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Vital New Light Shed On Iron Overload Disorder

ScienceDaily (Jan. 17, 2008) — Research in the New England Journal of Medicine shows hereditary hemochromatosis is much more common than previously thought and will spur more study to determine who is most likely to develop complications from the debilitating and potentially fatal disease, write two faculty members at the Saint Louis University School of Medicine. Their work appears in an editorial in the NEJM that accompanies the research.

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"This study gives us important new understanding into hemochromatosis – particularly how complications from the disease are common among men with the genetic predisposition for it," said Bruce R. Bacon, M.D., James F. King M.D. Endowed Chair in Gastroenterology, professor of internal medicine and director of the division of gastroenterology and hepatology at SLU School of Medicine, and one of the authors of the editorial in the NEJM.

"We believe this research will further the search for the factors that determine which people with the genetic markers for hemochromatosis go on to develop this very serious disease," added Bacon, who in 1996 was part of the team that identified the genetic mutation that causes the disorder.

Hemochromatosis causes the body to absorb up to three times the normal amount of iron. Over the years, the excess iron builds up in the vital organs, joints and tissues, where it can cause a number of debilitating and potentially fatal conditions, including liver and heart disease, diabetes and arthritis. The disease can be difficult to diagnose because its early symptoms can often be attributed to other causes.

The research published January 16 followed more than 31,000 people in Melbourne, Australia, over 12 years. The study was done by Katrina J. Allen, M.D., Ph.D., of the Royal Children's Hospital in Victoria, Australia, and the University of Melbourne in Australia, along with a more than a dozen other researchers in Australia and the U.S.

The genetic marker for hemochromatosis is a mutation called C282Y. Someone needs to inherit two copies of this defective gene, one from each parent, in order to be susceptible to the disease; when they do, they're called "C282Y homozygotes."

Previous studies have shown that about 1 in 250 people

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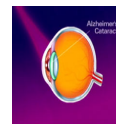
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overall have this genetic marker for hemochromatosis, as do about 1 in 200 people with northern European ancestry. But those studies were not clear as to what percentage of those with the marker would go on to develop the disease. Some estimates had put the percentage at less than 1 percent.

However, the research published today found that the proportion was far higher, particularly among men. Among men with the genetic marker for hemochromatosis, 28 percent were found to have the disease. Among females, the proportion was 1 percent.

Robert S. Britton, Ph.D., associate professor of internal medicine at SLU School of Medicine, co-authored the editorial with Bacon.

The study, Bacon and Britton write, emphasizes "that signs and symptoms associated with hereditary hemochromatosis have a strong male predominance and that C282Y homozygotes need careful clinical assessment to detect liver fibrosis and the arthropathy [joint disease] characteristic of this disease."

Bacon and Britton note that in clinical practice, testing for the genetic marker for hemochromatosis is recommended for any immediate family member of someone newly diagnosed with the disease.

They conclude: "The study by Allen et al. will spur the search for genetic and environmental factors that determine which C282Y homozygotes accumulate substantial amounts of iron and are at risk for clinically relevant tissue damage."

Adapted from materials provided by [Saint Louis University Medical Center](#).

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