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42-40 Bell Boulevard
Bayside, New York 11361-2820
718-224-2999 • Fax: 718-279-9596
e-mail: tourette@ix.netcom.com
http://tsa.mgh.harvard.edu

Editors

Lawrence Scahill, MSN, PhD
John Walkup, MD

Editorial & Production Management

Sue Levi-Pearl, Director, Medical & Scientific Programs

For additional copies, contact TSA

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THE GENETICS OF TS

even after achieving such certainty, the earlier MLS scores can lose significance in a larger sample. So even though we are off to a very promising start, we have a good ways to go.

An especially encouraging aspect of the sibpair study is the close and continuing collaboration of the TSA Consortium. This first publication attests to the success of this unique cooperative effort. Furthermore, recently, the TSA Consortium has received additional funding from the National Institute of Neurological Diseases and Stroke as well as from individual donors who have been impressed by the group's progress. This additional funding for TS genetics will contribute substantially to the Consortium's efforts to find the TS gene(s).

References: 1. The Tourette Syndrome Association International Consortium for Genetics: A complete genome screen in sib pairs affected by Gilles de la Tourette Syndrome; *Am J Hum Genet* 1999; 65:1428-1436.

PANDAS UPDATE

(Pediatric Autoimmune Neuropsychiatric Disorders Associated with Strep Infection)

The idea that some forms of TS and OCD might be sequelae of Group A, β hemolytic streptococcal infection (strep infection) is an important issue of ongoing investigation. Several papers have been published on PANDAS and related issues. More recent articles suggest that antineuronal antibodies are associated with TS and OCD. Singer et al, have identified a relatively specific protein in the sera of subjects with TS. In addition, plasmapheresis, which removes circulating antibodies believed to cause tics and OC symptoms, appears to be associated with both short-term and long-term clinical improvement in a preliminary study (Perlmutter et al, 1999).¹

In Singer et al², a protein of approximately 83 kilodalton (kd) was identified more frequently in the blood of subjects with TS/OCD than in controls. Although these findings are intriguing, their meaning is unclear. First, it remains to be shown whether this protein has any functional consequences. Secondly, the presence of proteins in periphery may have nothing to do with what is going on in the CNS. Thus identification of the same size protein in the CSF of a single subject is still a long way from supporting an antineuronal antibody theory of TS/OCD causation.

A small study by Susan Swedo and colleagues comparing plasmapheresis (N=10) to intravenous immunoglobulin (IVIg, N=9) and an IVIg placebo (N=10) suggested that the immunological treatments were more effective than placebo IVIg in the short term (Perlmutter et al, 1999). In addition, subjects who received the immunological treatments for short-term treatment also benefited long term from this treatment. In an accompanying editorial to the report, Harvey Singer raised a number of issues concerning the study. Perhaps most important, the authors and Dr. Singer³ suggest that these costly and unproven interventions be used *only* in research programs where such treatments are systematically evaluated. Singer specifically advocated that placebo controls are essential for the rigorous evaluation of immunological treatments for PANDAS including plasmapheresis. Further studies at the NIMH are ongoing, including an effort to test plasmapheresis against a sham procedure.

Another treatment approach based on the PANDAS concept is penicillin prophylaxis. Simply stated, ►