animals, measurement of N^{15} levels placed all the animals in their expected positions on the food web. That gave the researchers confidence that their placement of the Neandertal between the wolf and the fox was correct, although Mariotti stresses that study of one Neandertal does not imply that all were carnivorous. C^{13} analysis ruled out the possibility that this Neandertal, at least, traveled to the coast and ate fish.

With this first success, Mariotti is now optimistic that archeologists and paleoanthropologists will be convinced that isotopic studies can provide a window on an important aspect of prehistory. That, he hopes, will make museum curators less hesitant to yield bits and pieces of Neandertals and early Homo sapiens sapiens for analysis.

Schwarcz for one is not so optimistic, however. "Wishful thinking," he says. In particular, he notes, "Curators are very unwilling to allow specimens to go for destructive testing...the availability of bones to do this kind of work is very rapidly diminishing as the American Indian movement insists that ancient remains be re-buried." For North America, it seems, the new field of isotopic paleodietary studies may have arrived too late. ■ ALEXANDER DOROZYNSKI and ALUN ANDERSON

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On the Trail of the Errant T Cells of Multiple Sclerosis

Patients with multiple sclerosis (MS) are victims of a mutiny in their own immune system. The disease results when the white blood cells that normally help to ward off diseases by destroying foreign cells turn on the body, attacking a vital tissue in the nervous system and triggering a patient's slow decline into paralysis. Researchers had expected that the mutinous army of cells would be highly diverse, consisting of a varied array of so-called T cells. But according to a report in the 15 October *Proceedings of the National Academy of Sciences*, only a specific set of T cells seems to be taking part in the mutiny. In that case, the researchers argue, putting down the rebellion might turn out to be easier than had been thought.

The work—by Halina Offner, Arthur Vandenbark, and their colleagues at the Oregon Health Sciences University and the Veteran Affairs Medical Center in Portland, Brian Kotzin and his colleagues at the University of Colorado Health Sciences Center in Denver, and other collaborators at Xoma Corporation in Santa Monica and Berkeley, California—bears out a similar finding made last year by David Hafler and his colleagues at Harvard Medical School and Brigham and Women's Hospital (see *Science*, 25 May 1990, p. 1016). And because it parallels results in animal models of MS, it may brighten prospects for a treatment strategy that is already showing promising results in animals and has recently moved into limited clinical trials.

Offner and her colleagues searched through white blood cells from MS patients, looking for the T cells capable of recognizing—and hence attacking—myelin basic protein. That protein is a major component of myelin, the nervous system tissue that degenerates in MS. They found that a large number of these cells carried the same T-cell receptor, a surface protein that enables T cells to recognize their targets. That might not sound surprising: All the errant cells home in on the same protein, after all. But an antigen as complex as myelin basic protein would ordinarily be attacked by a wide array of T cells, bearing a variety of different receptors.

The researchers have no ready explanation for this surprising specificity, but they nonetheless think it bodes well for potential therapies. "The good news," Vandenbark says, "is that there is a strong enough bias in T-cell receptor use that you can treat with one or a few agents" to try and check the autoimmune response. The Oregon group's strategy is to inject part of the same T-cell receptor protein found on the harmful cells. In theory, the injected protein elicits another immune response, this one directed at the disease-causing cells. And that, in turn, might quell the autoimmune attack on myelin basic protein and relieve some symptoms of the disease.

Offner, Vandenbark, and their colleagues have started to work out the strategy in animals, where they say it does appear to reverse the progression of the disease. More recently, they have also begun testing the treatment for toxicity in 11 human patients. At the very low doses used so far, they see no clinical improvement, but the injected protein does seem to be evoking a protective immune response.

But that therapeutic approach may not suffice, says Howard Weiner, a member of the Harvard group that saw a similar restriction in the array of T cells responsible for the human disease. He notes that myelin basic protein may not be the only component of myelin under autoimmune attack in MS. The destruction of myelin proteolytic protein (PLP), he says, may also account for some of the disease's symptoms. And while the array of white blood cells attacking myelin basic protein may be limited, Weiner says, that might not be true for the cells responding to PLP. In that case, stimulating a specific immune response against PLP-reactive cells might prove difficult.

And even the cells attacking myelin basic protein may not be as uniform as the Vandenbark team's results seem to show, according to Hafler, the leader of the Harvard group. "Their findings confirm to some degree the major point of our finding: that the T-cell receptor repertoire for cells reactive to myelin basic protein is restricted," he says. But the Harvard team found that while the autoimmune cells in any one patient may bear one type of receptor almost exclusively, the specific kind of receptor may vary between individuals. That variability would mean that each individual would have to be tested for receptor type before the appropriate therapy could be administered, says Hafler.

Vandenbark and colleagues don't know whether the discrepancy between their results and the Harvard group's earlier findings is due to differences in the two subject groups or in the researchers' analytical techniques. But the Oregon group does agree that receptor typing would have to be done for each individual before therapy. "If the approach does work in humans," says Vandenbark, "we would have to know the biases [toward specific T-cell receptors] for each patient."

And it's surely too early to rule out other therapies, Hafler adds. He, Weiner, and their colleagues are exploring a sort of hair-of-the-dog-that-bit-you approach to autoimmune disease, in which patients are fed the very proteins that triggered the autoimmune response in the first place (see *Science*, 5 April, p. 27). Somehow, the proteins—myelin basic protein or PLP, in the case of MS—seem to summon a set of "suppressor" immune cells, which limit the autoimmune response. But only full-scale clinical trials, says Hafler, will show whether either their approach or the Oregon strategy offers any real hope in the fight against MS. ■ MICHELLE HOFFMAN