

CMSCenterPiece

The 2007 CMSC Annual Meeting Newsletter

Washington, DC

Edited by June Halper and James Simsarian, MD • June 2, 2007

Foundation of the CMSC Awards 2007 Whitaker Prize to Thaddeus Carlson

After a full day of talks covering basic research in MS, Thaddeus Carlson received the 2007 Whitaker Prize from the Foundation of the CMSC, a \$5,000 research grant. Carlson's presentation described the novel function that neutrophils may serve in animal models of MS—breaking down the blood/brain barrier and allowing inflammatory cells to access tissue.

Carlson is an MD/PhD student in the Medical Sciences Training program at the University of Rochester. He will complete his PhD studies with Benjamin Segal, MD, this summer. After finishing his MD, he plans to continue working in research on MS and other diseases with a neuroinflammatory component.

"All the presentations were very impressive," said Dr. Michael Racke, chair of the selection committee. "What distinguished Thaddeus Carlson's presentation was the question-and-answer session. He knew the literature very well about the role of neutrophils in associated conditions."

Recap of Genetics in MS--Stephen Hauser, MD

Stephen Hauser, MD, Chair of the Department of Neurology at UCSF, presented an overview of progress in understanding the genetics of MS in his lecture Friday morning. "The message that I would like to bring home," Dr. Hauser said, "is the opportunity and importance of genetics and epidemiology to get at the root of MS."

Because of disease heterogeneity, researchers believe that there are multiple root causes of MS. "I would say MS still has a surprise for us," Dr. Hauser said. Clinical criteria alone are not informative, he said, and genetics serves as a source of data that is not burdened by preexisting assumptions.

In recent years, researchers have shown a link between certain variants of two genes, DR β 1 and DR β 5 on chromosome 1. Caucasians with MS often have both modified genes, while African-Americans with MS typically have only the DR β 1 mutations and more severe disease. "Therefore we have concluded that these DR β 1 alleles may lead to increased susceptibility to MS, and the associated DR β 5 allele may be a modifier that decreases severity of the resulting disease," Dr. Hauser said. He and his colleagues speculate that DR β 1 may trigger an immune response by activating T-cells. DR β 5 may trigger regulatory T-cells that modulate the activity of the immune response.

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In addition, Dr. Hauser announced that he and his colleagues within the International MS Genetics Consortium have found a mutation on chromosome 5 within the interleukin-7 alpha chain gene associated with MS. They have evidence that the MS-associated gene could be functionally different based on this mutation. "I expect the interleukin-7 alpha chain to be the next big area in the immunologic exploration of MS," Dr. Hauser said.

Highlights from the Poster Session

- **BENEFITs of Early Treatment**-- Dr. Mark Freedman shared results from the BENEFIT study (**B**etaseron in **N**ewly **E**merging multiple sclerosis **F**or **I**nitial **T**reatment) that validates that immediate treatment of early stages of MS with a immunomodulatory agent can significantly slow the onset of disability. Betaseron is marketed by Bayer Pharmaceuticals and is indicated for the treatment of relapsing forms of MS to reduce the frequency of clinical exacerbations. Results showed that prompt initiation of Betaseron treatment in patients with a first MS attack was associated with a 40 percent reduced risk of confirmed disability progression over three years.

- **Design of Phase 3 trials of oral drug candidate (BG00012)**-- Dr. Robert Fox shared the design of two multicenter, randomized, 2-year, phase 3 studies of BG00012, an oral fumarate derivative. This drug is an immunomodulator that works by a novel mechanism. Such drugs have been used successfully for long-term treatment of psoriasis. Patient recruitment began early this year, and more than 2,000 subjects will be enrolled.

TODAY'S HIGHLIGHTS

- **Donald Paty Memorial Lecture & CMSC/ACTRIMS Joint Lecture, 8 – 8:45am**

Prof Herman Waldman, FRS, will speak this morning on Reprogramming the Immune System in Multiple Sclerosis. The immune system normally avoids self-reactivity through processes collectively embraced by the term "Tolerance." If some of these processes could be harnessed therapeutically, then it should be possible to reprogram the immune system to stop autoimmune damage, and to reinstate tolerance. Prof. Waldmann will overview our understanding of what reprogramming is possible.

- **Join us for the first joint CMSC/ACTRIMS Meeting from 9am – 4pm**

Symposia and workshops will cover emerging therapies in MS. CMSC participants may attend sessions by showing their badge.

- **Farewell Party 7 – 8:30pm at the Gazebo**—last chance for cocktails with colleagues.

Join us next year for the CMSC meeting, May 28-31, 2008, in "COOL" Denver, CO