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One Reader at a Time...

FREE

# PUBLIC HEALTH ALERT

Investigating Lyme Disease & Chronic Illness in the U.S.A.

## Immune Health & the GI Tract- Part 1

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The immune system of each individual must ultimately assume the management of infections like Lyme disease. In my experience, antibiotics level the playing field by reducing the abundance of bacteria, leaving the work of sustained recovery to our own immune system. Without the body's own immune vigilance there is a slow progression to health.

Our understanding of the immune system is ever evolving and the most recent 2010 research casts the spot light on the impact of gastrointestinal health on the ability of the immune system to function effectively.

In this Part One of a three-part series we will explore the most recent understanding of the enormous role the gut plays in the health of the immune system. Part Two will discuss evaluation and testing of immune dysfunction caused by the GI tract. Part Three will be putting it all together discussing how to strengthen the immune system.

### Immune Triggers

There are approximately 100 Trillion bacteria in

the human gut. Astoundingly, this represents 10 times more cells than what makes up the body. The presence of these bacteria has an immunological effect on the rest of the body. Under most circumstances, this immunological effect is greatly beneficial. However, disruption of this normal flora, if not tolerated, is inflammatory and can be significantly harmful.

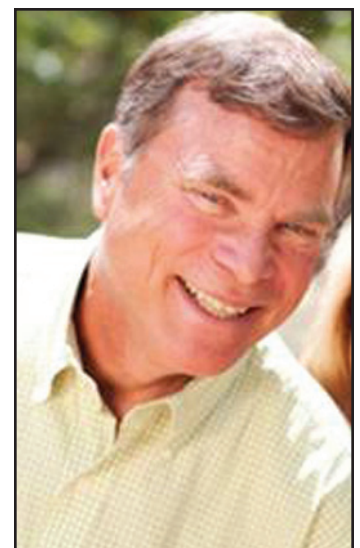
Intestinal bacterial disruption and inflammatory conditions are seen in the association of Klebsiella with ankylosing spondylitis; Citrobacter and Klebsiella with rheumatoid arthritis; Yersinia with thyroiditis; Escherichia coli and Proteus with autoimmune disorders. These examples may not reflect a direct cause by the bacteria, but rather molecular mimicry taking hold and eliciting an autoimmune response.

In addition to disruption of the normal flora, diet is the other major contributor to overall health of the GI tract. Diet has a direct response of an important component of the immune system within the "gut associated lymphoid tissue," or GALT. The GALT is a chain of lymph glands attached to the intestines. These lymph glands are very

similar to the lymph glands located at the neck under the jaw. When one has a throat infection, these glands become swollen and inflamed - as when the gut has inflammation, the GALT becomes swollen and inflamed.

Food sensitivities have a direct insult on the GALT. The response to this assault is more complex than just formation of antibodies. Thus, the measuring of the typical immunoglobulin titers for food allergies does not suffice in uncovering the source of this inflammatory component.

Most foods contain lectins, specialized proteins, which may be inflammatory for that individual without causing the classic antibody response. Studied food groups which include high levels of lectins are grains, 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. Other inflammatory triggers of the immune response are pathogen associated molecular patterns (PAMPs-typical structures of non-vertebrate



pathogens); damage associated molecular patterns (DAMPs- intracellular components); advanced glycation end products (AGES- produced when food is fried, grilled, dried, smoked or pasteurized); free radicals; trauma; and toxins.

The culmination of these inflammatory triggers occurs within the GI tract leads to a measurable systemic inflammatory response. The integrity of the cellular lining of the gut, epithelium mucosa, changes because of this inflammation leading to what is known as leaky gut syndrome. Leaky gut syndrome is the central antecedent of a wide range of disorders associated with

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chronic inflammatory condition.

This is best described by Dr Alessio Fasano in the article, "Mechanisms of Disease: The role of intestinal barrier function in the pathogenesis of gastrointestinal autoimmune disease." He states, "Together with the gut-associated lymphoid tissue and the neuro-endocrine network, the intestinal epithelial barrier, with its intercellular tight junctions, controls the equilibrium between tolerance and immunity to non-self-antigens. When the finely tuned trafficking of macromolecules is dysregulated in genetically susceptible individuals, both intestinal and extra-intestinal autoimmune disorders can occur. This review is timely given the increased interest in the role of leaky gut in the pathogenesis of gastrointestinal diseases and the advent of novel treatment strategies, such as probiotics." (Nature Clinical Practice Gastroenterology and Hepatology, Sept. 2005, Vol 2 No 9).

## The Immune System

Briefly, the body's immune system is divided into the innate and adaptive response. The innate response is usually within 0-96 hours. It is divided into 2 groups. One group is non-induced and/or nonspecific. The second group is broadly specific.

This broadly specific group contains a large list of immune responders including macrophages, mast cells, cytokines, complement system, polymorphonuclear leukocyte (PMN), antimicrobial peptide, natural killer cells and dendritic cells.

Current research

shows that if there is a modulator of the innate and adaptive immune system, it would be the dendritic cells.

Dendritic cells are throughout the entire body. Dendritic cells "sample" everything and determine if it is a friendly or a danger-stranger to the body. This sampling sets up a priming of the dendritic cells, which will then communicate with the remainder of the cells of the immune system, and respond accordingly.

The adaptive response system is usually activated within 4-5 days. The adapted system characteristically gives a sustained response to the offending agent or establishes antibodies to react to and prevent reinfection. It is a highly evolved specific system which specializes in effectors T and B lymphocytes cells. The information which the T and B cell lymphocytes use to differentiate is derived from the dendritic cell.

The B cell response is known as the humoral (circulating in our bodily fluids) mediated system. It is in this system where the antibodies for specific protein or infection are formed. The T-cell lymphocyte response is also known as the cell-mediated system. The T-cell lymphocytes can have many different responses, including triggering a B-cell response.

The dendritic cell is primed for response by the previously described triggers. This dendritic priming will derive differing sets of T-cell instructions resulting in conversion of naïve Th0 cells into Th1, Th2, Th17 and T-regulatory cells.

Depending on the signaling, a pathway can be redirected to express a healthier response.

This is most dramatic for the person with significant allergies and an underlying infection impacting the efficacy of the immune system to handle the infection. By modulating or balancing the pathways, an excessive response and its draining complications is decreased, thus allowing the immune system to focus on the more critical problem at the time.

Th1 cells produce cytokines that are involved in many types of cell-mediated immunity and delayed hypersensitivity response. It does this by increasing cell-mediated activation macrophages and neutrophils. The more predominant diseases seen with Th1 are rheumatoid arthritis, multiple sclerosis, thyroiditis, Lyme arthritis and Crohn's disease.

Th2 cells produce cytokines which are involved with further B cell activation and consequent allergy antibody response. It does this with the recruitment of mast cells, basophils and eosinophils. The more predominant diseases seen with Th2 are allergic diseases, asthma, contact dermatitis, scleroderma, ulcerative colitis and systemic lupus erythematosus.

Th17 cells mediate mucosal immunity to GI and pulmonary pathogens (especially Candida and gram-negative). This is a primary driver of chronic inflammation. It is the predominant driver to initiate inflammation of tissues and organs in Lyme arthritis, allergy, tumorigenesis, transplant rejection and autoimmune diseases (multiple sclerosis, Crohn's disease, ulcerative colitis, systemic lupus erythematosus, rheumatoid arthritis, scleroderma, ankylosing spondylitis).

As mentioned earlier, the gastrointestinal tract is full of potential immune triggers. For the most part, the immune system is able to recognize and tolerate non-harmful triggers. It does this by the means of the innate immune response. It is when there is a trigger (such as a harmful bacteria or fungus) causing the inflammatory response that cannot be completely removed, resulting in continued inflammation. If this continued inflammatory response is not switched off, then there is a move to the next level of chronic inflammation.

This next level of chronic inflammation has many facets. It includes leaky gut syndrome and dysregulation of the immune system's inflammatory profile. One can also develop an autoimmune response where there is a decrease in self recognition; a continued imbalance in the TH1/TH2/TH17 lymphocytes; and depletion of the natural killer cells, which could lead to an increase in chronic disease and cancer.

Most chronic diseases have been linked to excessive or persistent inflammation. This chronic inflammation is a systemic phenomena with local manifestations. Chronic inflammation occurs when the injury is ongoing or a predisposed immune system fails at counter-regulation. Unaddressed chronic inflammation can completely obstruct the path to recovery from an infection such as Lyme disease. In order to properly manage Lyme disease the immune system needs to be optimized. The first area of optimization is the GI tract.

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