Treatment Approach for Autoimmune Disease

Donna Sigmond

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Abstract

Autoimmune (AI) disease is a multifactorial condition involving genetic, environmental and infectious causes that turns the body against itself. In Western medicine the type of immune mechanism is involved in making an AI diagnosis. A general marker for AI disease is antinuclear antibody (ANA); and more specific markers would be TPO for hashimoto's; and anti-CCP, AKA or IgMRF in discriminating the rheumatic class of diseases. In Chinese medicine the root cause of AI diseases is yin deficiency. Yin deficiency can lead to other patterns complicating the presentation of the disease. Lifestyle, diet, genetic and environmental factors can impact AI disease. I propose that a multifactorial solution could be the best approach to a multifactorial problem as AI disease.

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The immune system is a Western medical concept. It is a complex system designed to protect the body from foreign invaders like bacteria and viruses. However, the immune system can get overactive and begin identifying self as a foreign invader and that condition is termed autoimmune disease. The body goes rogue and begins identifying its own tissues and/or organs as something that should not be there and can be quite disabling. Physicians and scientists have identified more than 80 clinically distinct autoimmune diseases and any part of the body can be involved ("Autoimmune diseases", 2017). Western medical treatment for AI disease are anti-inflammatories like non-steroidal anti-inflammatories and prednisone and/or immune-suppressants like Humira, Enbrel and Betaseron. These medications can be quite strong but they are not without risk or side effects. Unfortunately lifestyle is not often discussed with the patient.

Reducing inflammation and lessening the immune response in AI disease expression is an area of much research. There are many studies pointing to the role of inflammation as an AI trigger. Such as, the importance of eicosopentanoic acid (EPA) and docoxahexanoic acid (DHA), in the modulation of the prostaglandin E2 pathway and AI diseases characterized by a high IL-1 pro-inflammatory effect in IBD and SLE (Simopoulos, 2002). High levels of IL-6 have been implicated in several AI diseases like RA and psoriasis (Ishihara & Hirano, 2002, pp. 357-368). Dysregulation of IL-10 is associated with enhanced immunopathology in response to infection as well as increased risk for development of many AI diseases (Iyer & Cheng, 2012, pp. 23-63). IL-1, IL-6 and IL-10 are examples here but there are certainly other biomarkers used to measure the impact on expression of AI disease. Other predictive biomarkers include TH-17, IL-23, IFN-y, TNF-a, procalcitonin as there are many more.

Vojdani (2014) discusses the multiple environmental factors as AI triggers.

> New technology, new industries, new inventions, new chemicals and drugs, and new foods and diets are constantly and rapidly being introduced in this fast-paced ever-changing world. Toxicants, infections, epitope spreading, dysfunctions of immune homeostasis, and dietary components can all have an impact on the body's delicate immune recognition system. Although the precise etiology and pathogenesis of many autoimmune diseases are still unknown, it would appear from the collated studies that there are common mechanisms in the immune-pathogenesis of multiple autoimmune reactivities (p.1).

Molecular mimicry is one contributing factor of how exposure to environment confuses the immune system. For example a kiwi when consumed in some individuals can trigger an allergic response in someone with heightened latex allergies. Basically, when immune cells are reacting to a certain extrinsic antigen and then begins to react to a structurally similar self-antigens (Jörg, et al., 2016).

Diet and bio-actives from diet can have a significant impact on various immune mechanisms and AI disease. Foods high in omega-3 fatty acids like fish, walnuts and flax are great sources of EPA and DHA. The American Heart Association general recommendations are up to 3 grams a day for most healthy people focusing on improving cholesterol and triglycerides, both considered inflammatory processes (Kuller, 2018). Just as bio-actives can have an anti-inflammatory and/or modulatory effect on immune system, there is evidence that food additives can trigger the immune system negatively. Lerner and Matthias (2015) looked at changes in tight junction permeability from industrial food additives and the impact on AI. Their conclusion is that food additives studied—glucose, salt, emulsifiers, organic solvents, gluten,

mTG (microbial transglutaminase), and nanoparticles— increase intestinal permeability by bringing about tight junction paracellular transfer (p.8). This permeability fuels an immune response through the gut associated lymphatic tissue, the vehicle of which the diet impacts AI disease.

University of Southern California biochemist and researcher Valter Longo has done some interesting research on fasting and fasting mimicking diets that suggest fasting increases cortisone that may initiate a killing of AI cells. Fasting for 12-14 hours, essentially overnight, has been beneficial in some patient populations in reducing inflammatory cytokines in the joints. And a small 14 person study on fasting on patients with RA demonstrated that disease activity decreased. The conclusion of the study found a reduced ability to generate cytotoxins, reduced release of lysozyme, and a reduced leukotriene formation from RA neutrophils, together with an altered fatty acid composition of membrane phospholipids may be the anti-inflammatory mechanism as a result of fasting. (Hafström, Ringertz, Gyllenhammar, Palmblad, & Harms-Ringdahl, 2005).

Genetics help to better understand the disease expression in an individual. AI diseases are complex and have both genetic and non-genetic factors. Genome-wide association studies have shown that some 50% of the genetic risk factors for individual AI diseases overlap between different diseases. Thus, shared risk factors may converge to pathways that, when perturbed by genetic variation, predispose to autoimmunity in general (Jonkers & Wijmeng, 2017). Genes such as human leukocyte antigen (HLA), Galactside 2-alpha-L-fucosyltransferase 2 (FUT2) and Vitamin D Receptor (VDR) that are involved in many AI disease may be the cross-over genes impacting AI expression.

Biomarkers of disease risk in unaffected individuals would allow clinical trials of preventive approaches. This has been done effectively in type 1 diabetes prevention studies. For example, the recognition that relatives of diabetics who also carry certain human leucocyte antigen (HLA) genes and autoantibodies are at high risk for disease development has allowed prevention trials to proceed in this population. As a result, opportunities now exist to identify genetic, environmental, and infectious causes of certain autoimmune diseases and to develop novel approaches for treatment and prevention (NIH, 2002, p 9).

Up until now only Western concepts have been discussed. However,

> Chinese medicine does not address the immunity as such but describes the clinical symptoms of the body's response to invasion by external pathogens and internal imbalance. In the "Spiritual Axis" in chapter 18 says: Humans receive Qi from food. This food Qi separates into Defensive Qi and Nutritive Qi. The Defensive Qi (Wei Qi) is derived from the course part of food and water…circulates under the skin…it is on the exterior and it protects. Nutritive Qi (Ying Qi) is closely related to blood and functions in nourishing the internal organs and the whole body (Maciocia, 1998, p. 45).

In summary, Defensive Qi is on the exterior and protects from invasion of outside sources like bacteria and virus. Nutritive Qi is in the interior and nourishes the organs and tissues allowing for a balanced internal response.

AI disease is also complex in Chinese medicine theory as individual disease patterns can differ from person to person or AI disease from another.

As Hou and Wang explain (2011):

> What is common to all AI diseases in Chinese pattern differentiation is Yin deficiency as a root cause. Specifically, a deficiency of yin affects cells negatively and may lead to tissue degeneration, apoptosis and Qi and Yang deficiency, as well as blood stasis resulting in the body's greater susceptibility to pathogens (p. 14).

> Pattern differentiation is foundational to any Chinese medicine practitioner. Acupuncture and herbal treatment will vary on patient's presentation and not based on a western diagnosis. But using Chinese medicine principles we can treat multiple Western diagnosis. Alzheimer's, type II diabetes, infertility, menopause, Parkinson's, osteoporosis, and ALS are often treated by nourishing yin while scar tissue, overgrowth, hepatocirrhosis, amyloidopathy focus more on treating Blood stasis (p. 51).

Appropriate herbs and acupuncture points would be chosen for the patient. Actions of herbal medicinals are taught on ancient uses but there is much known about the pharmacology on many herbs today. For example: ru xiang's also known as boswellia, mechanism of action is as a 5-Lipoxygenase inhibitor, an effective anti-inflammatory (Schweizer, et al., 2002).

Discussion

The western approach often focuses more on the disease and the "attack" than the patient. Western prescriptions may be best used as the last resort instead of the first. By assessing factors contributing to inflammation putting an excessive burden on the immune system we can at a minimum support the patient. This lends more to modulation than overstimulation of the immune system. Looking at environmental stressors like toxicants, chemicals and drugs;

lifestyle like diet, blood sugar regulation, sleep and elimination habits, exercise; and family history along with genetic similarities we may better impact ones overall health. Acupuncture and herbal therapy tends to work synergistically with the body. Sometimes a gentle nudge can be more powerful than a hammer.

References

Autoimmune diseases. (2017, April 28). Retrieved May 18, 2018, from https://www.womenshealth.gov/a-z-topics/autoimmune-diseases

Hafström I., Ringertz, B., Gyllenhammar, H., Palmblad, J., & Harms-Ringdahl, M. (2005, November 29). Effects of fasting on disease activity, neutrophil function, fatty acid composition, and leukotriene biosynthesis in patients with rheumatoid arthritis. *Arthritis Rheumatology*, Volume 31, May 1988, Pages 585-592. Retrieved May 24, 2018, from https://doi.org/10.1002/art.1780310502

Hou, W., Xu, G., Wang, H., & Gould, J. M. (2011). Treating autoimmune disease with Chinese medicine. Edinburgh: Churchill Livingstone.

Ishihara, K., & Hirano, T. (2002, September 04). IL-6 in autoimmune disease and chronic inflammatory proliferative disease. *Cytokine & Growth Factor Reviews*, Volume 13, 2002, Pages 357-368. Retrieved May 23, 2018, from https://doi.org/10.1016/S1359-6101(02)00027-8

Iyer, S. S., & Cheng, G. (2012). Role of Interleukin 10 Transcriptional Regulation in Inflammation and Autoimmune Disease. *Crit Rev Immunol*, Volume 32, 2012, 23-63. Retrieved May 23, 2018, from https://doi.org/10.1615/CritRevImmunol.v32.i1.30

Jonkers, I. H., & Wijmeng, C. (2017). Context-specific effects of genetic variants associated with autoimmune disease. *Human Molecular Genetics*, 26, R185–R192. Retrieved May 23, 2018, from https://doi.org/10.1093/hmg/ddx254

Jörg, S., Grohme, D. A., Erzler, M., Binsfeld, M., Haghikia, A., Müller, D., … Kleinewietfeld, M. *Cell. Mol. Life Sci*. (2016) 73: 4611 Retrieved May 23, 2018, from https://doi.org/10.1007/s00018-016-2311-1

Kuller, L. H., MD. (2018, May 17). Omega-3 Fatty Acids and Coronary Heart Disease: A Very Fishy Story. Retrieved May 23, 2018, from http://professional.heart.org/professional/ScienceNews/UCM_501197_Omega-3-Fatty-Acids-and-Coronary-Heart-Disease-A-Very-Fishy-Story.jsp

Lerner, A., & Matthias, T. (2015, February 09). Changes in intestinal tight junction permeability associated with industrial food additives explain the rising incidence of autoimmune disease. *Autoimmunity Reviews,* Volume 14, Issue 6, June 2015, Pages 479-48. Retrieved May 23, 2018, from https://doi.org/10.1016/j.autrev.2015.01.009

Maciocia, G. (1998). The foundations of Chinese medicine: A comprehensive text for acupuncturists and herbalists. London: Elsevier Churchill Livingstone.

NIH. (2002, January). Autoimmune Diseases Coordinating Committee: Autoimmune Diseases Research Plan. Retrieved May 23, 2018, from http://autoimmune.pathology.jhmi.edu/adrp.pdf

Schweizer, S., Von Brocke, A. F., Boden, S. E., Bayer, E., Ammon, H. P., & Safayhi, H. (2002). Workup-Dependent Formation of 5-Lipoxygenase Inhibitory Boswellic Acid Analogues.

J. Nat. Prod. 63, 8, 1058-1061. Retrieved May 24, 2018, from https://doi.org/10.1021/np000069k

Simopoulos, A. P. (2002). Omega-3 Fatty Acids in Inflammation and Autoimmune Diseases. *Journal of the American College of Nutrition*, 21(6), 495-505. From https://doi.org/10.1080/07315724.2002.10719248

Vojdani, A. (2014, February 12). A Potential Link between Environmental Triggers and Autoimmunity. Autoimmune Diseases, Volume 2014, Article ID 437231, 18 pages. Retrieved May 23, 2018, from http://dx.doi.org/10.1155/2014/437231

BIO of Author - Donna Sigmond, RDN, DACM, LAc, LCh, CLT, FAAMFM, ABAAHP

Owner and practitioner of East West Wellness, LLC in Louisville, Colorado for seventeen years specializing in nutrigenomics, food sensitivities, cardiovascular, gastrointestinal, autoimmune, women and men's health concerns. Donna worked with The University of Colorado at Boulder as a Research Bionutritionist for six years and she did her internship in 2 oncology clinics and her hospital internship with North Austin Medical Center prior to her private practice.