RACE TO ERASE MS

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22nd Annual "Love to Erase MS"

Early this spring on April 24, 2015 we celebrated our 22nd Race to Erase MS at the Hyatt Regency Century Plaza. Our honoree and "Medal of Hope" recipient Tommy Hilfiger is dedicated to cure this disease and has high hopes for the future.

Nancy Davis, Tommy Hilfiger, Rita Ora, Ne-Yo

Presenters for the evening were Sharon Osbourne, Kelly Osbourne, Jack Osbourne, Randy Jackson, Anne Heche, James Tupper, Paris Hilton, G. Hannelius and Rowan Blanchard. The evening also included a live auction, during which lucky bidders walked away with items including a trip to Hawaii, Greece, an experience with Larry King , a stay on the Illusions Yacht, and a custom designed Aston Martin commissioned by Tommy Hilfiger. Race to Erase MS continued its legacy of incredible musical performances this year, with Grammy award winning artist Ne-Yo who sang an incredible set which included "Sexy Love, "So Sick", "She Knows" "Let Me Love You", "Miss Independent and more. Rita Ora sang hits "Doing It", "I Will Never Let You Down" and "Black Widow". Our Musical Director / Producer for the evening was the talented Greg Phillinganes. Guests could not stop talking about the spectacular floral arrangements generously donated by Mark's Garden. Everyone commented on their extraordinary beauty. Mindy Weiss, well known event designer, created a lively auction atmosphere. Guests couldn't leave without picking up the amazing Race to Erase MS gift bag so generously donated by Tiger J. Sweet E's Bakeshop spoiled guests with scrumptious treats to top off the evening. We would like to thank our amazing presenting sponsors Hyatt Regency Century Plaza, Aston Martin, Associated Television International and ALEX AND ANI® for their generous support. Our event sponsors were truly instrumental in making the Race to Erase MS an electrifying evening. Thank you to Bianchi Winery, Svedka Vodka, Neo North America, New Belgium and Nice Guy Limo for their incredible support. Thank you to Cristophe Salon and MAC, who created the amazing hair and makeup for our celebrity presenters. Please make sure to save the date April 15, 2016 for our 23rd Annual Race to Erase MS!

ECTRIMS Summary Update

By Christina Azevedo, M.D., M.P.H., Yale University

The annual European Consortium for Treatment and Research in MS (ECTRIMS) meeting was held in October 2015 in Barcelona, Spain. This meeting brought exciting news about a potential new MS therapy, as well as more information on current FDA-approved therapies and drugs still in investigation.

Potential New Therapy: Ocrelizumab:
Researchers announced the results of 3 large studies using ocrelizumab, an IV medication given every six months. Ocrelizumab specifically targets B cells, a type of white blood cell thought to be very important in the MS disease process. Two of the three studies (OPERA I and OPERA II) were performed in patients with relapsing MS. Patients treated with ocrelizumab did better on all outcomes compared to those on Rebif (fewer relapses, less disability progression, fewer new MRI lesions and less brain atrophy). Ocrelizumab was also tested in patients with primary progressive MS in the ORATORIO study. Compared to placebo, patients on ocrelizumab did better on all outcomes (less disability progression, better walking times, fewer new MRI lesions and less brain atrophy). Ocrelizumab seemed reasonably safe in all 3 studies, with most common side effects being mild-to-moderate infusion reactions.

Genentech plans to submit these results for FDA approval in early 2016. If successful, ocrelizumab could be available for widespread use as early as 2017. We anticipate that ocrelizumab

(ECTRIMS continued on Page 15)

Message from Nancy Davis

President and Founder

What an exciting year in development of MS research with 13 therapies now on the market. We are excited about the pending FDA approval of Ocrelizumab, a drug that we have been heavily involved with in pilot studies for many years. It is so amazing to see this come to fruition and it will be the first MS drug to help with primarily progressive MS. I can feel the tremendous energy in the air as we move forward toward a cure. In November our Center Without Walls participants met for their semi-annual symposium and I was filled with such hope as they spoke energetically about their findings that could lead to additional studies on the path to unlocking answers about this complex autoimmune disease.

Our Young Investigators continue to be the corner stone of our accomplishments and you can read about the current research we are funding in this issue. We would like to welcome to our Scientific Advisory Board (SAB) new member Anne Cross, M.D., Professor Neurology at Washington University. She will be joining Dr. Henry McFarland, Dr. Daniel Reich and Monica J. Carson, Ph.D,who comprise our brilliant SAB team who are the key to ensuring every dollar raised is only going to the most cutting- edge MS research.

We are thrilled to be honoring Founder, Creative Director, and CEO of ALEX AND ANI®, Carolyn Rafaelian, with our Race to Erase MS "Medal of Hope" Award at our 23rd Race to Erase MS gala on April 15, 2016. Her innovative company has been instrumental in accelerating our mission to find a cure for multiple sclerosis as well as providing inspiration and hope to so many with this disease. A guiding light and perpetual inspiration, Rafaelian is living proof that anything is possible with hard work and a little positive energy. And that is the same motto we live by at Race to Erase MS. Together as a team we will win this Race and find a cure for multiple sclerosis. Save the date April 15, 2016 for the Race to Erase MS gala and join us the following day on April 16th for our MS Forum and Expo at The Beverly Hilton. Wishing everyone love, health and happiness in the New Year!

North American Imaging in MS (NAIMS) UPDATE

Exclusively funded by Race to Erase MS

In conjunction with the 2015 Race Fall meeting, NAIMS hosted a two-day multisite meeting to discuss current and future projects. An update on the single subject NAIMS pilot study was given and data collection was completed on December 20th. This initial NAIMS project involves sending a single MS patient to 7 different NAIMS sites to undergo advanced MRI scanning.

Following the pilot project meeting, NAIMS steering committee members welcomed visiting scientists from across North America and Europe for an evening poster session and dinner meeting at Cedars-Sinai Medical Center that featured work from Race affiliated sites. The following day, the group convened again for a full day session on the USC campus. The theme for discussion was "The Role of the Central Vein in Identifying MS Lesions." The day included invited talks from experts in the fields of neuropathology, imaging and clinical trials in order to design and implement clinical research projects to better recognize MS lesions using advanced imaging approaches.

The NAIMS Cooperative will meet again at the American Academy of Neurology in April 2016 to review the pilot project results and draft manuscripts. We will review applications from other imaging centers in North America who have expressed interest in joining the Consortium. The strong foundation made possible by Race to Erase MS support will allow us to advance the field of advanced imaging to test the next generation of MS therapeutics and beyond.

What is NAIMS?

The North American Imaging in Multiple Sclerosis Cooperative (NAIMS) was established in 2012 with the support of the Race to Erase MS. The goals of the NAIMS group are to: (1) develop reliable imaging-based measures for disease-progression in multiple sclerosis (MS); (2) accelerate the pace of MS research in North America by creating standardized imaging protocols for use in clinical research; and (3) bring together a range of imaging expertise focusing on the study of MS

Source Note:
Summary provided by Dr. Nancy Sicotte, Cedars-Sinai

Our Race to Erase MS 2016 "Medal of Hope" Honoree!

Carolyn Rafaelian

CEO of ALEX AND ANI®

Carolyn Rafaelian

On April 15, 2016 we will celebrate our 23rd Race to Erase MS. I am so honored and proud to recognize Founder, Creative Director, and CEO of ALEX AND ANI®, Carolyn Rafaelian, with our Race to Erase MS "Medal of Hope" Award as the company has been instrumental in accelerating our mission to find a cure for multiple sclerosis as well as providing inspiration and hope to so many with this disease.

Race to Erase MS is just one of many charitable organizations who have generously benefited from the innovative ALEX AND ANI | CHARITY BY DESIGN® collection by contributing over $750,000 to MS research through the Cupcake Charm Bangle. This unique philanthropic driven collection was born from the ALEX AND ANI ideal of spreading positive energy worldwide and strengthens charitable organizations through collaborative experiences. Below is a brief biography on this inspirational human being and we are so honored to recognize Carolyn at our 23rd Annual Race to Erase MS:

Carolyn Rafaelian transformed a family tradition of jewelry making into a worldwide lifestyle brand. Recognized as much for her entrepreneurial spirit and skill as for her goodwill, Ms. Rafaelian learned the craft of jewelry design and production as a young apprentice to her father, who owned Cinerama, a jewelry factory in Cranston, Rhode Island. In the time since she launched ALEX AND ANI™ in 2004, the company — named for her first two daughters — has turned heads in both the business and fashion industries.

The ALEX AND ANI collection is MADE IN AMERICA WITH LOVE™ using recycled materials. The designs feature everything from spiritual symbols and charitable partnerships to licensed logos of major sports teams, colleges and universities, and Disney characters. Today, the company has expanded to include a beauty line and handbags.

ALEX AND ANI products deeply reflect Ms. Rafaelian's values, charitable focus and desire to spread positive energy across the globe. To date, ALEX AND ANI via their CHARITY BY DESIGN™ collection has donated over 26 million dollars to charities around the world. Her designs are created with a deep reverence for powerful and sacred symbols that express and inspire awareness, empowerment and inner beauty. Ms. Rafaelian is involved in all facets of her company's operations, particularly its designs and marketing efforts, and maintains close associations with its vendors.

Based in Cranston, RI, the company is a five-time Inc. 5000 honoree. ALEX AND ANI has been included on Inc. Magazine's list of fastest-growing U.S. companies in 2012, 2013, and 2014. In 2012, Ms. Rafaelian was named New England Entrepreneur of the Year in 2012 by Ernst & Young and the Small Business Administration's Rhode Island Small Business Person of the Year.

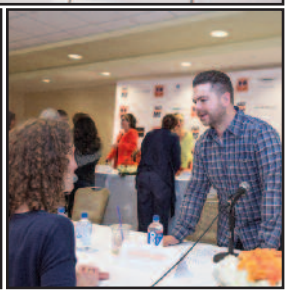
MS Forum and Expo

2016: Save the Date April 16th

Don't miss our Spring MS Forum and Expo on April 16, 2016. The event is free and open to the public and we welcome you, your family, and friends to attend this unique opportunity to ask questions, receive resources and information, and to speak directly to top MS research doctors from around the country.

Our forums this past year covered topics including emotional wellness, remyelination and repair, promising therapies for MS, clinical trials, brain atrophy and progressive MS. Photos are from both forums this year. Make sure to follow us on our social media platforms or check our website to obtain updates on our topics for 2016.

Dr. Adam Kaplin, Dr. David Hafler, Dr. Peter Calabresi, Dr. Emmanuelle Waubant, Jack Osbourne, Nancy Davis, Claudia Curry Hill, Dr. Leslie Weiner, Dr. Howard Weiner, Dr. Daniel Pelltier, Dr. Dennis Bourdette



Guests enjoy the resources provided by our invalubale Expo Partners and Panelists

RACE ERASE MS

CENTER WITHOUT WALLS
Collaborating Physicians

Dr. Katerina Akassoglou, UCSF
Dr. Lilyana Amezcua, USC
Dr. Dennis N. Bourdette, OSHU
Dr. Peter A. Calabresi, Johns Hopkins
Dr. Rob Bakshi, Harvard
Dr. Michele Cameron, OHSU
Dr. Tanuia Chitnis, Harvard
Dr. Margarita Dominguez-Villar, Yale
Dr. Roopali Gandhi, Harvard
Dr. Wendy Gilmore, USC
Dr. Pierre-Antoine Gourraud, UCSF
Dr. David Hafler, Yale
Dr. Roland Henry, UCSF
Dr. Jiwon Ho, Johns Hopkins
Dr. Adam Kaplin, Johns Hopkins
Dr. Eve Kelland, USC
Dr. Brett Lund, USC
Dr. Ellen Mowry, Johns Hopkins
Dr. Gopal Murugaiyan, Harvard
Dr. Kevin O'Connor, Yale
Dr. Jorge Oksenberg, UCSF
Dr. Daniel Pelletier, USC
Dr. Samuel Pleasure, UCSF
Dr. William Rooney, OHSU
Dr. Nancy Siccotte, Cedars-Sinai
Dr. Jack Simon, OHSU
Dr. Rebecca Spain, OHSU
Dr. Arun Venkatesan, Johns Hopkins
Dr. VJ Yadav, OHSU
Dr. Emmanuelle Waubant, UCSF
Dr. Howard Weiner, Harvard
Dr. Leslie Weiner, USC
Dr. Scott Zamvil, UCSF

2015-2016 Young Investigators

Dr. Oleg Butovsky, Harvard
Dr. Steve Fancy, UCSF
Dr. Jennifer Graves, UCSF
Dr. Naila Makhani, Yale
Dr. Shiv Saidha, Johns Hopkins

EVERYDAY HEALTH

Multiple Sclerosis Survey Results

Everyday Health collaborated with the Race to Erase MS team to produce a survey that would uncover factors related to MS diagnosis, progression and treatment. We set out to gain insights around the connections between behavior and the diagnosis, explore causal factors, behaviors at different ages and personal perceptions. Additionally we wanted to gain a better understanding of the patient/physician dialogue to uncover gaps and aid in better disease management. Questionnaire methodology and topics were discussed over several months with members of the Everyday Health research team and the Race to Erase MS. In order to garner widespread representation of patients diagnosed with MS we promoted the survey via Everyday Health channels (website, newsletters, social media, and email) as well as the Race to Erase MS community. Additionally data among those not diagnosed with MS was collected as a control group for comparison. One of the most interesting findings in patient behavior was that many people live and cope with MS symptoms more than a year before diagnosis which may have an impact on lifestyle modification adherence. Once a patient is on a therapy they are consistent about taking their medications, but slack off on healthy lifestyle habits. It is vitally important to take care of your body with exercise, good food and healthy behaviors in conjunction with medication in order to maximize positive outcome with MS. We thank Everyday Health for their tremendous support with this important endeavor.

Visual Investigation in Multiple Sclerosis

By Shiv Saidha, MD, MRCPI, Johns Hopkins University School of Medicine

Optical coherence tomography (OCT) is a cheap, reproducible, easily repeatable, non-invasive, well tolerated, and quantitative in addition to qualitative imaging technique enabling high-resolution imaging of the retina. Conventionally derived OCT measures include retinal nerve fiber layer (RNFL) thickness and average macular thickness (a non-specific measure of the combined thickness of all retinal layers). Advances in OCT technology have led to precise, automated segmentation techniques enabling individual layers of the retina to be measured. The combined ganglion cell and inner plexiform layers (GCIP), combined inner nuclear and outer plexiform layers (INL), and the outer nuclear layer including the photoreceptor segments (ONL) are now amenable to accurate and reproducible quantification. The RNFL is composed of unmyelinated nerve fibers (axons) and is the innermost layer of the retina. Under normal circumstances the retina is unmyelinated. As a result, retinal measures are not confounded by myelin (unlike brain volume measurements for example), making them ideal for investigating/monitoring neurodegeneration, and potentially neuroprotection and neurorestoration, in MS. RNFL axons, derived from ganglion cell nerve cells located in the ganglion cell layer below the RNFL, coalesce at the optic discs to form the optic nerves. There is a clear propensity for optic nerve involvement in MS, both clinically (acute optic neuritis occurs in 30-70% of patients), and sub-clinically. In fact, optic nerve pathology is evident in up to 99% of MS patients at post-mortem examination. Optic nerve demyelination results in retrograde degeneration of its constituent fibers, culminating in thinning of the RNFL and ganglion cell layer.

Although the GCIP (containing ganglion cell nerve cells) and RFNL (containing the nerve fibers of ganglion nerve cells) are similar, GCIP thickness seems to have superior structure-function relationships than RNFL thickness, with respect to high contrast (100%) and low contrast (2.5% and 1.25%) visual function, as well as expanded disability status scale (EDSS) estimates of disability in MS. Furthermore, the GCIP may not be susceptible to fluid related swelling, unlike the RNFL, during optic nerve inflammation. For these reasons, monitoring neurodegeneration in MS by tracking GCIP rather than RNFL thicknesses may be better. It is noteworthy that patients exhibiting disease activity (relapses and/or new lesions on MRI) or disability progression during the course of follow-up appear to have faster rates of GCIP thinning, further underpinning the utility of this neurodegenerative measure for tracking patients clinically. OCT also reveals quantitative and qualitative abnormalities of the INL and ONL (the deeper retinal layers) in MS. The origin of these abnormalities has been the source of lively scientific debate, with some investigators proposing they may be the result of primary retinal neuronal mechanisms of pathology, and others suggesting they may be the sequelae of optic nerve pathology, either as a result of neurodegeneration or simple mechanical alterations, such as related to traction effects. Despite this debate, the OCT identification of INL and ONL thinning in MS eyes without a prior history of optic neuritis, in which there is relative preservation of the RNFL and GCIP is referred to by some investigators as the macular thinning predominant (MTP) phenotype. From a clinical monitoring perspective, this OCT pattern may be associated with more rapid accumulation of disability. Although this pattern may be consistent with the post-mortem demonstration of drop-out of nerve cells in the INL in MS eyes, qualitative assessment of OCT scans in a small proportion of MS patients reveals macular microcystoid changes, predominantly in this same layer.

As mentioned, the origin and significance of such abnormalities in MS eyes remains a topic of debate. Nonetheless, macular microcystoid changes in MS may be a harbinger of more aggressive disease. Since macular microcystoid changes may change over time,

(VISUAL continued on Page 13)

highlights from the lab

Center Without Walls Program Young Investigator Research Update

Our mission is to fund cutting-edge, innovative research programs in our quest to find a cure for multiple sclerosis. We are thrilled to support the best and the brightest young minds in scientific research. Below are annual updates from our second year Young Investigator grant awardees that began their basic science research in 2013 as well as research study summaries from our newly awarded 2015-2016 Young Investigators and Pilot Studies.

Young Investigator (YI) Summaries: Support for a promising new investigator performing state-of-the-art MS research.

1st Year YI Grant Recipients:

Naila Makhani MD MPH
Assistant Professor of Pediatrics and Neurology, Yale University

Identification of Novel Biomarkers for Pediatric Multiple Sclerosis:

We have begun to realize that the first symptoms of MS may present as early as in childhood. The challenge is that only some of the children who present with an MS-like first attack go on to develop MS, while most do not. A current gap in MS research is the lack of robust markers to identify those children who will go on to have relapsing MS after a first clinical attack.

With this support from Race to Erase MS, we will aim to identify novel markers for pediatric MS. First, we will look at patterns on brain and spinal cord magnetic resonance imaging (MRI) scans that characterize pediatric MS using both standard and newer MRI techniques. Second, we will use a recently developed laboratory test to examine the behavior of immune cells that may be unique to children with MS. We will use what we learn in the imaging and immune analyses to build a statistical model to try and better predict which children truly have MS when they first present with possible symptoms. Identifying these children early will help us begin MS treatments in a timely manner and, in so doing, potentially reduce long-term disability in our very youngest MS patients.

Stephen P.J. Fancy, DVM PhD
Assistant Professor of Pediatrics and Neurology, UCSF

Remyelination in MS:

Permanent damage to white matter tracts, comprising axons and myelinating oligodendrocytes, is an important component of multiple sclerosis. In MS, myelin sheaths are lost through injury or death of mature oligodendrocytes (OL) as a result of autoimmune damage. In these conditions, myelin sheaths can be regenerated by oligodendrocyte progenitors (OPC) that are recruited to lesions and differentiate in a process called remyelination. But regulatory factors relevant in human myelin regeneration are unclear. Evidence suggests that myelin repair often fails in MS, and that this failure can result from failed OPC migration into lesions as well as their failed maturation into myelin forming OL once recruited to lesions.

The inhibition of myelin repair contributes significantly to ongoing neurological dysfunction, axonal loss and disease progression in MS, and there are currently no treatments for promoting remyelination. We are investigating key signaling pathways within OPCs that regulate their ability to migrate into lesions and differentiate into myelin forming oligodendrocytes. In particular, we are investigating the role of the Wnt pathway in both OPC migration and differentiation, and its role in co-ordinating the timing of these two events. Evidence suggests this pathway is a potent regulator of myelin repair, with the potential for dysregulation in MS, and also a pathway that may serve as a key therapeutic target in the future.

2nd Year YI Grant Recipients:

Oleg Butovsky Ph.D.
Instructor in Neurology,
Brigham and Women's Hospital
Harvard Medical School

Mechanism of regulation of inflammation by microglia in EAE:

Microglia are resident myeloid-lineage cells in the CNS and function in the maintenance of normal tissue. Microglia can become activated and/or dysregulated during disease, and thus affect disease progression or resolution in MS.

Understanding the biology of microglia is a challenge due to absence of markers and genetic signatures in health and disease that distinguish them from hematogenous infiltrating macrophages with identical morphologies. Recently, we identified a unique homeostatic molecular microglia signature (M0) and generated novels tools to specifically identify and isolate microglia in homeostasis and disease including mouse models and human MS. Now we succeeded for the first time to specifically isolate microglia from fresh postmortem human MS brain lesions. Using advanced genetic tools we identi-

fied a molecular signature of neurodegenerative microglia phenotype (MGnD) in the EAE animal model of MS.

The focus of our current research is to identify the mechanism, which regulates microglia phenotype switch from the homeostatic (M0) to neurodegenerative (MGnD). We identified key regulators including transcription factors and surface receptors unique to microglia that control microglia phenotypes. Curently, we utilizing genetic and pharmacological approaches to target these key regulators in order to restore the unique properties of microglia in the EAE animal model of MS. The support of the Race to Erase MS will help us to identify the mechanism of regulation of inflammation by microglia in MS. Moreover, it will help to design new therapeutic strategies for the treatment of MS.

3rd Year YI Grant Recipients:

Jennifer Graves
Assistant Professor of Neurology in the UCSF Adult and Pediatric MS Centers

Genetic Risk:

There is strong evidence for vitamin D and exposure to Epstein-Barr virus (virus that causes mono) as risk factors for multiple sclerosis. As environmental factors, these are potentially modifiable. Genetic risk is also important in multiple sclerosis, with the strongest known genetic risk factor for MS affecting immune function. As the majority of people with low vitamin D levels and exposure to common viruses do not develop MS, it is important to understand how genes may modify the influence of the environment.

The next frontier for MS care is identification of early markers of future severity. As newer therapies become available, some with concerning side effects, patients will require personalized treatment based on such markers. My work focuses on how MS related genes interact with the environment to affect relapse rate in multiple sclerosis. Our preliminary work suggests that genes do enhance the effects of vitamin D levels causing increased relapses in children with MS. With continued support from the Nancy Davis Foundation we plan to extend our findings to create new genetic risk scores to better describe the role of vitamin D in MS severity and to define metrics of common viruses that may relate to relapses. We will also expand the study to new genes and gene-environment interactions and include a larger group of patients from nine pediatric MS centers. We are using cutting edge genetic and viral technologies in collaboration with eminent scientists.

Shiv Saidha, MD, MRCPI
Assistant Professor of Neurology, Johns Hopkins University School of Medicine

Optical Coherence Tomography in MS:

Optical Coherence Tomography in MS

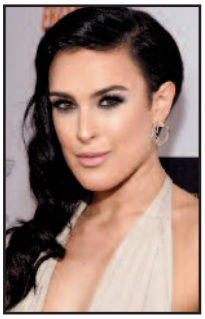
MS is the leading cause of non-traumatic disability in adulthood in the developed world. Although MS is clasically defined as an inflammatory demyelinating disorder of the central nervous system, axonal and neuronal degeneration (neurodegeneration) are also clinically important pathologic hallmarks of the disorder. In fact, numerous studies have shown that disability in MS correlates best with neurodegeneration, and therefore it is no surprise that in recent years there has been growing investigation to determine the mechanisms underlying neurodegeneration in MS, the impact of neurodegeneration in MS, as well as the development of neuroprotective agents, or even potentially neurorestorative agents, that may combat neurodegenerative processes in MS. Critical to the success of such endeavours is the necesstiy to be able to measure neurodegeneration, and indeed neuroprotection, in a precise and objective fashion in-vivo in MS. MS has a predilection to affect the anterior visual system both clinically and subclinically, such that virtually all MS patients demonstrate optic nerve lesions at post-mortem. Demyelination and inflammation within the optic nerve result in retrograde degeneration of the constituent fibers of the optic nerve. These fibers are derived from the inner-most layer of the retina, called the retinal nerve fiber layer (RNFL), and the axons which comprise the RNFL are derived from ganglion cell neurons (which are located in a layer below the RNFL termed the ganglion cell layer). Therefore, MS related optic nerve pathology results in thinning of the RNFL and ganglion cell layer (GCL).

Our group has been utilizing optical coherence tomography (OCT), a non-invasive, precise, cheap, reproducible, easily repeatable and well tolerated imaging technique to quantitatively and qualitatively investigate in-vivo changes in retinal axonal and neuronal sub-populations in MS. We have employed non-conventional OCT techniques, in particular OCT-segmentation, which allow us to quantify discrete retinal layers of the macula in MS patients. Through our work, we have shown that GCL+inner plexiform layer (GCIP) thickness may be an ideal candidate outcome measure in trials of neuroprotection, and even neurorestoration. Moreover, we have shown with this technique that the eye is truly a window into the global MS disease process. For example, patients exhibiting disease activity (such as relapes, new T2 lesions on MRI, new contrast enhancig lesions on MRI) or even disability progression during the course of follow-up have the highest rates of GCIP thinning. More recently we have also shown with this technique that over time thinning of the GCIP mirrors loss of global brain tissue. In addition, we have also found that primary retinal neuronal mechanisms of pathology in deeper layers of the retina (such as the inner and outer nuclear layers) may be operative in MS, and that such processes may not necessarily relate to optic nerve pathology or demyelination. Along these lines, thicknesses of the RNFL and GCL appear to correlate best with gray matter volume in MS, while thickness of the inner nuclear layer in MS seems to correlate mostly with T2 lesion volume. Interestingly, we have shown that patholo-

(LAB continued on Page 10)

photo gallery: 22nd Annual Race to Erase MS


Steve Hash, Ally Hilfiger, Ioan Gruffudd, Alice Evans, Dee and Tommy Hilfiger


Rumer Willis


"Lean on Me" Finale


Jack, Lisa, Kelly and Sharon Osbourne


Nancy Davis and Tommy Hilfiger


Tommy Hilfiger and Rita Ora


Kym Johnson and Robert Herjavec


Ne-Yo and Nancy Davis


Whitney Davis with Guest and Barbara Davis and Brooke Weiderhorn


Paul and Lynn Palmer


Randy Jackson

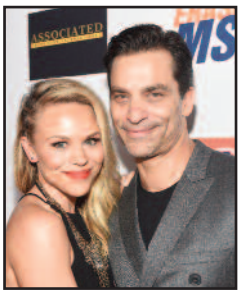

James Tupper and Anne Heche


Teri Hausman and Guests


Dana Davis and Nancy Davis


David and Laura McKenzie


Sherry Corday and Debbie Eaton


Julie Solomon and Johnathon Schaech


Burton Morris, Nancy Davis and Matthew Lubkeman


Melina Glavas, Debora Mendelson, Claudia Curry Hill, Betsy Walton


Jimmy and Debbie Lustig and Shereen and Michael Pollack


Francesca Eastwood and Frances Fisher

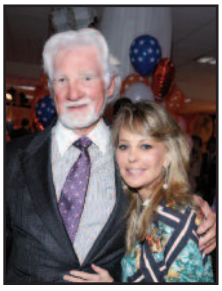

Lisa Cohen, Nancy Davis and Jimmy Cohen


Mary Jo Hausman and Stacy Bartlett Renshaw


Nancy Davis and Twitterati


Alice Evans and Ioan Gruffudd


Cammy MacMillan, Jourdan Block, Iana Lebovici and Caroline Friedman


Tawny and Jerry Sanders


Tommy Hilfiger, Rita Ora, Dee Hilfiger


Paris Hilton


Nancy Davis, Kenny, Isabella and Mariella Rickel


Lynn Palmer, Carolyn Hinsey and Tracy Danza

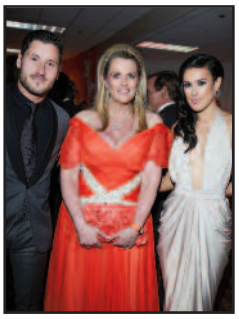

Kathy and Rick Hilton


Rita Ora


Tonya and Dave Winfield


Vipin Sareen, Nicky, Rebecca and CC Mitchell


Kelly, Sharon and Jack Osbourne


Val Chmerkovskiy, Nancy Davis, Rumer Willis


Jennifer Alewelt, Elizabeth and David Anderson


Anne Heche and Nancy Davis


Tommy Hilfiger and Richard Hilfiger


Nancy Davis and Brandon Davis


Marivi Garcia, Sheri Disney, Ann Lopez, Judy Angel Salvaria, Melissa Beyeler, Silvia Baker, Sherry Corday, Debbie Eaton, Beth Preece, Robin Correll


Eric Valdez and Mathew Clarke


Adam Scott Roberts and Lyndi Hirsch


Camille Guaty


Mark Locks and Guest


Paul Berman, Alexis Berman, Charlotte Broadbent, and Joshua Rosenzweig


Nancy Davis and Jason Davis


Keltie Knight and Duane Mah


Mark and Shainaz Burg and Guest


Nicole Whitmore and Cindy Locke


Susan and Mark Bozek


Jordyn, Abby and Stacey Katz


Debbie Mendelson, Dan Silverberg, Patrick Gaydos, Jennifer Gaydos

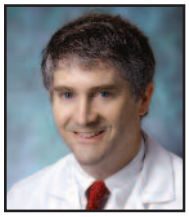
highlights from the lab (continued from page 7)

gy within the inner nuclear layer at baseline in MS seems to predict the subsequent development of relapses, new T2 lesions on MRI, new contrast enhancing lesions on MRI, as well as disability progression. Using OCT, we have also recently shown that not only do the RNFL and GCIP thin following acute optic neuritis, but that there might also be transient increases in INL and ONL thickness immediately following acute optic neuritis. Such changes may have implications for helping to further our understanding of the efects of inflammation in MS.

The above studies not only inform us of potential outcome measures for tracking neurodegeneration in MS and monitoring neuroproetective/neurorestorative effects, but also further our understanding of the pathobiological underpinnings of these processes in MS. The support of the Race to Erase MS is essential for us to continue our work to unravel the basis of neurodegeneration in MS, to identify novel outcome measures for measuring neurodegeneration, and ultimately for the successful identification of effective neuroprotective and neurorestorative agents.

Pilot Study Summaries 2015-2016:
Support for a focused research project that is novel and will unravel new aspects of MS.

Peter Calabresi, M.D.
Professor of Neurology at the Johns Hopkins School of Medicine,
Director of the Johns Hopkins Multiple Sclerosis (MS) Center



Immune Cells and MS:
Recently, it has been described that in multiple sclerosis (MS) that immune cells may collect in the coating around the brain and spinal cord called the meninges. These clusters of immune cells are called lymphoid follicles and are more common in progressive forms of MS. The follicles are associated with increased demyelination of the grey matter of the brain and likely play a role in worsening of the disease. Similar follicles have been noted in a mouse model of MS called experimental allergic encephalomyelits (EAE). We have found that it is possible to detect these follicles in mice on MRI of the brain with contrast. The follicles consist of B and T-cells and are associated with demyelination in the underlying brain tissue. In this pilot project we plan to study the mechanism of formation of these follicles and to test whether an antibody targeting B-cells injected into the mouse brain will eliminate these follicles and prevent damage to myelin. Determining how these follicles are formed and cause demyelination could lead to new avenues of treatment for patients with progressive MS.

Dennis Bourdette, M.D.
Chair and Roy and Eulalia Swank Research Professor
Department of Neurology
Oregon Health & Science University

Stimulating natural repair in MS:
Inflammation in MS destroys myelin, the insulation around nerves, and also destroys the cells that make myelin, the oligodendrocytes. Remyelination, a naturally occurring repair process in MS, is part of how people with MS recover after a relapse. However, as time goes on, this natural repair process begins to fail. With support from the Race to Erase MS, OHSU researchers are developing a drug that is intended to stimulate this natural remyelination to help reverse disability and hope to fully arrest MS.

Thyroid hormone is a key stimulus for myelination as the brain develops. It also is important for remyelination. For remyelination to occur, oligodendrocyte precursor cells (OPCs) need to mature into oligodendrocytes that can make new myelin. While there are lots of OPCs in MS lesions, for unclear reasons these OPCs do not mature normally. A key stimulus for OPC maturation is thyroid hormone. Using high doses of thyroid hormone could stimulate the OPCs to mature — but excessive amounts of thyroid hormone can cause serious side effects.

OHSU investigators, Drs. Dennis Bourdette and Thomas Scanlan, at OHSU, and colleagues have shown that thyroid hormone is highly effective at stimulating OPC maturation. To avoid the side effects of thyroid hormone, they are making drugs, called thyromimetics, that can induce the positive effects of thyroid hormone while avoiding the side effects. The first of these thyromimetic drugs, called GC-1, stimulates OPCs in culture and accelerates remyelination in a mouse model of MS. The research team at OHSU is currently synthesizing a large number of thyromimetic drugs. The goal is to create thyromimetic drugs that enter the brain readily and are highly effective at promoting remyelination. The team hopes to have a drug ready for further development for the treatment of MS within the next 12 months.

Roopali Gandhi, PhD
Assistant Professor of Neurology
Head of MS Biomarker Group
Brigham and Women's Hospital
Harvard Medical School

MicroRNAs (miRNAs):
miRNAs are single stranded, small non-coding RNA molecules that regulate gene expression and are implicated in many autoimmune diseases including Multiple Sclerosis (MS). In addition to its expression in cells, stable expression of miRNAs has been reported in circulating biofluids like plasma, serum and urine. Circulating miRNAs are proposed as disease biomarkers both for autoimmune diseases and cancers. Recent studies have shown that ex- miRNAs serve as versatile intercellular communication vehicles. These findings have established the novel concept that miRNAs have potential, not only to serve as putative biomarkers, but also to influence disease progression. We found that expression of miRNA "miR-92a" is linked to disease stage & response to treatment in MS and is differentially expressed in MS

patient's blood compared to the healthy controls. With support from Race To Erase MS, we begun to look into the function of miR92a in immune cells from MS patients and healthy controls. We believe that the result from this study will increase our understanding of how miRNA could be linked to MS immunology and if miR-92a regulates genes and immune cell function differently in progressive MS patients compared to relapsing remitting MS patients and controls. This understanding might introduce new pathways related to MS, disease progression and novel treatment options in MS.

Jack H. Simon MD, Ph.D.
Professor Radiology, Oregon Health & Science University (OHSU)

Characterization of the Leptomeningeal Inflammatory Pathology in MS by Ferumoxytol (USPIO) MRI:

By The North American Imaging in MS (NAIMS) Cooperative. Leptomeningeal pathology with inflammatory cell infiltration has been detected in biopsy specimens of the brain and has been implicated in theories of persistent disease and is possibly related to the mechanism of disease progression. Recently, D. Reich and colleagues at NIH have documented regions of perivascular leptomeningeal enhancement by in vivo gadolinium (Gd)-contrast enhanced magnetic resonance imaging (MRI) in MS in about 25% of cases. Importantly, in two cases with pre and post-mortem correlation, the NIH group has shown that enhancing nodules identified by MRI correspond to regions of cellular inflammation (T cells, B cells, macrophages) and underlying cortical demyelination, a finding concordant with the neuropathology literature. This proposal specifically addresses a novel, potentially more specific and sensitive cellular imaging approach to leptomeningeal disease in MS based on MRI after ferumoxytol infusion. Ferumoxytol is an ultrasmall superparamagnetic iron oxide (USPIO), which can accumulate within inflammatory cells and possibly within migrating macrophages potentially marking the leptomeningeal sites. In this study, the comparative sensitivity of gadolinium enhancement and ferumoxytol enhancement will be determined using optimized 7T MRI. The use of ferumoxytol MRI to detect leptomeningeal disease in MS would be a novel but logical research application given the histopathology recently described for leptomeningeal disease in MS, with the potential to lead to development of an important biomarker for disease, disease progression and as a treatment outcome measure.

Scott W. Wong, Ph.D.Professor, Department of Molecular Microbiology and Immunology Oregon Health & Science University

Viral etiology associated with MS:

Epidemiologists have long thought that a virus causes MS because the characteristics of MS are consistent with viral infection, these include 1) nature of the demyelination and damage to the nervous system, 2) intermittent deficiency in sensation, movement, cognition and 3) duration. Unfortunately, no single virus has been definitively identified as the causative agent, arguing the case that either an unknown virus is associated with induction of MS or that multiple viruses and other unknown variables including environmental factors may also contribute to the disease. The evidence supporting a virus causing MS is strong, and efforts to find an infectious agent that is potentially associated with MS must be vigorously pursued if we are to discover what causes MS. With support from Race to Erase MS, we have begun to investigate whether there is a novel virus associated with MS. These studies are guided by our findings that a novel herpesvirus is associated with an MS-like encephalomyelitis that clinically resembles MS in monkeys. This class of viruses has co-evolved with their hosts, suggesting that a related and yet to be identified virus is present in humans. In fact, we have serological evidence to support this, as a higher percentage of MS patients have antibodies that recognize the monkey herpes virus compared to normal healthy controls. These results strongly suggest that a novel human herpesvirus distinct from the known human herpesviruses is present in the human population and may be associated with the induction of MS. To prove this, we will perform exhaustive studies on samples acquired from MS patients and healthy controls to acquire molecular evidence that this novel virus exists. We will then test if this virus is the etiological agent associated with MS.

Christoph Juchem, Ph.D.
Assistant Professor in Diagnostic Radiology and Neurology, Yale University School of Medicine

Elevated lipid and protein concentrations :

Elevated lipid and protein concentrations have been reported with magnetic resonance spectroscopy (MRS) in acute MS lesions and are speculated to reflect de- and remyelination processes. Despite early reports, the role of lipids and proteins in tissue injury and repair has not been widely considered, at least in part due to methodological obstacles to quantify them in vivo. MRS allows the non-invasive description of biochemical processes associated to MS pathology directly in the in vivo brain. However, the quantification of lipids and proteins is severely complicated by low measurement sensitivity and spectral overlap. Both effects can be mitigated by stronger fields of the magnetic resonance scanner. In our research, we apply a novel MRS technique at ultra-high 7 Tesla magnetic field that allows us to characterize lipids and proteins directly in the brain, including MS lesions and normal appearing white matter tissue. The objective of this research is to investigate the metabolism of lipids and proteins in acute (contrast-enhancing) MS lesions over time. More specifically, we will comprehensively assess and follow the biochemistry of

(LAB continued on Page 12)

(LAB continued from Page 11)

non-symptomatic lesions over a 6 months period including lesion onset, development and fading. The longitudinal characterization of lipids and proteins is expected to shed light on their role in MS tissue injury and repair processes - information that is not accessible otherwise in vivo. This pilot research represents an important step towards validating the novel MRS method for MS research and towards establishing it as a tool for clinical diagnostics and monitoring. It is our expectation that successful completion of the proposed research will allow to correlate clinical prognosis to injury severity and the potential for repair, e.g. remyelination, in future studies.

Eve Kelland, Ph.D
Assistant Professor of Neurology
Keck School of Medicine
University of Southern California

Assessment of a novel neuroprotective agent, angiotensin 1-7, in models of Multiple Sclerosis:

Multiple Sclerosis (MS) is a disabling disease in which the immune system attacks the nervous system. The cause of MS is unknown and no treatments as yet exist that can cure the disease or least, stop disability progression. New therapies are desperately needed that not only control the imbalanced immune system but also protect the nervous system and make it permissible to repair. With the support of the Race to Erase MS we plan to examine the effects of a novel peptide, called angiotensin 1-7, that is thought to help protect the nervous system from damage and may even help to facilitate repair. To help us to understand the role of this peptide in neuroprotection and repair in the context of MS we will assess the therapeutic effects of this peptide when it is delivered directly into the nervous system in two animal models of MS: one model will be immune driven and the other will be via a toxin that specifically kills the myelin ensheathing cells known as oligodendrocytes. In this study we will also assess how this peptide directly protects cells of the nervous system such as nerve cells and oligodendrocytes, as well as the brain repairing stem cells, oligodendrocyte progenitor cells, from inflammatory damage. The pharmaceutical formulation of angiotensin 1-7 (TXA127) is in clinical trials and has already undergone extensive safety profiling for other indications, therefore application to the clinic for the treatment of MS could be very rapid. Support from the Race to Erase MS for this research will help us to determine whether angiotensin 1-7 is an ideal treatment for MS in order to alleviate the symptoms and progression of MS.

Center Without Walls
Summary Research Update

During this past year, the Foundation has supported five very promising junior scientists to help them establish their cutting-edge research. In July 2015, the Foundation has started to support 2 new junior scientists, Dr. Stephen Fancy (UCSF) and Dr. Naila Makhani (Yale). Dr. Fancy is studying the remyelination potential related to migration of immature myelin forming cells along vessel scaffolds. Dr. Makhani is studying MRI features of early phases of pediatric MS in addition to immune characteristics in these patients. During the coming year, the Foundation will continue supporting Dr. Oleg Butovsky (Harvard) for his second year and Drs. Shiv Saida (Hopkins) and Jennifer Graves (UCSF) for their third year of young investigator award. Dr. Butovsky is studying immune cells, aka microglia to understand better their very important role in MS such as regulation of inflammation and damage in the brain, but also to unravel the mechanisms that control these cells. Dr. Shiv Saidha at Hopkins utilizes a novel technology called optic coherence tomography (OCT), which can very precisely measure the health of nerves in the back of the eye called the retina and study how they degenerate. He examines whether OCT measures of nerve damage in the eye can predict nerve damage in other parts of the brain. Dr. Graves is studying factors in the environment that together with genes contribute to higher frequency of MS relapses. This is a novel area of research as the interactions between genes and environment in MS are poorly understood and some may be amenable to treatment. The newly developed MRI project called the North American Imaging in MS (NAIMS) is a collaborative effort led by Drs. Sicotte at Cedar-Sinai and Jiwon Ho at Hopkins including MRI experts from the Race to Erase MS Center Without Walls and other North American colleagues. The aim of this project is to develop a standardized MS imaging protocol across multiple centers in North America and sensitive, reliable imaging based surrogates for disease progression that will accelerate MS research.

Six pilot research grants were also awarded this year to highly exciting and innovative research projects. Dr. Calabresi at Hopkins will study the mechanism of formation of immune cell clusters in the coating of the brain and spinal cord and test whether an antibody targeting B-cells injected into the mouse brain will eliminate these follicles and prevent damage to myelin. Dr. Wong at OHSU will screen saliva and blood samples from patient with MS and healthy donors for the presence of viruses to determine whether some of those viruses may be associated with the risk to develop MS. Dr. Bourdette from OHSU will study a thyroid hormone-like drug in mice to evaluate if it stimulates the repair of myelin and helps the recovery of brain function. Dr. Kelland at USC will examine the nervous system protective effects of a novel peptide, called angiotensin 1-7, that is a part of a pathway regulating blood pressure in animal models of MS. Dr. Gandhi at Harvard will study microRNAs which are small non-coding molecules that regulate gene expression and protein synthesis in fundamental biological processes including inflammation. Finally, Dr. Juchem at Yale will study proteins and lipids in the brain of patients with MS using high field imaging to understand better the processes involved in tissue injury and repair in the brain. Several times a year, all these talented scientists and doctors share the results of their research at Race to Erase MS symposia which foster the blossoming of promising junior researchers in order to one day cure MS.

(VISUAL continued from Page 5)

some authors have suggested that increased INL thickness in the absence of visible microcystoid changes may enable detection of the same process. As such, increased INL thickness at baseline has been shown to predict disability progression, relapses, and the development of new lesions on MRI. Therefore, while RNLF and in particular GCIP thickness may be useful for tracking MS patients from a neurodegenerative perspective, the identification of quantitative and qualitative abnormalities of the INL and ONL may also be highly informative and have predictive clinical utility.

The above findings suggest the eye may provide a window into the global MS disease process. Combined with its numerous positive attributes, this makes OCT a potential tool for monitoring MS patients. RNFL and GCIP thicknesses correlate with whole brain volume in MS, while INL thickness seems to correlate mostly with lesion volume.

These findings suggest that one may glean information regarding various processes operative in the MS disease process from different compartments of the retina. More recently, it has also been shown that thinning of the GCIP in the eye mirrors reduction in whole brain volumes in MS over time. Estimates of RNFL and GCIP thicknesses derived from OCT, a technique that can be quickly performed in the office, provide insight regarding high and low contrast visual function, global disability, and brain tissue loss. GCIP thickness may be superior to RNFL thickness for the purpose of tracking neurodegeneration in MS, with rates of GCIP thinning being faster in patients exhibiting disease activity. Quantitative and qualitative abnormalities of the deeper retinal layers, and in particular the INL, may have clinical utility for predicting disease activity and disability progression. Collectively, these factors underscore the importance of visual system investigation in MS.

IVIG Postpartum

By Lisa Arnold

I was diagnosed with MS in the summer of 2008. I had just had my second child and was filled with all the joy of a new mother. My family came to visit and while having a girl's lunch with my mom and sister I suddenly went numb on the right side of my face. Needless to say I was alarmed! I was convinced I must have been allergic to the mushroom ravioli I had been enjoying…

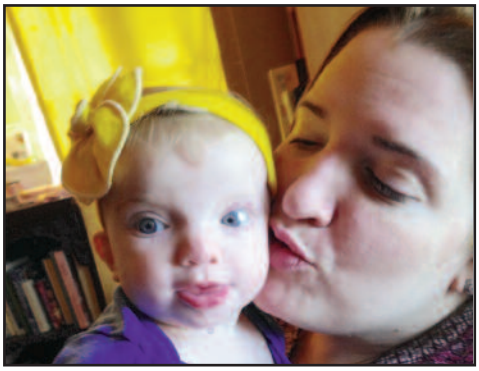
Unfortunately, it wasn't the ravioli. Instead, it was MS. I spent the first year convincing myself I must have been misdiagnosed. Life had resumed, I was young, in my twenties and clearly healthy. I had been a college athlete, a part of my university's sailing program and had a list of accomplishment's I felt worthy of sparing me from this diagnosis. I was wrong.

Several years after my initial diagnosis I began a free fall, intermittent issues with my legs, arms and hands, were paired with severe cognitive problems, vision trouble and nerve pain; not to mention a slew of "unmentionable" MS side effects. Stunned, I thought perhaps I was on an episode of "Punk'd," and that any day a television host would jump out and give me back the old body I had depended on for the first part of my life. Instead I became a fighter. At almost 34 I found myself still fighting, I had tried oral steroids and IV steroids. I tried interferon medications and the latest oral MS treatments. Moreover, elusive prescriptions like Athcar and Ampyra and the latest books on lifestyle changes, MS diets, natural doctors, acupuncturists, homeopaths and more. Nothing hit the brakes on my disease progression until I decided to have a baby.

At one of my regular neurologist appointments I brought up my ticking biological clock. With two wonderful boys our family still did not feel complete, but given my failing health and inability to walk it seemed like a dream. There my neurologist sat with his infinite knowledge and kind eyes, explaining to me why I wasn't "medically stable" and now was NOT a good time to expand my family. That said he went in to all the obstacles that were between me and a precious baby… While my health would likely improve during pregnancy I would almost certainly relapse after giving birth and may have to have IVIG in the hospital post-partum. These magic words caused me to glaze over, smile at my husband from ear to ear and ignore the rest of the appointment. IVIG was like a ghost I had been chasing, always out of reach thanks to my insurance company.

I was interested, my husband was interested and 2 months later I was pregnant. My pregnancy was a dream, the usual morning sickness was a blessing next to the abatement of some of my regular MS symptoms. For me pregnancy was an MS vacation. After a natural birth I was transferred from labor and delivery and my OB/GYN ordered two rounds of IVIG and I was placed in the maternal fetal care unit to be monitored. There were complications but in the end, the IVIG has been a blessing. Years of trying to find help with my MS symptoms were answered in part with the birth of our daughter Gigi.

Things have come full circle, today I accept my diagnosis of MS, but I am a MS fighter. The disease that was triggered by the birth of our son has started a new chapter with the birth of our daughter. I hope this information will help other expectant mothers obtain their best possible medical outcome, as we all Race to Erase MS together.



Lisa and her daughter

Now I have MS

By Tamara Feldman

I thought getting diagnosed would change my life dramatically. It has, and it hasn't. I had so many big ideas I tried to rush into but what I have learned is that everything takes time. When I first started feeling strange sensations, I think I knew exactly what was going on deep down. From the first moment. It still took me over a year to get to the doctor though. Maybe the symptoms weren't clear enough at first, or I was just in denial. Who knows what the real first symptoms were anyway. Was it the few times the top of my head went numb? I went to the doctor then and we agreed it was an anxiety attack symptom that just lasted a few weeks. Or the time a couple years later when I felt warm water flowing down my hip and thigh while hiking, and there was nothing there. That one lasted a week or so also. The big one though was Lhermitte's sign. Every time I played guitar I would shock myself really hard from my fingertips to my neck somehow. I reconfigured wires and tried not to touch my guitar and the mic or the metal table at the same time. The shocks were really painful. Finally, I was sitting on my couch with a friend and I laughed really hard, one of those laughs where you lose control of your body and throw your head forward and back in pure joy. That was the strongest shock yet and I wasn't touching anything. That was when I knew there was something going on for sure. But I still didn't go to the doctor. I thought of my sister having MS and thought I must be commiserating with her. I didn't want to be a copycat or a hypochondriac. Besides, it was probably just a pinched nerve. I went to a masseuse. I went to a chiropractor. I went to an acupuncturist. As the acupuncturist was putting needles in my skin, some places I got huge painful shocks and some there was nothing. She stuck one in my right hip, the one that had felt wet a couple years before. "Nothing?" she said. "nothing?" She put it in deeper. "Still Nothing?" "Nope" Deeper. "Now? Down to the toes? Nothing?" "Nope" "There's something really wrong. You need a Neurologist. I can't help you." I stood up, getting zinged all over the place, and got light headed. Mei Mei, the Chinese Doctor, called for her husband Dr. Wu. He rushed in and shoved a spoonful of sugar in my mouth and told me to swallow. It was dry and gross and I felt like Mary Poppins gone wrong with needles. He said to sit until I got very warm. I went to my car and burst into tears. It was all too much. When I finally called a primary care physician and told them my symptoms they thought I was having a stroke and told me to call 911. I laughed and said no, I'm fine, just please refer me to a neurologist.

Once at the neurologist I calmly told him that I had Multiple Sclerosis. My sister had it for 10 years and now I have it. I have Lhermitte's sign and paresthesia. I told him of the treatments I had heard of and was interested in trying. The "natural" route. No drugs. The Swank Diet. He thought I was well informed and probably was used to neurotically obsessed new patients. He asked me to please promise not to try the bee stings, that one is really dangerous. He wanted to do a spinal tap to make sure, but not to worry, he would do it old school. He said I didn't need to go to the hospital where everyone just gets more sick and they strap you down, he would do it in his office. He said his hands shake, but he can take a pill for that, don't worry. I came back in a couple days for the spinal tap. No big deal he said. No need to rest after, just drink some coffee and be on your way. If it were only that simple. A nerve was hit, I almost jumped off the table as electricity shot down my leg. I was chastised for moving because it could cause me to be paralyzed. If anyone knows how to not jump when a nerve in your spine is hit by a needle, please let me know. Maybe that's why they strap you down at the hospital. The jolt got blood in the spinal fluid and more than normal was drawn. Walking out of the building I was hit by the weirdest and most intense head pain I've ever felt and it brought me to my knees. I had lost a lot of spinal fluid and the needle used was "old school" so it was bigger than the needles used in the hospital now. The hole didn't seal and I was leaking spinal fluid for about a week. Thus started a journey of spinal headache, steroids, paralyzing anxiety attacks, and being shot with an epi-pen while I felt like my veins were on fire. Obviously after all this, I wanted a second opinion. I went to a different Neurologist. His comment… "well, you have MS. You'll be in a wheelchair in probably 10 years. You might have longer but it's not a matter of if, it's when. You should get pregnant." What!

There is so much more but I feel like it's getting a little dramatic. I'll wait to tell you about getting my teeth pulled because maybe root canals cause MS. What a journey that was/is. Or the fasting and supplements, road trips to different alternative doctors, hours of internet research on South American or African medicines, etc. Here's the real deal though. I just had my 1 year anniversary. I looked at myself and saw that the actual MS hasn't done that much to me. I have had a couple attacks and they were scary for sure. When I run my legs start tingling and if I don't stop it can get super intense until I have to sit down and wait for it to pass. However, what I put myself through in my fear and trying to control was actually worse. Thankfully I'm starting to learn my lesson. I'm calming down. I'm getting focused. I'm relaxing, meditating. I'm plotting a goal to work towards and a middle of the road regimen that I can stick to. I am taking medicine that has some side effects but they aren't intolerable, just a little inconvenient. I'm being healthy, trying to get the right amount of exercise, taking the supplements that are known to help but not going overboard. It's still going to be a long road but if I have learned anything in the last year, it's to not let the fear determine my actions and to just slow down. As someone who loves to go real fast, that's quite a lesson.

Tamara Feldman

Multiple Scerlosis and Children

By Dr. Jennifer Graves, UCSF Adult and Pediatric MS Centers

One of the most common questions I hear from parents, is what caused MS in my child? They want to know if there was anything they could have done differently. I can immediately alleviate this fear and concern and tell them there is not a single cause of MS but likely complex sets of factors have come together to cause the onset of the disease. The thermostat for MS risk is set by genetics, but genes account for less than fifty percent of MS risk. Through research within the U.S. Network of Pediatric MS Centers, we are in pursuit of the environmental factors that contribute to developing MS and how these factors may interact with the genes that are associated with the disease.

Looking for environmental risk factors involves gathering data about the child's early life environment and measuring factors in the blood. There is strong evidence for low vitamin D levels, smoking and exposure to Epstein Barr virus (virus that causes mono) as risk factors for multiple sclerosis. As environmental factors, these are potentially modifiable. We are also investigating new potential risk factors for MS in children, including the effects of events around the time of pregnancy and toxic exposures in the environment.

Some of the risk factors for MS also affect the severity of the disease. Smoking cessation and avoidance of passive smoke exposure may improve disability outcomes. Low vitamin D levels, which have been associated with increased relapses and accrual of disability, may be corrected with supplementation. Many families ask if low vitamin D can be corrected by spending more time outdoors. While this may help some, most patients require vitamin supplementation to significantly increase their average blood levels.

Other factors that may be helpful to improve quality of life with pediatric MS include maintaining a healthy, well balanced diet, regular exercise, and remaining active in school activities. Unless there are specific restrictions from an individual's doctor, kids with MS can participate in sports, dance and other extracurricular activities. For some girls and boys with significant fatigue, they may need to practice energy conservation techniques, such as doing the most important activities of the day in the morning or planning rest or naps before performances or evening events. Most experience improved energy and mood from exercise and enjoy the social benefits of continuing their extracurricular activities.

There is not an accurate estimate of how many children 10 years of age and under have been diagnosed with MS, but we can say that 20% of all pediatric MS patients at our center are 10 or younger. In the US we think there are at least 5,000 pediatric MS patients so it would mean about 1,000 children that are 10 years old or younger. The youngest of our patients on treatment at our center are 4 years old. Most of the adult approved therapies are given to children with MS and in the patients that are 10 years and younger we tend to use the medications we know are safe like and interferon and Copaxone.

The search continues for the cause of MS, with significant achievement and momentum in unraveling the genetic and environmental factors involved. In the meantime, families can support the health of children with MS with modification of lifestyle factors as above and if needed vitamin D supplementation.

(ECTRIMS continued from Page 1)

will be FDA-approved for relapsing MS and for primary progressive MS, meaning that ocrelizumab could be the first FDA-approved drug for primary progressive MS.

News about Existing FDA-Approved MS Therapies:

Results of the alemtuzumab extension studies were announced. In the main studies (CARE-MS I and CARE-MS II), most patients received alemtuzumab only at baseline and year 1. Despite the fact that most did not receive any more medication after year 1, the treatment effects were still maintained at year 5 (very few relapses and a much slower rate of brain atrophy).

Investigational Therapies in Earlier Phase of Development:

Anti-LINGO-1 is an antibody that may help with the repair process following an MS attack. It is currently being tested in a Phase 2 study of patients with acute optic neuritis. The subgroup analyses were announced in Barcelona; the benefit of anti-LINGO-1 appeared greater in patients older than 33 years old, those who received treatment early (<25 days from onset) and those with more severe optic neuritis.

Phenytoin is a commonly used antiseizure drug that may be neuroprotective according to a Phase 2 trial in acute optic neuritis. Patients treated with phenytoin appeared to have better optic nerve thickness after an episode of optic neuritis.

Biotin is a vitamin that is currently being studied in progressive MS. In a study being conducted in France, biotin was effective at delaying or preventing disability progression compared to placebo.

Minocycline is a tetracycline antibiotic that may reduce the risk of developing MS in patients who experience their first clinical demyelinating symptoms according to the results of a Phase 3 study performed in Canada. These results are intriguing, as minocycline is a safe, low cost, widely available medication.

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Pay tribute to friends and family for special occasions and help us win the RACE to Erase MS!

We will send a card with your personal message to your friends and family on your behalf. It is a thoughtful gesture for any occasion.

See the self mailer included in this newsletter or go online www.erasems.org, or gifts can be made by calling our office at (310) 440-4842

SAVE THE DATE

23rd Race to Erase MS Gala "Love to Erase MS"

Friday, April 15, 2016
The Beverly Hilton

For more information please call 310-440-4842 or visit www.erasems.org

2016 MS Forum and Expo
Saturday, April 16th
The Beverly Hilton

Free to the public. Open forum with our top MS research scientists speaking on the latest advancements in multiple sclerosis research.


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Dr. Robert Katz, Kenny Rickel and Dr. Peter Waldstein


Loni Anderson


Melina Glavas and Guest


Pam and Howard Levine


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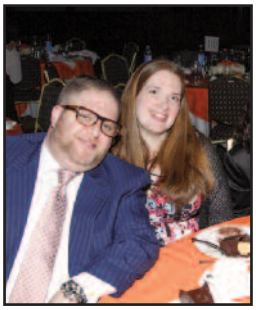
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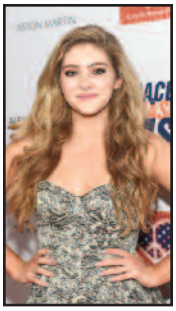
Jamie Winkler and Nancy Davis


Aston Martin

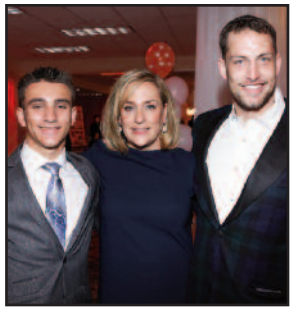

Dr. Daniel Reich, Dr. Ellen Mowry and Dr. Peter Calabresi


Nicki Yassini and Guests and Allison Marmur


Christine Devine and Guests


Willow Shields


Shayne Henderson, Dana Davis, Guest and Nick Chavez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Mariella and Isabella Davis and Guests


Steven Cojocaru and Guest


Bobby Margolis and Guests


Carmen Electra


Anthony Arvizu, William Fleming, Martin Benitez, Carl Freedman


Lydia Hearst


Bryan Carter and Kacie and Matthew Flowers


Martyn Lawerence Bullard and Guest


Rumer Willis, Keltie Knight, Lea Thompson


Kathy Hilton and Camille Grammer

Aly and Aj Michalka


Nancy Davis, Tommy Hilfiger, Rita Ora, Ne-Yo, Mariella Davis, Ava Dyborn, Remy Navarre


Guest with Reza Farahan

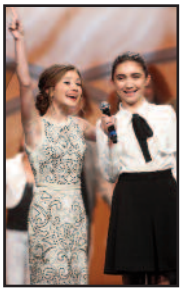

Cindy Farber, Debbie Lustig, Jennifer Gardner, Carol Mizel and Guest


Mariella Davis, Francesca Capaldi, Isabella Davis


Burton and Sara Morris, Brooke and Michael Lande


Ken Todd and Lisa Vanderpump

Pamela Johananoff and Guest, Bonnie Pfeifer, Nancy Goldstein


G. Hannelius and Rowan Blanchard


Adi and Erica Weinberg

Katherine McNamara


Brandy Navarre, Angela Dyborn, Nicki Yassini


Cheryl Burke


Matty B. with Kids


Ryan and Carmel Geise, Travis and Erin Holowach


Nancy Davis and Kurt Knutsson


Russell Anmuth, Jordan Cohen and Carin Morris

Kevin Farley


Arlene and Barry Hirschfeld, Shereen and Michael Pollack


Richard and Karen Levine


Edina and Dave Dwork, Dr. Michael and Rhonda Wadzinski


Dana Davis and Guests


Ben and Robert Knepper