

germ-free environments show a higher incidence of diabetes compared with those in regular housing, suggesting that a diverse flora may help block development of autoimmune disease.

Myasthenia Gravis

A patient with myasthenia gravis produces antibodies that bind the acetylcholine receptors (AChRs) on the motor end plates of muscles, blocking normal binding of acetylcholine and subsequent muscle activation. In addition, the anti-AChR auto-antibody activates complement mediated lysis of the cells which damages the motor end plate of the muscle. The result is a progressive weakening of the skeletal muscles. Ultimately, the antibodies cause the destruction of cells bearing ACh receptors. The early signs of myasthenia gravis include drooping eyelids and inability to retract the corners of the mouth. Without treatment, progressive weakening of the muscles can lead to severe impairment in eating as well as problems with movement. Treatments are aimed at increasing acetylcholine levels (eg. using cholinesterase inhibitors), decreasing antibody production (using corticosteroids or other immunosuppressants), and/or removing antibodies (via plasmapheresis: the removal and exchange of blood plasma). Experimental autoimmune myasthenia gravis is an animal model of myasthenia gravis in which rabbits immunized with AChRs purified from electric eels became paralysed. The rabbits develop antibodies against the AChR that cross reacted with their own AChRs. These autoantibodies then blocked muscle stimulation by acetylcholine at the synapse and led to progressive muscle weakness.

Systemic lupus erythematosus

Systemic lupus erythematosus (SLE) has a higher incidence in women than men, and a higher incidence in African American and Hispanic women than white women, SLE symptoms appear between 20-40 years of age. Affected individuals may produce auto-antibodies to DNA, histones, clotting factors, red blood cells, platelets, and leukocytes. SLE presents clinically with fever, weight loss, arthritis, kidney dysfunction, and a characteristic butterfly rash across the nose and cheeks. Auto-antibodies specific for red blood cells and platelets can lead to complement mediated lysis, resulting in haemolytic anemia and thrombocytopenia, respectively. When immune complexes of auto-antibodies with various nuclear antigens are deposited along walls of small blood vessels, a type III hypersensitivity reaction develops. The complexes activate the complement system and generate membrane-attack complexes and complement fragments (C3s and C5a) that damage the walls of the blood vessels, resulting in vasculitis and glomerulonephritis. In severe cases, excessive complement activation produces elevated serum levels of certain complement fragments, leading to neutrophil aggregation and attachment to the vascular endothelium. Over time, the number of circulating neutrophils declines (neutropenia) and occlusions of various small blood vessels develop (vasculitis), which can lead to widespread tissue damage. Laboratory diagnosis of SLE involves detection of antinuclear antibodies directed against DNA, nucleoprotein, histones, or nucleolar RNA. indirect immunofluorescence staining, using serum from patients with SLE, produces a characteristic nuclear staining pattern. The New Zealand mouse is an animal model for SLE. F1 hybrids of New Zealand black (NZB) and New Zealand White (NZW) mice spontaneously develop a severe autoimmune syndrome that closely resembles human SLE, although each of the parent strains display only mild autoimmune symptoms. NZB/W F1 mice develop