

Good example of psoriasis and inflammatory bowel diseases: an analysis of maladapt...

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Psoriasis and Inflammatory Bowel Disease are two of the most prevalent types of immune disorders. Being diseases of the immune system, both diseases have similarities and, of course, striking differences. Reviewing both of their pathophysiology, incidences of occurrence and maladaptive responses may help us understand how these two immune disorders manifest their symptoms and affect the human body.

Psoriasis is considered as the most prevalent autoimmune disorder, particularly the psoriasis vulgaris type (Krueger and Bowcock, 2005). Additionally, the prevalence of psoriasis considerably varies worldwide (Langley, Krueger, and Griffiths, 2005). In USA, an estimated 2% of the population is said to be affected by the diseases while higher rates can be observed in Faroe Islands, where the rate of the affected amounts to 2.8% of the entire population (Langley, Krueger, and Griffiths, 2005). Common patients affected by the first symptoms of psoriasis are young adults whose ages range from 15 to 20 years old (Langley, Krueger, and Griffiths, 2005). A second wave of psoriatic symptoms occurs during the 55th up to the 60th year of a patient (Langley, Krueger, and Griffiths, 2005). While ethnicity seems to play negligible roles in the onset of the disease, Japanese show low incidences of psoriasis while it may be completely absent among members of the aboriginal Australians and Indians from South America (Langley, Krueger, and Griffiths, 2005).

The pathophysiology of psoriasis includes the interaction of various immune responses, such as the infiltrating leucocytes, resident skin cells, and a selection of proinflammatory cytokines, chemokines, and chemical mediators in the skin which are all regulated by the cellular immune system (Krueger

and Bowcock, 2005). In an experiment conducted to observe the pathophysiology of the psoriasis in mice, it was found that self-reactive T-cells fail to destroy the synoviocytes and their proliferation was stimulated instead (Krueger and Bowcock, 2005). Such characteristic of T-cells in mice affected by psoriasis is hypothesized to be caused by a genetic irregularity or alteration which may predispose synoviocytes or keratinocytes in psoriasis patients to be highly sensitive to various activating stimuli (Krueger and Bowcock, 2005). Aside from high sensitivity to various stimuli, such genetic irregularity or alteration is also hypothesized to cause immune cells to have a lower-than-normal threshold for activation or prolonged activation (Krueger and Bowcock, 2005). Further research on the matter led to the discovery of the possible role played by the dysregulation of the gene SLC9A3R1 which results in the delay of synapse formation and an increase in the time needed for an antigen to be presented to the T-cell receptor, which collectively leads to prolonged inflammation—a contributory phenomenon to the development of psoriasis as described earlier (Krueger and Bowcock, 2005).

Maladaptive immune response of psoriasis disease includes the appearance of red, raised, and scaly lesions on the skin (Krueger and Bowcock, 2005).

The maladaptive immune response caused by psoriasis affects the epidermis in such a manner that keratinocytes proliferate in an abnormal speed, forcing them to mature rapidly as well causing the terminal differentiation that occurs in the granular keratinocytes and squamous corneocytes to be incomplete (Krueger and Bowcock, 2005). The result is the overwhelming growth and dilation of blood vessels, causing the characteristic redness, and equally overwhelming epidermal hyperplasia, or increased growth of skin

tissue (Krueger and Bowcock, 2005).

Another immune disorder that causes alarming maladaptive immune response is the inflammatory bowel disease. Inflammatory bowel disease is generally classified as either Crohn's disease or ulcerative colitis—both of which are idiopathic, meaning they develop spontaneously (Abraham and Cho, 2009). Inflammatory bowel disease is estimated to affect approximately 1.4 million Americans, with the highest prevalence among young adults aged 15 to 30 years old (Abraham and Cho, 2009). Unlike the probable connection of psoriasis to ethnicity, inflammatory bowel disease is heavily associated with unhealthy habits, particularly smoking (Abraham and Cho, 2009). Frequent changes in diet, antibiotic use, and intestinal infestation may also be contributory factors to the development of the disease (Abraham and Cho, 2009).

Crohn's disease differs from the ulcerative colitis in the sense that it largely affects the ileum and colon, although it may also affect any region of the intestine, while the ulcerative colitis generally involves the rectum and part of, or sometimes the entire, colon (Abraham and Cho, 2009). Disturbance of the immune responses that regulate cells in the intestine is the usual cause of inflammatory bowel diseases (Abraham and Cho, 2009). The characteristic cause of inflammation of the intestinal tissues is the infiltration of innate immune cells, such as the neutrophils, macrophages, dendritic cells, and natural killer T-cells, and adaptive immune cells, such as the B-cells and T-cells, into the lamina propria (Abraham and Cho, 2009). Increased levels of activation of those cells stated above may trigger inflammatory responses, therefore causing the excessive inflammation of intestines and parts along

its vicinity (Abraham and Cho, 2009). Just like psoriasis, inflammatory bowel disease, specifically Crohn's disease, may be associated with the autophagy gene ATG16L1 (Abraham and Cho, 2009).

References

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