



CHAPTER PRESIDENTS

April 30, 2010	CC: Development
<u>Promise 2010/Research Update Call May 11</u>	
Action Requested/Deadline: COB on May 10th	

Please join us on **Tuesday, May 11th at 12pm -1:30pm EDT** for a special follow-up call to our Promise: 2010/Research updates. Information shared on this call will help prepare us to speak knowledgeably and confidently with our boards, other volunteers, donors and prospects about these critical research areas

During this call we will cover the following:

- **Progress of the Promise initiatives**
- **Overview of where Promise Projects are headed**
- **Overview of Identified fundraising focus areas for the balance of fiscal year 2010**
 - **Pilot Grants**
 - **Fast Forward**
 - **Rapid Response (CCSVI)**

Dr. Patricia O’Looney, SVP of Biomedical Research for the Society, Dr. Tim Coetzee, President - Fast Forward and Mary Milgrom, EVP of Individual Giving will be presenting and will be available to answer questions.

While all are welcome, we especially recommend that Chapter Presidents and staff who are operationally responsible for raising money for the Promise 2010 Campaign and other research initiatives join the call.

You must register for this call in advance. Registration information will be sent out shortly. Please allow 90 minutes for this call. The call will be recorded for future playback.

If you have any questions or need any additional information regarding the Promise: 2010 Campaign or research in general, please contact Carrie Radant at 303-698-6100 ext. 15165 or carrie.radant@nmss.org.



MARKETING

April 30, 2010	CC: All
<u>May 2010: E-communications Update</u>	

Matching Gift E-fundraising Campaign

Send dates: 5/11, 5/17, 5/19 and 5/20

Audience: ~800,000

This will be our fourth Matching Gift campaign since the inception of the national email program in November 2007. The campaign is closely integrated with the direct mail channel and is made possible thanks to Pure Protein®, makers of high quality snacks, who will match every donation made toward this campaign dollar-for-dollar up to our campaign goal of \$225,000.

Each one of the four emails – **and** every donation form (Convio Web page) related to the Matching Gift campaign – will clearly communicate to potential donors **before** they donate that the Pure Protein match does **not** apply to other Society fundraising efforts, such as Walk MS and Bike MS. We used this executional element last year as well, and we had little to no reports of donor (e.g. event participant and/or sponsor) confusion.

Please note the send dates have been slightly adjusted to optimize campaign results.

As usual for our e-fundraising campaigns, the Matching Gift campaign will include a variety of promotions on the national website, as well as outreach via our national social media pages.

Notes

Individuals with a ‘no email’ classification on their Altair accounts will be suppressed, along with standard Direct Marketing Program excludes/suppressions. If you would like to review the updated Direct Marketing Program excludes, please visit the new Intranet: Development → FY09_Direct_Marketing_Overview_CD_Master_Exclude_Document.

The current Constituent Communications Calendar is also on the new Intranet: Marketing → Constituent_Communications_Calendar_FY10.

Contact Information

For questions about our online fundraising campaigns, please contact Katharine at katharine.grant@nmss.org or 303-698-6100 x15139.

For questions about our national e-communications strategy, please contact Rich at rich.sarko@nmss.org or 303-698-6100 x15171.



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RESEARCH/CLINICAL UPDATE

cc: Chapter President, Programs

April 30, 2010

Cutting Edge Genetics Study of MS in Twins Points to New Research Avenues to Understand What Causes the Disease

Researchers report on a new study using novel methods to compare genetic material in three pairs of identical twins, where one twin had multiple sclerosis and the other did not. The team found no evidence of any genetic differences that would explain why only one twin developed MS. The authors point out further experiments that might pinpoint such differences. Sergio E. Baranzini, PhD (University of California, San Francisco) and Stephen F. Kingsmore, MB, ChB, BAO (National Center for Genome Resources, Santa Fe) report their findings in *Nature* (2010;464:1351-1356,

<http://www.nature.com/nature/journal/v464/n7293/abs/nature08990.html#/>). Dr.

Baranzini is a Harry Weaver Neuroscience Scholar of the National MS Society, and the study was funded by grants from the Society, the National Institutes of Health, Small Ventures USA Inc., A. J. Brass Foundation, and the Nancy Davis Foundation.

Background: MS involves immune attacks against the brain and spinal cord. It is thought to occur when individuals who have genes that make them susceptible to the disease encounter some unknown triggering factor. The average person in the United States has about one chance in 750 of developing MS. The identical twin of someone with MS, who shares virtually all the same genes, has a one in four chance of developing the disease, suggesting that some factor(s) other than genetics are involved.

Advances in technology have now made it possible to examine the genetic makeup of individuals. This collaborating team decided to take advantage of these advances to develop profiles of genes and gene activity of twins, and explore whether there were any obvious differences that might pinpoint why one twin developed MS and the other did not.

The Study: The team used next generation sequencing methods (which analyze data at unprecedented speed) to screen the genetic material in three pairs of identical twins, where only one member of each pair had MS. They screened the entire genome (all genetic material) in one pair of twins; studied millions of genetic variations in all of the pairs (SNPs, or single nucleotide polymorphisms, the most common type of variation in genetic material); and studied the genetic sequences from T cells (immune cells that are key players in the immune attack on the brain and spinal cord in MS) in all pairs.

The group did not find any differences in genes or gene products, using any of these methods, that would explain why one twin developed MS and the other did not. They comment that further study – such as studying more specific subsets of immune cells – might yield further information of the molecular basis of MS and its risk factors.

Comment: This study of identical twins reveals no genetic difference between twins with and without MS. This is a striking finding, given the significant role that genes play in MS, but the study is small and the authors do cite opportunities for further study that might yield better understanding of why individuals with virtually the same genetic blueprints, such as identical twins, are at different risk for developing MS. The authors also present novel methodology for studying the genetics of disease in twins.

-- Research and Clinical Programs Department



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RESEARCH/CLINICAL UPDATE

cc: Chapter President, Programs

April 30, 2010

Study Suggests that Smoking and Exposure to Epstein-Barr Virus May Interact as Risk Factors for Developing MS

In a new study, researchers show that two individual factors that were previously identified as increasing the likelihood of developing MS – exposure to Epstein-Barr virus and tobacco smoking – may interact and multiply to substantially increase the risk of developing MS in those with both risk factors. Claire Simon, ScD, Alberto Ascherio (MD, DrPH) (Harvard School of Public Health, Boston) and collaborators in Australia and Sweden report their findings in *Neurology* (early online publication, April 7, 2010, <http://www.neurology.org/cgi/content/abstract/WNL.0b013e3181dad57ev1>). The results warrant confirmation in further studies.

Background: MS is thought to occur when people whose genes make them susceptible encounter something in their environment that triggers this immune-based neurological disease. Although many genes probably contribute to susceptibility, a specific gene that has been shown to confer higher susceptibility to MS is called HLA-DR15, which helps control how the immune system identifies targets for destruction. Although many infectious agents have been investigated at various times as possible triggers of MS, no single virus or bacterium has been proved to cause the disease. However, previous studies have suggested that the risk of MS is increased in persons who have had a history of infectious mononucleosis (caused by the Epstein-Barr virus, or “EBV”) or who have high levels of blood serum antibodies against EBV, which indicate past exposure to the virus. Smoking has been associated with an increased risk of developing MS, as well as the rate of MS progression.

In 2008, the Harvard team found that people who had both the HLA-DR15 gene and high levels of antibodies to the Epstein-Barr virus in the blood serum were nine times more likely to develop MS than those without that gene and with low levels of viral antibodies. (*Neurology* 2008;70:1113-18) Exploring such interactions between genes and the environment may help us

understand what triggers MS and also may point to ways to interfere with the development of the disease.

The Study: For the newly published study, the Harvard team investigated even more complex relationships between MS genes and risk factors. They gathered information on people with MS enrolled in the ongoing Nurses' Health Studies (a questionnaire-based longitudinal study that track risk factors for chronic diseases in female nurses); the Tasmanian MS Study (a study that identified people with MS in Tasmania); and the Swedish MS Study (in which people with MS were identified in a national health registry). Dr. Simon and colleagues looked at smoking history, presence of EBV antibodies in blood serum, presence the HLA-DR15 gene, and their relationship to MS risk in the combined group of 442 people with MS and 865 controls without the disease.

By pooling results from each of the three studies, the researchers found that each of the factors raised the risk of developing MS at levels consistent with previous studies: those with the HLA-DR15 gene were about three times more likely to develop MS than those without this gene; those with serum EBV antibodies were about two and a half times more likely to develop MS than those without EBV antibodies; and those who ever smoked were about one and a half times more likely to develop MS than those who never smoked.

However, when EBV exposure was taken into account, smoking only increased MS risks among those with high levels of serum EBV antibodies. Current or previous smokers with low levels of EBV antibodies had no increased risk for developing MS, whereas current or previous smokers with the highest levels of EBV antibodies were 70% more likely to develop MS than those with neither risk factor. The presence of the HLA-DR15 gene variation did not appear to modify any of these effects.

Comment: The National MS Society's Task Force on Epidemiology of MS met in 2007 to establish research priorities for epidemiologic studies that examine aspects of people who get MS for clues to its cause. Investigating the interactions of genes and environmental factors was identified as an important avenue of research. This study is novel in its approach, examining possible interactions between several risk factors, rather than looking at factors in isolation. If confirmed, the results may help to explain some of the complexities of MS, and open up new explorations into its cause and prevention.

Read more about research in MS [genetics](#) and [risk factors](#).

Research and Clinical Programs Department