Endocrine Hypofunction and Receptor Defects

Last, and perhaps most important, there is a growing understanding of an extraordinary phenomenon, known as autoimmunity, as a cause of endocrine deficiency. Certain antibodies generated by the body against its own tissues (see IMMUNITY), have been found to be active against certain endocrine tissues. Thus, not only are specific antibodies formed against specific endocrine glands, but there are also antibodies that affect specific aspects of endocrine function.

For instance, in the case of the thyroid there are cytotoxic antibodies that eventually destroy the gland by attacking the cells; there are blocking antibodies that can, in effect, inactivate thyroid cell surface receptors and cause hypothyroidism; and there are stimulatory antibodies, which are a major cause of hyperthyroidism.

Constant exposure of an endocrine gland to blocking antibodies results in a reduction in its cell size and number, a condition known as atrophy. If long-lasting, atrophy may lead to irreversible destruction of the gland. Another cause of atrophy is a receptor defect that results when autoantibodies exert their actions against endocrine surface cell receptors. This kind of receptor damage has been found in females with premature ovarian failure associated with menopause, which can occur as early as the teenage years. It remains debatable whether a natural menopause is an example of hypofunction of the ovary, which should be viewed as pathological, or whether it represents another example of compensatory hypofunction with "survival value."