Dear Friends,

A cure or treatment for HSP & PLS came oh so much closer as we aggressively stepped forward in 2016 with very decisive and effective research. We could not have done it without you. Our sincere and heartfelt thanks go out to the community at large and to all our generous supporters for making this possible. Let me tell you about just some of the highlights of the progress that is taking place with the research we are sponsoring.

Dr. Benjamin Cravatt, Professor and Chair of the Department of Chemical Physiology at the Scripps Research Institute in La Jolla, CA has been working on Brain Lipid Metabolism in HSP. To do this, he and his associates have developed new technologies that bridge the fields of chemistry and biology. He ascribes to the notion that it is the activity of an enzyme and not its abundance that dictates its roll in cell physiology and pathology and so has introduced a set of proteomic technologies called activity-based protein profiling (ABPP) to measure how metabolic signaling networks are regulated in vivo. He has focused this study on the HSP related enzyme DDHD2 which is a principal brain triglyceride lipase. They have discovered that the form of HSP with DDHD2 enzyme has an unknown fat molecule principally in the brain. They identified them as triglycerides - a major component of stored fat in the body. Using both light and electron microscopy they determined that droplets of triglyceride-rich fat are present in the neurons of DDHD2-knockout mice, in several brain regions, but are not present in normal mice. DDHD2 normally breaks down triglycerides and its inactivity allows triglycerides to build up. Therapies to counter these effects are being developed, including the possible use of diacylglycerol transferase inhibitors which reduce the natural production of triglycerides.

Dr. Mauricio Delgado-Ayala, MD and Dr. Jonathan Rios, PhD are neurologists at Texas Scottish Rite Hospital for Children in Dallas TX. Whole-exome sequencing has led to several cases where alternative diagnoses have developed. Also, genetic variants have been discovered in many patients, the significance of which has yet to be determined. Preliminary data indicates that they have discovered a new HSP gene as well as a dominant disease causing variant. They are in the process of using zebrafish experiments to confirm this. This whole-exome sequencing approach has opened a world of possible discoveries as more and more single mutations in recessive HSP genes are being discovered.

Dr. John Fink, MD, Professor of Neurology, University of Michigan Medical Center, Ann Arbor, MI, and the Medical Advisor for The Spastic Paraplegia Foundation, has been working on “New Treatment Strategies for Primary Lateral Sclerosis.” He is assessing the effect of a new compound for the treatment of PLS and HSP and a few patients have reported a significant improvement. He has been defining the nature of gait disturbance and its pattern of progression. A detailed neurologic evaluation system has been developed and subjects have been studied serially for 3 years. Interestingly, although assessed neurologic parameters may change, the patient’s overall ability to walk may remain stable. This suggests that functional parameters like timed walking may be more valuable outcomes by which to judge natural history. What’s more, Dr. Fink is trying to determine the reason that many people with HSP and PLS reach a point of clinical plateau. This is thought to be caused by either a change in the rate of neurodegeneration or the effect of neurocompensatory changes or both. He is working to observe the mechanisms that cause neurocompensation and test them in an animal model.

Dr. Andrew Grierson, PhD, Sheffield Institute for Translational Neuroscience, University of Sheffield, UK, is working on a Gene Therapy for the treatment of HSP. Dr. Grierson is identifying the molecular mechanisms underlying the regulation of axonal transport in HSP and PLS. He is also performing a preclinical assessment of histone deacetylase 6 (HDAC6) inhibition as a therapy for HSP and performing a preclinical assessment of spastin gene replacement therapy as a treatment for HSP. He is identifying the molecular mechanisms underlying regulation of mitochondrial axonal transport in HSP patients. Novel vertebrate models of HSP in zebrafish
Dr. Melissa M. Rolls, PhD, Assistant professor, Biochemistry and Molecular Biology, Penn State University, has shown that the HSP-related proteins, Spastin and Atlastin promote axon regeneration at the growing axon tip of motor neurons (the ends of the axon furthest from the cell bodies in the brain). She has shown that a mutation in the Spastin Gene, SPG4 that causes the most common kind of HSP, shuts down the process by which axons, the parts of the nerve cell that are responsible for sending signals to other cells, regrow themselves after being cut or damaged. To be able to survive, nerve cells need to be resilient and be able to repair damage by growing new axons. Microtubules, the intracellular “highways” along which basic building blocks are transported, need to be able to be rebuilt in the event of necessary repair. In order to grow a new part of a nerve, raw materials will be needed and the microtubule highways will need to be organized to take the new materials to the site of growth. Dr. Rolls used a fruit fly model to determine that a disruption in the SPG4 gene caused the axons to have no regrowth. They found that an impaired spastin gene affected only the axon furthest from the cell bodies in the brain). She has used a combined MRI/PET scanner to discover that patients with PLS had neuro-inflammation in the motor cortex and corticospinal tracts. The same patients will be examined again in one year of her SPF sponsored fellowship focusing on PLS research. The majority of her time was spent working on a primary neuroimaging project. Dr. Paganoni, a primary expert on the use of olfactory (nasally derived) stem cells to treat or help find treatments for injuries or disorders. It was in honor of his work, finding a treatment to cure spinal cord injury, termed by some as the scientific equivalent of a moon landing, that he was elected as Australian of the Year for 2017. He has been working for years on finding a treatment for HSP, using these same olfactory HSP stem cells in HSP corticospinal neurons. SPG4 HSP patients have a mutation in SPAST, a gene that encodes Spastin, a microtubule severing protein. Patients with SPG4 have 50% of the spastin, 50% of the acetylated atubulin and 150% of the stathmin, a microtubule destabilizing enzyme compared to people without HSP (control). Patients with SPG4 compensate for reduced spastin with stathmin but this makes the microtubules less stable and alters organelle trafficking. The results with human HSP neurons are positive, confirming previous findings that two possible drugs, Nocaspine and Epothilone D, increase the acetylated atubulin levels in SPG4 patient’s cells to normal. He has also performed an HSP mouse study which determined that more measurement and analysis is necessary and that HSP in mice are not necessarily a good model for HSP in human beings. We are now in the process of planning to make application to the regulatory authorities for conducting clinical drug trials for SPAST HSP.

Dr. Sabrina Paganoni, PhD, MD, Physiatrist and Assistant Professor at Harvard Medical School has spent the first year of her SPF sponsored fellowship focusing on PLS research. The majority of her time was spent working on a primary neuroimaging project. Dr. Paganoni, a primary expert on the use of olfactory (nasally derived) stem cells to treat or help find treatments for injuries or disorders. It was in honor of his work, finding a treatment to cure spinal cord injury, termed by some as the scientific equivalent of a moon landing, that he was elected as Australian of the Year for 2017. He has been working for years on finding a treatment for HSP, using these same olfactory HSP stem cells in HSP corticospinal neurons. SPG4 HSP patients have a mutation in SPAST, a gene that encodes Spastin, a microtubule severing protein. Patients with SPG4 have 50% of the spastin, 50% of the acetylated atubulin and 150% of the stathmin, a microtubule destabilizing enzyme compared to people without HSP (control). Patients with SPG4 compensate for reduced spastin with stathmin but this makes the microtubules less stable and alters organelle trafficking. The results with human HSP neurons are positive, confirming previous findings that two possible drugs, Nocaspine and Epothilone D, increase the acetylated atubulin levels in SPG4 patient’s cells to normal. He has also performed an HSP mouse study which determined that more measurement and analysis is necessary and that HSP in mice are not necessarily a good model for HSP in human beings. We are now in the process of planning to make application to the regulatory authorities for conducting clinical drug trials for SPAST HSP.

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Dr. Sabrina Paganoni, PhD, MD, Physiatrist and Assistant Professor at Harvard Medical School has spent the first year of her SPF sponsored fellowship focusing on PLS research. The majority of her time was spent working on a primary neuroimaging project. Dr. Paganoni established a broad portfolio of PLS centered projects ranging from molecular mechanisms to epidemiology and clinical research. She used a combined MRI/PET scanner to discover that patients with PLS had neuro-inflammation in the motor cortex and corticospinal tracts. The same patients will be examined again in one year to determine if this neuro-inflammation is stable or changes over time. Additional imaging analysis showed that areas of increased PBR28 uptake correspond to areas of cortical thinning, suggesting an anatomical link between neuro-inflammation and loss of upper motor neurons. Results from this study could greatly accelerate proof of concept clinical trials of drugs that target inflammation in PLS. Dr. Paganoni, as a member of NEALS, has been instrumental in spearheading a proposal to set up the largest available retrospective PLS registry. 21 NEALS centers across four countries volunteered to contribute data. Data from 148 patients with PLS has already been collected and data collection should be complete this year. Dr. Paganoni has worked with Dr. Mary Kay Floeter, MD, PhD, at NIH to analyze a unique cohort of 56 PLS patients who were followed prospectively at NIH between 2000 and 2015. The manuscript, that is in preparation, will show the rate of PLS disease progression. Dr. Paganoni is also working with Dr. Hiroshi Mitsumoto, MD at Columbia University on a new project that is intended to develop a new outcome measure of disease progression that is specific to PLS. This study should be completed in 2017. She is also working with Dr. Dale Lange, MD at the Hospital for Special Surgery in New York, NY to work as a site for the Ampyra PLS clinical trial. Enrollment will be open at 3 sites including Michigan General Hospital.

Dr. Melissa M. Rolls, PhD, Assistant professor, Biochemistry and Molecular Biology, Penn State University, has shown that the HSP-related proteins, Spastin and Atlastin promote axon regeneration at the growing axon tip of motor neurons (the ends of the axon furthest from the cell bodies in the brain). She has shown that a mutation in the Spastin Gene, SPG4 that causes the most common kind of HSP, shuts down the process by which axons, the parts of the nerve cell that are responsible for sending signals to other cells, regrow themselves after being cut or damaged. To be able to survive, nerve cells need to be resilient and be able to repair damage by growing new axons. Microtubules, the intracellular “highways” along which basic building blocks are transported, need to be able to be rebuilt in the event of necessary repair. In order to grow a new part of a nerve, raw materials will be needed and the microtubule highways will need to be organized to take the new materials to the site of growth. Dr. Rolls used a fruit fly model to determine that a disruption in the SPG4 gene caused the axons to have no regrowth. They found that an impaired spastin gene affected only the axon’s regrowth, that is, the gene did not seem to play a role in the development stage when axons were being assembled for the first time. They also found that the dendrites, the parts of the neuron that receive information from other cells and from the outside world continued to function and repair themselves normally. Dr. Rolls is now looking into whether other HSP disease genes may play a role in nerve-cell regeneration.

Dr. Holger Sondermann, PhD, Associate Professor, Department of Molecular Medicine, Cornell Univ., Albany NY, is studying a mechanistic understanding and targeted therapies in HSP type SPG3A in a 3 generation
Amish family in Lancaster County PA. SPG3A deals with a defect in the protein that creates Atlastin. Atlastin is necessary to catalyze membrane fusion and required to form the Endoplasmic Reticulum network. Dr. Sondermann’s lab is interested in deciphering the basic regulatory principles in signal transduction networks on a molecular level, focusing on growth factor receptor signaling. They are studying the role of scaffolding proteins, using a combination of X-ray crystallography, biophysical and cellular approaches. His attempt to understand the architecture, mode of action and regulation of these processes will elucidate how cells respond to various inputs producing distinct outputs by using a limited set of molecules and hopefully determine a method to counteract the effect of the SPG3A gene.

Dr. Evan C. Reid, PhD, Department of Medical Genetics, University of Cambridge, UK is trying to define a pathway that could be targeted to increase age at onset of HSP.” The most frequent cause of HSP is an autosomal dominant mutation in SPAST SPG4. Families with SPAST mutations show significant variations in the age of onset of HSP symptoms. Dr. Reid has found an explanation for some of this variation. Families with spastin mutations that also have a deletion of an adjacent gene, DPY30, have a significantly younger age at onset. Dr. Reid has found that regulating contacts between the endoplasmic reticulum and endosomes, spastin controls traffic of digestive enzymes to the lysosome (the cellular dustbin). This results in enlarged and abnormal lysosomes. This abnormal lysosomal appearance is found in tissue culture cell lines, in cortical neurons (and their axons). He has also found this abnormal lysosomal appearance in at least 4 other genetic subtypes of HSP. They have thus identified a unifying pathway culminating in lysosomal dysfunction for many of the HSP proteins. This is a major advance in the HSP field. Their next step will be to test whether this lysosomal disfunction can be targeted as a viable therapeutic strategy.

Dr. Rebecca Schule, PhD, University of Tuebingen, Tuebingen, Germany, is working to establish a global collaborative network (Alliance for Treatment in HSP & PLS) between major national HSP/PLS initiatives, including networks across Canada, Europe, and the US to create a global HSP/PLS Registry that connects existing registries under one umbrella to create a ‘Clinical Trial ready’ infrastructure. Dr. Schule has further created an Alliance of 14 world renowned scientists across the globe, specializing in HSP and PLS research, in the hopes of establishing data harmonization. All participants have agreed that sharing of de-identified clinical data among researchers is essential and agreed to contribute their data collections to a joint Alliance registry. So far, the alliance has completed several research projects that include: an SPG5 study in which they have collected a large cohort of more than 30 SPG5 patients and have defined the mutational and phenotypic spectrum of SPG5 as well as examined the correlation of disease severity and progression with oxysterol concentrations. They have demonstrated that oxysterols are neurotoxic at concentrations found in SPG5 patients and have begun a Clinical Trial of Statin Treatment. They are currently working with Dr. Alexandra Durr in Paris, France on a project determining how mutations in the SPG7 gene cause a wide range of neurodegenerative diseases ranging from HSP to cerebellar ataxia. They are studying the evolution of SPG7 over time and are determining the relationship between the mutation and the resulting phenotype. Dr. Rebecca Schule has performed a natural history study in SPG4 including data on more than 200 SPG4 cases. In this study she will determine the progression rate of SPG4 as well as factors influencing disease severity and progression. She is currently collecting a cohort of SPG4 cases to perform a large-scale genetic screening for SPG4 modifiers to better understand the variability in age of onset and severity of symptoms.

Our most recently sponsored research for which there has not been enough time for their development are:

Anjon Audhya PhD, Associate Professor, Biomolecular Chemistry, University of Wisconsin- Madison, “Identifying underlying causes of hereditary spastic paraplegia and creating avenues toward the development of new therapeutic interventions.”

Emanuele Panza, PhD, Assistant Professor in Medical Genetics, Department of Medical and Surgical Sciences, University of Bologna Italy, “Understanding Hereditary Spastic Paraplegia: Lessons and therapeutic options from a rare form of Hereditary Spastic Paraplegia.”

Henry Houlden, PhD, Professor of Neurology and Neurogenetics, The National Hospital for Neurology and UCL Institute for Neurology, London and Viorica Chilban, M.D., Research Fellow, Department of Molecular Neuroscience, UCL Institute of Neurology, London, UK, “Development of the UK clinical research network and biobank in HSP.”

Hiroshi Mitsumoto, MD, Wesley J Howe professor of Neurology, Columbia University, Development of a PLS-specific clinical rating scale, capable of evaluating the clinical functional state of patients with PLS in a multi-site study.”

Thanks so much to everyone who has helped fund this research that will lead to treatment and eventually to a cure for HSP and PLS.

Sincerely,

Frank Davis
SPF President
The Spastic Paraplegia Foundation, Inc. (‘‘SPF’’) is a not-for-profit corporation that is a United States & Canada, volunteer-run, health organization dedicated to funding cutting-edge scientific research to discover the causes and cures for Hereditary Spastic Paraplegia and Primary Lateral Sclerosis, and to diminishing suffering by education and support.

The SPF home corporate office is located at 1605 Goulart Place, Fremont, CA 94539-7241. A copy of our latest annual report or financial statement may be obtained by writing to this same address or calling 877-773-4483.

Financial Activities

Where your dollars go

<table>
<thead>
<tr>
<th>REVENUE</th>
<th>2016</th>
<th>2015</th>
<th>2014</th>
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<td>Convention Fees &amp; Products</td>
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<tr>
<td>Investment Income</td>
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<td>202</td>
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<td><strong>Total Support and Revenue</strong></td>
<td>$646,521</td>
<td>$565,634</td>
<td>$669,113</td>
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DIRECT EXPENSES

| Fundraising                    | 5,043   | 24,279  | 23,747  |
| Management and Administration  | 53,134  | 82,112  | 40,028  |
| Program Expense               | 60,338  | 42,002  | 22,278  |
| **Total Expenses**             | $114,015| $148,393| $86,053 |

GRANTS PLEDGED

| $697,250 |
| $615,955 |
| $280,000 |

NET ASSETS

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<th>(as of December 31)</th>
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<td>$321,192</td>
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<td>$487,686</td>
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<td>$686,327</td>
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The Board of Directors continues to maximize your donations as 92% of each dollar raised supports the foundation’s mission of research, information and support. The majority of Program Expenses are the costs of holding our Annual Conference which brings hundreds of people with HSP & PLS together with the world’s leading HSP & PLS scientists for knowledge, support and fellowship. These costs are mostly offset by Program Fees and Corporate Sponsorships.

Management and Administration which are valuable and necessary foundation expenses are services which are donated to the foundation. Legal, accounting, income tax preparation, management and medical grant review services are all provided at zero cost but are recorded for tax purposes. Fundraising expense in 2015 was the cost of managing a golf tournament which in 2016 was supported by donations.

We are pleased to report that a total of $697,250 has been approved for research funding for 2017. This is made possible by the continued support of our generous donors. 2016 was highlighted by the Match My Gift program. Over $389,000 was raised as the result of anonymous donor matches. Our heartfelt Thank You goes out to them.
Would you like more information about us?

Research to discover the causes and cures for Hereditary Spastic Paraplegia and Primary Lateral Sclerosis, the world's leading HSP & PLS scientists for knowledge, support and fellowship. These costs are mostly offset by Program Fees and Corporate Sponsorships.

Where your dollars go

My Gift program. Over $389,000 was raised as the result of anonymous donor matches. Our fundraising which are donated to the foundation. Legal, accounting, income tax preparation, management and administration 8% Management and Administration 92% Mission

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