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*****Scientists search among
disease-controlling genes
for a predictor of multiple sclerosis

DALLAS--Disease-controlling genes, some of which are suspected to increase a person's susceptibility to multiple sclerosis (MS), are being probed by researchers at The University of Texas Health Science Center at Dallas.

The researchers, including immunologist Dr. J. Donald Capra and neurologists Drs. Richard Tindall and J.T. Phillips, are taking blood samples from patients known to have multiple sclerosis in search of a common genetic marker to indicate who will likely get the disease and who won't.

Funding for the research is being provided by the National Multiple Sclerosis Society.

"With recent knowledge and improved scientific techniques, we hope we can now identify the culprit gene or genes that predispose a person to get multiple sclerosis. And we can learn how they operate," says Capra.

"This offers the potential of influencing genes in order to prevent disease. Also, it may yield pertinent information for curing the disease by means of modifying the immune system," he says.

Multiple sclerosis is an "autoimmune disease" in which the body's immune system goes awry and begins to attack normal tissue. Affecting about 4,000 patients in the North Texas area, MS is a puzzling disease in which neurologic symptoms--numbness or weakness of arms or legs, tremor or paralysis--come and go over a prolonged period of time.

Neurologic symptoms result when the patient's immune system attacks and injures the myelin sheath covering nerves of the brain and spinal cord. Steroid medications may help with sudden relapses of MS, but only for a short time. There are no medications to halt the progressive disabilities produced by the disease when it stays active, says Tindall, a member of the medical advisory board of the Multiple Sclerosis Society's North Texas Chapter.

"One of the reasons MS remains untreatable is that no one knows where to intervene in the disease process," says Tindall. "No one knows what happened in the first place to cause the disease."

Like many diseases with a variety of symptoms, multiple sclerosis may be many diseases masking as one, Phillips says. This offers a clue suggesting that more than one gene may be involved.

"Now we are using DNA probes that will allow us to look in a more detailed way and become more specific in identifying a genetic marker," Capra says.

Some important genetic information is already known about multiple sclerosis, Capra says. For example, people with certain histocompatibility types get the disease at a higher rate than others.

The histocompatibility system, also known as the HLA (human lymphocyte antigen) system, as it was originally defined 20 years ago, is a way of grouping tissue types to determine whether transplanted tissue or organs are compatible with a recipient. The system is named for the genetically determined protein structures (or antigens) that sit on the surface of most cells and trigger an immune response when transplanted tissue or organs are grafted onto a genetically different individual.

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But researchers know that the HLA system is much more than that. Genes within the chromosomal region that code for these HLA antigens also play a role in regulating interactions between lymphocytes and macrophages, white blood cells involved in immune defense.

And it has been found that some HLA "markers" are associated with a predisposition to certain diseases.

An autoimmune disease, such as multiple sclerosis, may be triggered when the body, while defending itself against a foreign invader, provokes an immune reaction with some of its own antigens. This would imply that the foreign invader, such as a bacteria or virus, has at least one antigen identical to an antigen of the victim.

The health science center team is searching for a genetic marker, some product of the gene's coding system, that can predict who will get the disease and who will not. With methods apparently at hand, they are hopeful that identifying a multiple sclerosis predictor is now only a matter of time.

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