Archaeological evidence shows that Greenland's other inhabitants, the Thule, flourished throughout the 14th century, thanks to their prowess in hunting ringed seals below the sea ice in late winter, when few other sources of meat and fat were available. But the Norse failed to adopt ringed-seal hunting methods and other technology from their highly successful neighbors, says McGovern.

In fact, the cultural flow seemed to go in only one direction. Teams excavating Thule campsites have uncovered scavenged or stolen Norse artifacts, including chess pieces, iron nails, and fragments of cloth, but Arneborg and others working in the Norse farmsteads have found scarcely any Thule goods. "If you looked at the distribution of artifacts on the Norse side, you would say that there's been no contact," says McGovern. It may be, he adds, that the fervently Christian Norse spurned contact with the shamanistic Thule.

The styles of clothing in Greenland further underline the isolation of the two cultures. Naturally mummified Thule women exhumed at the Qilakitsoq site in 1978 were swaddled in warm sealskin parkas and trousers, while women buried in a Norse churchyard were dressed in low-cut, narrow-waisted woolen gowns like those then fashionable in Europe.

To McGovern, all the evidence to date suggests that for the Norse, ethnic purity triumphed at the expense of biological survival. While the starving settlers slaughtered their cattle and dogs, "there were seals in the fjord, right under the ice." But without harpoons and the skill to find the seals' breathing holes in the ice, the Norse couldn't reach them. It seems, says McGovern, that the Norse in Greenland remained true to the laws and customs of their warmer homeland—and paid the final price for it.

–Heather Pringle

Heather Pringle, a Vancouver-based science writer, is the author of In Search of Ancient North America.

AUTOIMMUNITY

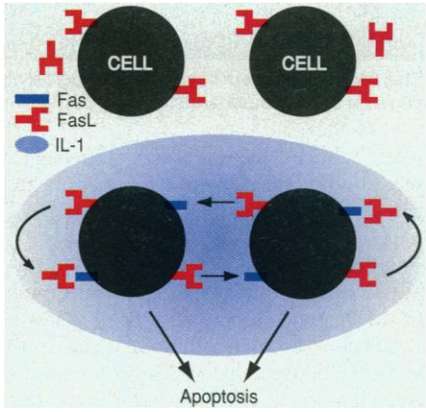
Thyroid Disease: A Case of Cell Suicide?

The name tells the story: Autoimmune diseases, such as diabetes and rheumatoid arthritis, result when the immune system goes awry and turns on the body's own tissues. But how it does so is far from clear. Researchers do not know which of the immune system's several types of killer T cells carries out the attack. And even more puzzling, killer cells are scarce at the site of tissue destruction in many autoimmune diseases. New results published in this issue (p. 960), however, could resolve those puzzles. In at least one disease, they suggest, the immune system itself may not carry out the final act: Instead, the target cells commit suicide through a process called apoptosis.

"These are intriguing results and present an appealing mechanism," says autoimmune disease researcher Ricardo Pujol-Borrell of the University Hospital "Germans Trias i Pujol," in Barcelona, Spain. "What's interesting is that apoptosis is a natural process, and I've always believed autoimmune diseases result from an exaggeration of natural processes," adds immunologist Noel Rose of Johns Hopkins University. So far, the results apply only to Hashimoto's thyroiditis (HT), a disease marked by a gradual destruction of thyroid tissue that is among the commonest autoimmune diseases. But they suggest that in this and possibly other autoimmune diseases, the immune system may have a less direct role than currently thought.

The new work, done in four laboratories in Italy, builds on rapid progress in the past few years in understanding apoptosis, a normal process for eliminating unwanted cells in tissues and organs during development and for reining in immune responses. One key to triggering this process of cell suicide is a molecule called Fas, found on the surface of many different types of cells. When another molecule, the Fas ligand (FasL), binds to it, Fas initiates a series of events inside the cell that leads to its death.

Although many cell types can express Fas on their surfaces, FasL at first seemed to occur mainly on the immune system's activated T lymphocytes. Expression of the ligand allows these T cells not only to kill unwanted cells by prompting them to undergo apoptosis, but also to moderate their own activity by triggering apoptosis in other T cells, which express both Fas and FasL. Researchers soon found that a small number of other cell types expressed FasL, such as cells in the eye chamber, parts of the nervous system, and the testis. These sensitive sites use FasL to protect themselves from immune attack by prompting apoptosis in attacking T cells.



Kiss of death. IL-1 prompts FasL-bearing thyroid cells to express Fas, and hence to die.

Now, the Italian researchers have identified a role for Fas in the converse phenomenon—autoimmune disease. The team was studying cells from patients with HT, a chronic disease, most common in middle-aged women, that leads to loss of thyroid hormone–producing cells. The team's first clue that apoptosis was involved came when they found Fas on the surface of cells taken from the thyroid glands of several patients, while it did not appear on thyroid cells from control glands. They then showed that interleukin-1 (IL-1), an immune messenger molecule found in the diseased thyroid glands, induced control thyroid cells to express Fas.

But what came next really surprised the team. They found that both normal thyroid cells and cells from patients with HT expressed high levels of FasL. "That was totally unexpected," says immunologist Roberto Testi of the University of Rome "Tor Vergata," who is one of the team members. This result suggested that the abnormal Fas expression leads the cells to trigger apoptosis in each other or in themselves.

To bolster this picture, the team took IL-1, which they had shown in lab studies induces Fas expression, and added it to normal thyroid cells in culture. They found that large numbers of cells died with the characteristic features of apoptosis. "This puts a different slant on the role of FasL and suggests a completely unexpected pathological role for the molecule," says Doug Green at the La Jolla Institute for Allergy and Immunology in California.

There are some problems with the apoptosis theory, however: The rapid cell death demonstrated in the laboratory does not square with the normally slow progression of the disease, which can last for years. The team believes that tight control of Fas expression within the body may explain this slow pace. "We need to know the sequence of events," says Testi. Another key unknown is the source of the IL-1 that sets the process in motion. IL-1 is normally produced by activated cells of the immune system to stimulate other cells within the system. "People have not thought of IL-1 as a destructive cytokine, but they now may want to look again," says Green. But if these loose ends in the theory can be tied up, researchers can begin looking for ways to block cell death to prevent thyroid destruction.

The Italian team's results may also hold out hope for a better understanding of other autoimmune diseases—and why T lymphocytes are puzzlingly scarce in so many of them. Says Green: "The new work is a fascinating hint at an entirely new disease mechanism. I think we are going to see more of this."

–Nigel Williams