Autoimmunity

Genetics
Susceptibility to autoimmunity is influenced by genetic factors including defects in specific chromosomal loci, genetic mutations, and epigenetic alterations. The resultant altered patterns of genetic expression are associated with a growing number of autoimmune diseases.

Triggers
With the failure of immune tolerance and/or repeated or acute exposure to environmental triggers, immunity to non-self antigens can elaborate into a self-directed immune process. Many types of non-self antigens can trigger this type of response including dietary sources, bacterial, parasitic, viral, or mold/fungal infections.

- Toxicants
- Mold
- Diet (Gluten)
- Bacteria
- Parasite
- Virus

Hormonal Imbalances
The majority of autoimmunity occurs in females while low testosterone in men has been shown to precede onset.

GALT
is composed of immune inductive sites including Peyer’s Patches, and isolated lymphoid follicles (ILF). Together Peyer’s Patches and ILF generate intestinal IgA responses to mucosal challenges.

Intestinal Permeability & Altered Immunity
Tight junctions are dynamic structures that regulate intestinal permeability. Tight junction dysfunction allows for increased paracellular antigen transport and seems to be a primary defect in many autoimmune diseases.

- Multiple Sclerosis
  Destruction of myelin
- Autoimmune Hepatitis
  Hepatocellular inflammation
- Type 1 Diabetes
  Destruction of insulin-producing beta cells
- Ankylosing Spondylitis
  Chronic inflammation of axial skeleton
- IgA Nephropathy
  Inflammation of glomeruli

GALT serves as a containment system that prevents potentially harmful intestinal antigens from reaching circulation, and induces tolerance against luminal antigens through IgA secretion and induction of T-regulatory-cell activity communicated through the lymph system.