td>*</td>
<td></td>
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<tr>
<td>Stomach</td>
<td></td>
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<td></td>
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<tr>
<td>Parietal cells</td>
<td></td>
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<td>*</td>
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<tr>
<td>Intestinal brush border</td>
<td>*</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Colonic mucosa</td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
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<tr>
<td>Connective tissue</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
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<td>*</td>
</tr>
<tr>
<td>Thyroid</td>
<td></td>
<td>*</td>
<td></td>
<td>*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cartilage</td>
<td></td>
<td>*</td>
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<td></td>
<td></td>
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<tr>
<td>Liver</td>
<td>*</td>
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<tr>
<td>Pancreas</td>
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<tr>
<td>Kidney</td>
<td></td>
<td></td>
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<td>*</td>
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<tr>
<td>Prostate</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skeletal muscle</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Cardiac muscle</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Breast</td>
<td>*</td>
<td>*</td>
<td></td>
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<td></td>
<td>*</td>
</tr>
<tr>
<td>Pituitary</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>Eye</td>
<td>*</td>
<td>*</td>
<td></td>
<td>*</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Brain (myelin)</td>
<td></td>
<td></td>
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<td>*</td>
<td>*</td>
</tr>
</tbody>
</table>

*Fig. 26-2.*  
WGA, wheat germ agglutinin; SBA, soybean agglutinin; PNA, peanut agglutinin; LA, lentil agglutinin; MA, mushroom agglutinin; TA, tomato agglutinin; PA, pea agglutinin; POT.A, potato agglutinin; KBA, kidney bean agglutinin; JBA, jack bean agglutinin.  
**Lectins and Disease**

A causative link between dietary lectin consumption and specific diseases has not been established except for the known toxicity caused by consuming uncooked or undercooked kidney beans and possibly other dried beans.

Potential concerns include:

- Anemia
- Autoimmune reactions
- Binding of erythrocytes and lymphocytes
- Disruption of GI microbiota
- Immune reactivity
- Individual susceptibility due to enzyme deficiency (e.g. G6PD deficiency)
- Inflammatory response
- Intestinal damage with resulting nutrient deficiencies
- Tissue binding and disruption

Animal research has demonstrated that effects of lectin consumption on the small intestine and stomach included:

- Stripping of mucous coat
- Overgrowth of abnormal bacteria and protozoa
- Increased release of gastric histamine
- Theoretical increased risk of H. pylori infection

**What does the science say?**

Extensive, conclusive research is not yet available to determine the exact effects of dietary lectins within the body and whether they cause chronic inflammation, autoimmune disorders, gastrointestinal damage, cardiometabolic disease, or other chronic diseases. Early research suggests that lectins may indeed influence these conditions if not completely deactivated or destroyed prior to consumption.

Research and observation in the 1990s notes that wheat gliadin is a “lectin-like” substance and its binding to GI mucosa may contribute to celiac pathophysiology. Research also revealed that wheat lectin binds to tissues in the human kidney (glomerular capillary walls, mesangial cells, and tubules), and binds IgA antibodies in rodents. Researchers surmise that human IgA nephropathy may be linked to wheat lectin and noted that elimination of wheat gluten in children with this disease resulted in a decrease in proteinuria and immune complex levels.

Current research published in 2017 suggests that plant lectins represent “danger molecules” and are capable of activating the NLRP3 inflammasome, promoting inflammation and mitochondrial damage. This evolving research should help define any potential role that dietary lectins may play in disorders such as inflammatory bowel disease, insulin-dependent diabetes, rheumatoid arthritis, and food allergy, intolerance, and sensitivity.

The innate immune system recognizes “danger molecules” and mounts an inflammatory immune response to them via protein complexes known as inflammasomes. The inflammasome is then responsible for the release of proinflammatory cytokines which in turn may underlie common inflammatory disorders including Alzheimer’s, arthritis, atherosclerosis, gout, and diabetes. Developing research suggests ingestion of plant lectins may be a trigger for such inflammatory diseases.
Increased Intestinal Permeability

Research demonstrates that WGA can increase intestinal permeability. Researchers theorize that an increase in intestinal permeability will increase passage of potentially harmful compounds such as bacterial toxins or lectins themselves may trigger systemic inflammation and other adverse effects.

Autoimmunity

Some researchers suggest that the cascade of events following increased intestinal permeability may be directly related to autoimmune disorders if harmful antibodies are formed and react or cross react with tissues in the body. Increased intestinal permeability caused by WGA may contribute to the pathophysiology of celiac disease. It was observed that celiac patients had higher antibodies to WGA than healthy individuals did.

The proposed link between lectins and autoimmune reactions may lie in stimulation of class II HLA antigens in tissues such as thyroid and pancreatic islet cells. Research suggests that cytotoxic antibodies associated with insulin-dependent diabetes bind a disaccharide (N-acetyl lactosamine) that also binds tomato, potato, wheat, and peanut lectins, possibly contributing to autoimmune attack of the islet cells.

A link to rheumatoid arthritis may be related to an abnormal IgG molecule that exposes N-acetylglucosamine to which certain lectins can bind (e.g. wheat lectin). Indeed, wheat ingestion appears to be a trigger for some with rheumatoid arthritis. Provision of exogenous N-acetylglucosamine may help to block lectin interaction and reduce symptoms.

Individual differences may determine whether exposure to lectins leads to adverse effects. For example, lectins may only affect and cause rheumatoid symptoms in those with a genetic propensity for rheumatoid arthritis.
Some of the most intriguing human subjects research on lectins appears to be done by cardiologist Dr. Steven R. Gundry who has conducted trials and published abstracts in various cardiology journals. Although the study protocols incorporated different variables including specific nutritional supplementation, incorporation of anti-inflammatory foods, and provision of pre- and probiotics, Dr. Gundry emphasizes that restriction of lectins is a cornerstone underlying observed benefits.

One study looked at the premise that autoimmunity is related to dysbiosis, increased intestinal permeability, and lectin exposure. The study included 102 patients with markers of epithelial inflammation, autoimmune disease activity, and signs/symptoms of autoimmune diseases including inflammatory bowel disease, rheumatoid arthritis, Sjogren's, and scleroderma. Biomarkers included adiponectin, hs-CRP, TNF-alpha, IL-6, fibrinogen, and myeloperoxidase.

The study protocol eliminated major dietary lectins (all grains, pseudo grains, beans/legumes, peanuts, cashews, nightshades, squashes, and casein A1 cow's milk products) and supplemented with prebiotics, probiotics, and polyphenols. Within 9 months, autoimmune and inflammatory markers were completely resolved in 95 of 102 patients and demonstrated improvement in biomarkers in the remaining 7 patients.104

Another study looked at implementation of a lectin-restricted diet combined with targeted nutrition supplementation in 800 patients with known coronary artery disease. The study protocol included restriction of high-lectin foods (grains, legumes/beans, nightshades, seeded vegetables, casein A1 milk), fruits, and commercial poultry. Patients with an Apo E genotype were instructed to eliminate animal fats and cheeses. Intake was supplemented with one liter/week of olive oil, large amounts of green vegetables, polyphenol-rich coffee or tea, 1 ounce/day dark chocolate, 4000 mg high DHA fish oil, 200 mg grape seed extract, and 25 mg pycnogenol daily.

Results were compared to an average 30-40% new event rate per 5-year period in patients following standard treatment protocols (low-fat/cholesterol diet, exercise, lipid-lowering medications). Patients on the study protocol were followed for an average of 4.5 years during which only 0.5% received a new stent; no patients had an MI or unstable angina, and Corus® scores decreased significantly in 92% of patients. (Corus measures likelihood of having obstructive coronary artery disease.)105

**Frequently Asked Questions**

**Should everyone avoid lectins?**

It is certainly important to avoid lectins in their active form (mostly in the raw form of foods that are naturally high in lectins). However, the question of whether it's detrimental to consume those foods after they have been soaked, cooked, or processed would depend on if lectins were adequately deactivated or destroyed. It is possible that some individuals may be more sensitive to lectins or more prone to negative effects from their inadvertent ingestion.

**Who would benefit from a low-lectin diet?**

Even though cooking and processing is found to destroy/deactivate most lectins, a low-lectin trial should remove those foods naturally high in lectins to avoid unintentional ingestion.

A low-lectin approach would ideally eliminate those foods highest in lectins during the trial phase as it would be nearly impossible to avoid all lectins in all foods. Individuals suspected of being sensitive to lectins may be able to tolerate incorporation of those foods once they have been treated adequately with soaking, boiling, fermenting, or sprouting.

**Can we remove lectins from the foods we eat?**

Foods that contain lectins in their raw state can still be considered healthy foods as long as they are heated, fermented, or processed enough to remove potentially harmful lectins. Nutrition professionals give the “go ahead” on these commonly consumed foods (as long as lectins are adequately eliminated):106

- Peanuts (difficult to eliminate lectins but health benefits believed to outweigh lectin risk)
Potatoes (although up to 50% of lectins may remain following heating, no definitive research demonstrates negative effects from consuming potato lectins)

- Red kidney beans and other dried beans (thoroughly soaked and cooked)
- Soybeans (must be fermented (e.g. soy sauce, miso, tempeh), sprouted, or boiled at 212°F for at least 10 minutes)
- Tomatoes
- Wheat/wheat germ (adequately cooked at 149°F or above during cooking or processing)

Is there testing available for lectin sensitivity?

Though there are no specific tests for lectin sensitivity, eliminating and then reintroducing foods highest in lectins may help determine if an individual is sensitive. Testing immune reactions to foods that contain lectins may also help narrow down which foods an individual may truly be sensitive to.

Do all healthcare practitioners agree on the effects of lectins on the body?

Here we have more confusion as not all nutrition professionals or healthcare practitioners agree on the role that lectins may play in health and disease. Some physicians such as Steven Gundry, M.D. express concerns and feel confident that lectins in foods are associated with an increased risk of disease and should be eliminated by most. At the opposite end of the spectrum, Michael Greger, M.D., refutes and actually dismisses Dr. Gundry’s premise. Dr. Greger emphasizes that a number of studies associate consumption of foods such as dried beans and whole grains with a lower risk of chronic disease.107 (Note that dried beans and grains are always consumed cooked so lectin levels in these studies are likely low.)

What questions remain?

Do well-cooked foods still contain lectins?

There appears to be a possibility that some lectins resist heating and digestion and may enter the bloodstream. It is unclear whether this level of lectin exposure poses any health risks.

Do lectins definitively cause disease?

The question of whether lectins cause disease in humans continues to be researched and debated. Unfortunately, there are no large scale clinical trials to fully answer that question. Researchers are proposing a variety of hypotheses related to the effects of lectins at the cellular and tissue level.

The Bottom Line

It can be hard to sort through what is conclusive research and what is hyperbole when it comes to what effects lectins may have on human physiology.

In general, it is accepted that lectins in their active form can bind carbohydrate moieties on human cells and cause agglutination. It is also accepted that if lectins are degraded or deactivated (proper cooking or via digestion) they will not be able to cause adverse reactions.

Until further research is done to specifically demonstrate that ingestion of dietary lectins definitively causes inflammation, autoimmunity, or gastrointestinal damage, individual sensitivity to lectins may need to be explored on a trial/challenge basis.

Small amounts of lectins may be tolerated by some individuals. However, it is prudent to soak, heat, or treat high-lectin foods adequately to minimize lectin activity. Using a pressure cooker may also reduce lectin activity.

Individuals who do suffer from chronic inflammation, gastrointestinal disorders, or autoimmune disease may benefit from elimination of foods high in lectins. A controlled reintroduction should help determine if lectin-containing foods trigger symptoms.

It is important to note that many individuals consume grains and legumes (high lectin foods) on a regular basis (e.g. the Mediterranean diet) and don’t appear to suffer detrimental effects. However, dried beans are most often soaked and boiled and grains are usually consumed after cooking at fairly high temperature so it’s possible that lectin levels are reduced in those foods. Individual sensitivity to lectins may depend on genetic makeup or other biochemical or physiological differences.
It is essential that high-lectin foods are soaked, cooked, heated or processed adequately to eliminate or minimize lectin activity.

Consider trial of supplements when consuming lectin-containing foods:

- N-Acetyl Glucosamine
- Bladderwrack (Fucus vesiculosus)
- Okra fruit
- D-Mannose
- Mucin
- Sodium Alginate
- Pepsin
- Larch Arabinogalactans
- Methylsulfonylmethane (MSM)
- Vegetable Peptase

Trial elimination of high-lectin foods may be most beneficial for

- Individuals reporting adverse or negative effects when they eat foods high in lectins
- Food sensitivities
- Gastrointestinal disorders
- Increased intestinal permeability
- Rheumatoid arthritis
- Autoimmune disorders
- Celiac disease

Eliminate (in general)\(^\text{108}\)

- Foods in the Leguminosae family (e.g. dried beans, peanuts, soybeans)
- Nightshade family foods (e.g. eggplant, potatoes, tomatoes, bell peppers)
- Grains, especially wheat and wheat germ
- Dairy products (A2 milk may theoretically be tolerated)

Ensure adequate fiber intake while following a low-lectin plan by incorporating an abundance of low-lectin vegetables and fruits. A psyllium supplement may be beneficial.

The most detailed (and restrictive) guides and shopping lists for a low-lectin plan appear to be from Dr. Gundry and can be accessed at GundryMD.com including:


Shopping list [https://gundrymd.com/plant-paradox-shopping-list/](https://gundrymd.com/plant-paradox-shopping-list/)

PDF of shopping list: [https://gundrymd.com/wp-content/pdf/Plant-Paradox-Shopping-List.pdf](https://gundrymd.com/wp-content/pdf/Plant-Paradox-Shopping-List.pdf)

**Takeaways**

**Foundational Resources**

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