Toward a CURE
A RESEARCH UPDATE
Huntington's Disease Society of America
WINTER 2012

A Message from Louise Vetter, HDSA CEO

Welcome to a new issue of *Toward a Cure*. We are pleased to welcome George Yohrling, PhD to the staff of HDSA as the new Director of Medical and Scientific Affairs. George will be leading efforts to launch the new HDSA Research Program and re-establishing the former Medical and Scientific Affairs Committee (MSAC) as the new HDSA Scientific Advisory Board. George comes to us from our partner, CHDI, after an exhaustive search. You can read more about George and his vision for the new HDSA Research Fellowship program on page 3.

Also in this issue is a thought-provoking article by Michael Hayden and colleagues at the University of British Columbia examining the rate of incidence of HD in North America and in British Columbia, in particular.

The research indicates that rates of HD may indeed be increasing, an observation shared by our HD families with researchers for several years. The original rate of incidence for HD, used by the NIH, HDSA and other health organizations, was based upon several epidemiologic studies conducted in the United States from 1945-1980. Now, more than 30 years after those benchmark studies, Dr. Hayden and his colleagues postulate that the increase in rate of incidence may be due to the introduction of new HD alleles – that is individuals who have no family history of HD. You can read more about Dr. Hayden's ground breaking research on page 4.

Also included in this issue is an article about zinc finger proteins which are found naturally in all living beings. Zinc finger proteins are special because they can recognize and bind to specific DNA sequences thus making them a potentially potent tool in the development of a therapy for HD. Read more on page 6.

And in this issue is news of an $18.9 million grant from the California Institute of Regenerative Medicine (CIRM) to the University of California, Davis School of Medicine, and HDSA's own Dr. Vicki Wheelock and her colleague Dr. Jan Nolta.

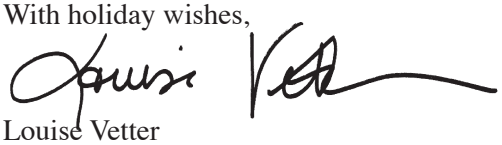
In this study Wheelock and Nolta will study the use of a novel therapy to treat HD. Dr. Nolta's team will establish safety and efficacy studies for the potential therapy using animal models of HD, and in a parallel study, Dr. Wheelock will enroll early stage HD patients in an observational study that will establish clinical, laboratory and neuro-imaging baselines prior to a planned clinical study of the new therapy. You may read more about these exciting research projects on page 8.

As we welcome 2013, HDSA plans to release its request for proposals for the very first grants that will be issued under the new HDSA Research Fellowship program. This new and innovative approach merges our two benchmark programs, our HDSA Centers of Excellence and our Fellowship program, in a new and collaborative way that will bring basic research literally from the laboratory bench to the patient's bedside.

We are elated that the launch of this new scientific endeavor will coincide with the 20th Anniversary of the discovery of the gene that causes HD. Throughout the next year, HDSA will be commemorating this important moment in history. Be sure to check HDSA.org regularly for special events that will celebrate this historic milestone.

In closing, I wish each of you a happy and healthy holiday season. You are the most important part of our mission. Everything we do to improve the lives of people with HD begins and ends with you - our HD family members, friends and community. Let us work together to make 2013 a truly memorable year for all of us.

With holiday wishes,



Louise Vetter

Toward a CURE

The purpose of *Toward a Cure* is to provide information and opinion and to relay items of interest to individuals with Huntington's Disease and their families, healthcare professionals and interested friends and supporters.

The Huntington's Disease Society of America (HDSA) is a national not-for-profit organization. The Society is dedicated to eradicating Huntington's disease by promoting and supporting HD research; to helping families cope with the problems presented by HD; and to educating the public and healthcare professionals about HD.

HDSA is a member of the National Voluntary Health Agencies, the National Health Council, the National Organization of Rare Disorders, the International Huntington Association, the Alliance for Genetic Support Groups and the Independent Sector.

The Huntington's Disease Society of America meets all nine standards of the National Charities Information Bureau

Amaryllis Season is here!

Amaryllis bulbs are among the easiest bulbs to grow indoors and the most rewarding. These premium Dutch bulbs need minimal attention but you will be rewarded with spectacular blooms to brighten up the dark winter days. Planting Amaryllis and watching them grow is a fun activity for the entire family.

Amaryllis are available in individual kits for gift giving or cases of solid colors and assorted.

The COMPLETE KIT will contain everything you need to have your own spectacular Amaryllis this winter including: one 26/28 cm Amaryllis Bulb, decorative pot, professional growing medium, planting and aftercare instructions.

Each kit costs only $10.00.

Amaryllis make the perfect gift that can be enjoyed for many weeks.

Some gift ideas include:

- Thanksgiving
- Christmas
- Chanukah
- Housewarming
- Get Well
- Birthday
- Gifts for friends, teachers, co-workers

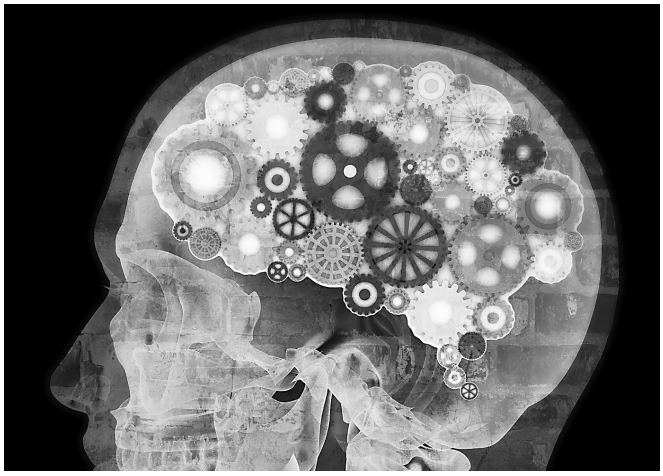
All proceeds support HDSA's fight to improve the lives of people affected by HD and their families.

Amaryllis can be purchased from your local chapter or on line at **www.hdsa.org.**

In This Issue

From the Bench:
The Future of HD Research at HDSA

From the Desk of George Yohrling, PhD; HDSA Director of Medical and Scientific Affairs



The opportunity I have been given to represent the families and faces of Huntington's disease as the Director of Medical and Scientific Affairs at HDSA is both a privilege and an honor. I am excited about the prospects of bringing a renewed focus to research back to HDSA. Before joining HDSA, I spent nearly 4 years at the CHDI Foundation in Princeton, NJ where I was the Director of Target and Pathway Assessment. One of my jobs was to perform the research necessary to build confidence around new molecular targets whose function or activity, if altered, could beneficially impact HD.

HDSA has a proud history of pioneering research, particularly the work originating from the 16 Coalition for the Cure researchers that were supported by you, the families. These scientists have played critical roles in HD research ranging from identifying the gene that causes HD to the identification of biological mechanisms that become dysfunctional during the course of the disease. While the Coalition as it was previously known is gone, HDSA leadership has heard loud and clear that the desire for the Society to remain involved in research is as strong as ever. Over the coming months, my number one priority here at HDSA will be to execute on the goals set forth in the 2012-2016 Strategic Plan to develop a new HDSA-driven research initiative.

The research to be supported by HDSA must meet a number of criteria. First, the data/outcomes must be impactful by better informing clinicians and scientists on the design of future clinical trials. For example, this could be achieved by identifying a new biomarker in the blood of patients that could be monitored to assess efficacy of a novel therapeutic. Second, the work must not replicate research being done at other institutions such as NIH and CHDI. To ensure this, we will improve lines of communication between all of the major institutions supporting HD research. Finally, the new research must involve the incorporation of a unique strength of HDSA, the 21 Centers of Excellence across the United States. This is an incredible resource not available to other HD organizations and one which HDSA must capitalize. While incredibly innovative work is ongoing in labs across the world to develop better pre-clinical animal models of HD, there is no argument that the best and most physiologically relevant observations that will guide us in the hunt for effective therapies to slow the progression of HD are those that will be observed in HD patients. I strongly believe that observations from patients at the Centers of Excellence can significantly impact the future of HD research.

I would like to briefly outline the next steps we will take to make this new research initiative at HDSA a reality. By the end of 2012, it is my goal to establish a world-class Scientific Advisory Board (SAB) for HDSA. The SAB members should represent a diverse area of expertise ranging from the academic, medical, non-profit and pharmaceutical/biotech arenas. The SAB will establish regular meetings and be instrumental in helping to craft the final directives for the HDSA Fellowship Program. By early 2013, we will publish a Request for Proposal (RFP) to research investigators to solicit ideas from scientists on ways to interrogate human biology questions in HD patients at the COEs. It is envisioned that COE investigators and the fellow's principal investigator will work together to mentor the young scientists. The SAB will review all proposals on their merit and determine the best that meet the criteria mentioned above. All grant awardees will be invited to future HDSA Conventions to present the results of their research. Despite limited resources, I have no doubt that HDSA can sponsor impactful research, while also creating the next wave of dedicated HD researchers.

I hope you share my excitement for the future of HD research at HDSA. I will continue to update you on our progress and look forward to meeting with you all at the 2013 HDSA Convention in Jacksonville.

Clinical Research **In HD**

Clinical trials and observational studies remain the gold standard for proving the efficacy and safety of new therapies that might provide potential benefit for people affected by HD. Volunteers are always needed for these studies. Please consider enrolling in ***HDTrials.org*** which asks only for an email address (which you can create just for HDTrials.org) and zip code but could yield a potential trial or study site in your neighborhood.

HDTrials.org provides clinical trial sponsors with the number of potential participants based upon zip codes. Sponsors use this information to determine where the study should be conducted as rapid recruitment means a less costly trial or study. When a trial or study is scheduled in your area, only then are you contacted by ***HDTrials.org*** with the contact information you need to connect with the trial or study investigators. ***HDTrials.org*** never releases any information because it only has an email and zip code. It is completely anonymous.

Below is a list of the Trials and Studies ***currently recruiting*** participants.

Multi-Site Observational Studies: no therapy is being tested.

- ENROLL-HD: an international registry of clinical information about HD. There are currently 9 sites recruiting participants in the U.S. with another 7 slated to begin shortly. More sites will be added in the near future but these are currently recruiting:

  University of Tennessee Health Science Center (Memphis)

  Columbia University (New York)*

  Colorado Neurological Institute (Englewood, CO)*

  Hereditary Neurological Disease Center (Wichita, KS)

  University of California (Los Angeles)*

  Wake Forest University School of Medicine (Winston-Salem, NC)

  University of Alabama (Birmingham)*

  University of California (San Diego)*

  Kansas University Medical Center (Kansas City, KS)

  **HDSA Center of Excellence*

  For continuing updates on ENROLL-HD, please visit HDSA at ***www.hdsa.org***.

- PREDICT 2.0: an ongoing study of the early stages of HD. Currently 14 sites in U.S. recruiting.

Single Site Observational Studies:

- Evaluation of {123I} MNI-420 and SPECT as a Marker of the Adenosine A2a Receptor in PD, HD and Healthy Participants. Recruiting at the Institute of Neurodegenerative Disorders, CT.
- A Biospecimen and Clinical Data Study on Patients with Alzheimer's, Multiple Sclerosis, Parkinson's and Huntington's for Drug and Biomarker Discovery. Recruiting at Sanguine Biosciences, CA
- A PET Brain Imaging Study of mGluR5 in subjects with Neuropsychiatric Conditions (FPEB). Recruiting at the Institute of Neurodegenerative Disorders, CT.

Multi-Site Clinical Trials: involves a therapy or intervention

- CREST-E (Creatine Safety, Tolerability and Efficacy in Huntington's Disease). Recruiting at 36 sites in U.S.
- Reach2HD (Effect of PBT2 in Patients with Early to Mid Stage HD). Recruiting at 15 sites in U.S.
- SEN0014196 (An Open Label Food Effect Study with SEN0014196 in Subjects with HD). Recruiting at 7 sites in U.S.

Single Site Clinical Trials:

- Impact of Xenazine (Tetrabenazine) on Gait and Functional Ability in Individuals with HD. Recruiting at the Ohio State University, OH.

For more information on these and new trials and studies, please visit www.hdsa.org/research or www.clinicaltrials.gov.

The Changing Prevalence of Huntington's Disease

By: Chris Kay, Emily Fisher, Michael R Hayden

How common is Huntington's disease? At present, the disorder is estimated to afflict 1 in 10,000 individuals of European ancestry. But recent reports suggest that HD prevalence may be increasing. Our laboratory is investigating the underlying genetic and demographic basis of HD, in order to illuminate the molecular origins of the disease and why it may be growing more common.

First Modern North American Prevalence Estimate

In British Columbia, we recently completed the first comprehensive assessment of HD epidemiology in North America since the introduction of genetic testing. Our study has produced the first prevalence estimate in British Columbia and the first ever assessment of population at risk in North America. The results indicate that nearly 1 in 7,000 people are afflicted with HD in the province, and likely a greater number are pre-symptomatic for the disease. Our prevalence estimate reveals an increase over the last Canadian assessment, which took place four decades ago in the Prairies. Given similar reported increases of HD prevalence in Australia and the United Kingdom, a rise appears to be common across Western populations.

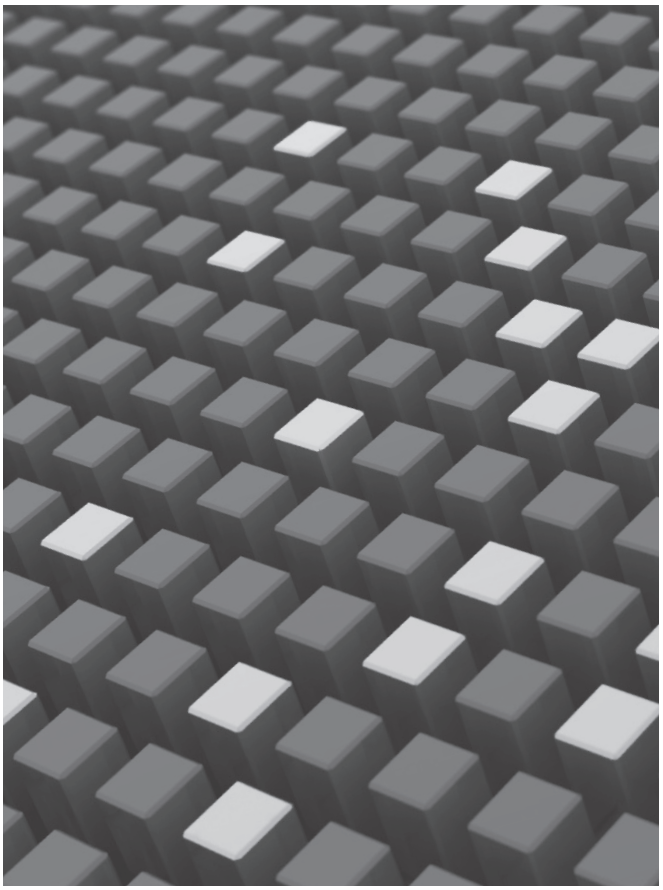
New Mutations from Intermediate Alleles

What might be causing this reported increase in prevalence? One possibility is the introduction of new HD alleles. Up to 25% of individuals diagnosed with HD lack informative family history of the disease. Many such cases have been shown to represent new mutations, where neither parent carries the causative HD allele, and yet it is found in the affected son or daughter. What is the true contribution of these new HD alleles to the prevalence of the disease?

New mutations for HD are known to originate from a pool of pre-mutations that do not cause the disease, called intermediate alleles (IAs). To determine the contribution of new mutations to HD prevalence, we assessed the frequency and mutability of IAs in the general population. Among individuals in a large sample of the general population, nearly 1 in 17 carry an IA and hence the risk of passing a new mutation for HD to their children. Taken together with our revised instability estimates across the IA range, we estimate a substantially higher new mutation rate than previous reports. We are now investigating IA frequencies in populations with documented concentrations of HD, in order to determine whether higher IA frequencies contribute to higher HD prevalence.

Aging Populations

A second and more immediate contribution to rising HD prevalence are aging populations around the world. Today, approximately 11% of the world population is over 60 years of age. By 2050, this figure is expected to double to 22%. Since HD is an adult-onset disorder, aging populations are expected to result in a higher frequency of the disease.



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The Changing Prevalence **of Huntington Disease**

(Continued from Page 4)

The length of the CAG repeat that causes HD is inversely associated with the average age of symptom onset. At shorter CAG repeat lengths, onset tends to occur later in life. At longer CAG repeats, onset tends to be earlier. Across a small range of repeats at the lower end of the affected HD range, from 36 to 39 repeats long, average age of onset occurs beyond normal life expectancy in the developed world. Only some carriers of these alleles will develop HD, usually very late in life, while the rest will die from other common causes such as cancer and heart disease. However, as life expectancy increases and mortality from other disease decreases, more carriers are expected to become symptomatic for HD. Using our knowledge about HD onset, we looked to quantify the magnitude of these age-related increases in HD prevalence.

Joining average age of onset in the lower HD range with projected changes in population structure, we have been able to estimate the growth of HD cases in coming decades and the age distribution of future cases. Our results paint a changing picture of HD, where the disease becomes both more common overall and a disease more common to old age. By 2050, the majority HD cases are likely to occur in individuals over the age of 60, often with more mild symptoms, resulting in a new profile of the disease.

Our knowledge of rising HD prevalence makes the need for treatment and research all the more urgent.

About the authors:

Chris Kay is a graduate student in Michael Hayden's laboratory, studying the genetic basis of HD prevalence around the world.

Emily Fisher is a recent graduate of Michael Hayden's laboratory. Emily conducted the first province-wide assessment of HD epidemiology in British Columbia and is currently developing a provincial HD register.

Michael R. Hayden is a Killam University Professor of Medical Genetics at the University of British Columbia and a Canada Chair in Human Genetics. He is the world's most cited author on Huntington's disease.

HDSA Clinical Trials **Diplomat Program**

Volunteer participation in clinical research studies is key to developing new treatments for any disease. Study sponsors have noted the correlation between slow recruitment and the high cost of conducting a trial or study. Delays and additional costs can deter many pharmaceutical companies from entering the orphan or rare disease drug discovery pipeline.

To address these concerns, HDSA has developed a program that links previous family study participants with HD educational events in order to educate the community, from a personal perspective, about what it means to volunteer as a participant in a clinical (interventional) or observational (non interventional) study.

HDSA Clinical Trial Diplomats are trained by HDSA to talk about their personal clinical research experiences. They provide information and answer questions about clinical trials in small group settings such as support group meetings or as panelists at educational events that are offered by HDSA Chapters, Affiliates and Centers of Excellence throughout the year.

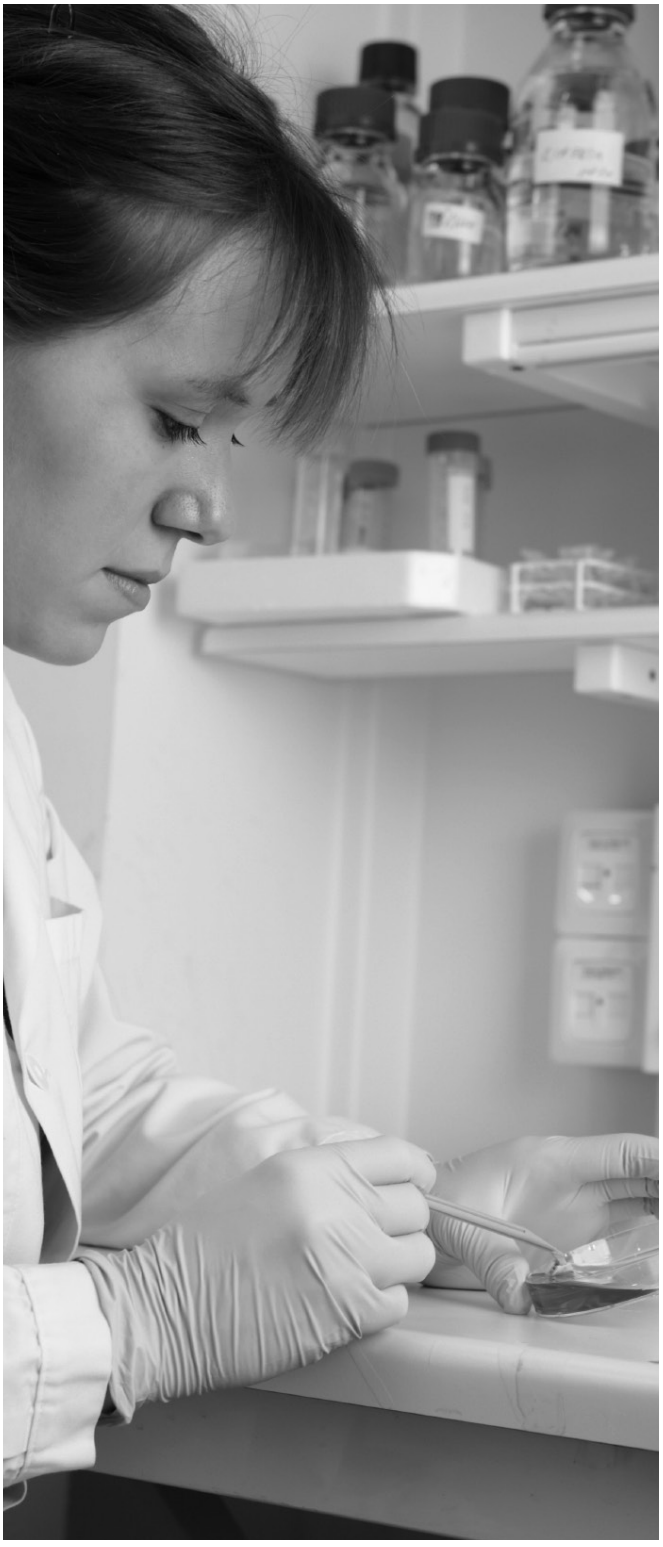
HDSA Clinical Trial Diplomats believe that clinical trials are important for HD drug discovery and that the HD community will play as important a role in finding new treatments as they were in finding the gene 20 years ago.

If you would like to learn more about being an HDSA Clinical Trial Diplomat, please contact Jane Kogan at HDSA. Call 800-345-HDSA extension 226 or email ***Jkogan@hdsa.org.***

Zinc Finger Proteins:
Novel technology to target huntingtin DNA

By: George Yohrling, PhD, Director of Scientific and Medical Affairs

There is no better validated protein target to combat Huntington's disease than that of huntingtin (htt). Diversified small molecule and non-conventional strategies, such as gene therapy, are being developed to lower the expression of the mutant protein that causes HD. Over the past few years a great deal of excitement has been generated by the pre-clinical results that use non-conventional approaches to specifically silence mutant htt. The two major types of htt silencing are antisense oligonucleotides (ASOs) and short-interfering RNA (siRNA). Both work at the level of the htt mRNA and interfere with the ability of the cell to make the huntingtin protein (reviewed in Matsui and Corey, 2012).



There is another, complementary approach that is also being explored that can target the htt DNA. Researchers have found that they can engineer zinc finger proteins (ZFPs) that can bind the expanded CAG repeats in the htt DNA sequence with greater efficacy than the shorter CAG repeats found in the normal htt allele. ZFPs are small proteins that can be experimentally linked together. ZFPs are identified by their unique three-dimensional protein structure. Each ZFP has an α-helical and β-sheet structure that contain amino acid residues that coordinate the binding of metals such as zinc. Other proteins, such as transcriptional activators or repressors can also be fused to the array of engineered ZFPs. The ZFPs can bind near the promoter of any gene and be used to either increase or decrease the transcription of the targeted gene.

Recently published results from Garriga-Canut et al and work from Sangamo Biosciences have both used ZFPs to selectively target the mutant htt DNA in animal and neuronal models of HD respectively.

In the work from Garriga-Canut et al, they tested their most promising ZFPs in the R6/2 mouse. The R6/2 mouse is a widely used model of HD that expresses just a short fragment of the human htt protein (~3%). The ZFPs were delivered to the striatum of R6/2 mice with an adeno-associated virus (AAV) to ensure widespread delivery to the striatum. With a number of different ZFPs, they observed a selective repression of mutant htt RNA, reductions in htt protein aggregates in

the striatum, and improvements in the motor deficits in the R6/2 mice. They also found that the ZFPs designed to target expanded CAG repeats in the htt gene did not repress other important CAG-containing genes. These results provide important proof-of-concept that ZFPs can be delivered to an HD animal model and can effectively repress molecular and behavioral phenotypes.

Similar work from Sangamo Biosciences, in collaboration with Shire AG and the CHDI Foundation, was reported by Steve Zhang, PhD during a scientific session dedicated to experimental models of HD at the 2012 Society for Neuroscience meeting in New Orleans. Sangamo is a biotechnology company that focuses on the development of DNA-binding proteins as therapeutics to regulate gene expression for diseases such as HD. Sangamo reported they have identified both allele-specific and bi-allelic repressors for the htt gene. Dr. Zhang showed that when Sangamo's ZFPs were tested at doses as low as 10 ng in neurons from R6/2 mice they were able to significantly lower mutant htt levels while not affecting the wild-type htt. Sangamo also investigated whether their htt-directed ZFPs silenced other CAG-containing genes. They found that the stanniocalcin 1 (STC1) gene was repressed by htt ZFPs. The next step for Sangamo is to test their ZFPs in animal models of HD. Intrastriatal injections AAV-ZPFs are planned to test for safety, spread and efficacy. It will be important to see if STC1 or other CAG-containing genes remain affected when ZFPs are tested *in vivo*.

The use of ZFPs to target the mutant htt gene holds great promise. While much work remains to be done in pre-clinical animal models of HD, this new therapeutic modality could serve as another weapon in our fight against HD.

References:

Garriga-Canut M, Agustín-Pavón C, Herrmann F, Sánchez A, Dierssen M, Fillat C, Isalan M. Synthetic zinc finger repressors reduce mutant huntingtin expression in the brain of R6/2 mice. Proc Natl Acad Sci U S A. 2012 Oct 10.

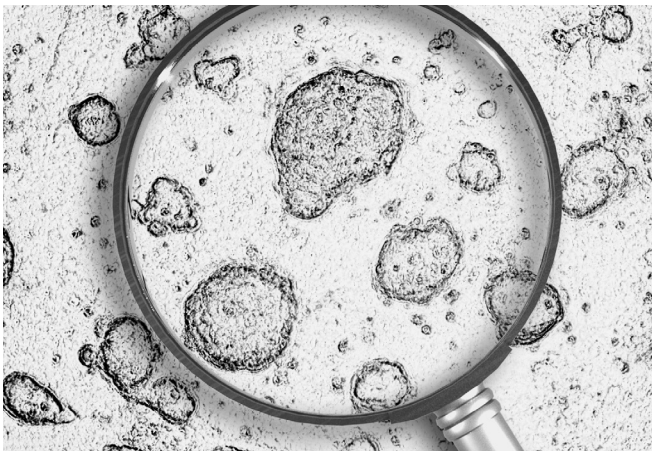
Matsui M, Corey DR. Allele-selective inhibition of trinucleotide repeat genes. Drug Discov Today. 2012 May;17(9-10):443-50.

UC Davis **Stem Cell Grant for HD**

By Vicki Wheelock, MD, Director, HDSA Center of Excellence at University of California, Davis Medical Center

On July 27, 2012, HD family members celebrated when the California Institute of Regenerative Medicine approved an $18.9 million grant to fund research at UC Davis School of Medicine that aims to engineer adult stem cells to help treat patients with Huntington's disease.

The project is entitled, "MSC engineered to produce BDNF for the treatment of Huntington's disease." The principle investigator is Dr. Vicki Wheelock, Director of the HDSA Center of Excellence at UC Davis, and the co-principle investigator is Dr. Jan Nolta, Director of the Stem Cell Program and Institute for Regenerative Cures at UC Davis and editor of the journal *Stem Cells*. An internationally respected stem cell researcher for decades, Dr. Nolta began to focus her work on HD



after attending the Celebration of Hope dinner in 2008. Former HDSA northern California chapter president Judy Roberson inspired Dr. Nolta and has worked tirelessly to advocate for stem cell research funding to help fight HD.

Mesenchymal stem cells (MSC) are adult stem cells that arise from bone marrow. With established safety in phase I-III human clinical trials, MSC have special properties that make them ideal candidates for HD therapy. Dr. Nolta and her team have demonstrated that MSC are remarkably effective delivery vehicles, moving robustly through tissue and infusing therapeutic molecules into each damaged cell that they contact. MSC can be transferred from one donor to the next without tissue matching because they shelter themselves from the immune system. Implantation of MSC into the brains of HD animal models has significant neurorestorative effects and is safe.

The medium spiny neurons in an area of the brain called the striatum are vulnerable to cell death in patients with HD. The striatum is involved in the control of movement, thinking and behavior, leading to the progressive, disabling symptoms. The mutant huntingtin protein dramatically decreases brain-derived neurotrophic factor (BDNF), a factor needed by striatal neurons to remain alive and healthy. Rescue of striatal neurons is an excellent target for HD treatment.

In this study, Dr. Wheelock and Dr. Nolta propose a novel therapy to treat HD: implantation of MSC engineered to secrete BDNF (MSC/BDNF) to rescue at-risk neurons in the striatum. Dr. Nolta's team has demonstrated the safe and effective production of engineered molecules from human MSC for at least 18 months in pre-clinical animal studies. They have shown with collaborator Dr. Gary Dunbar, at Central Michigan University, that MSC/BDNF delivery can have significant effects on reducing disease progression in HD rodent models. The CIRM grant will enable the research team to carry out further safety and effectiveness studies of MSC/BDNF in Years 1 and 2 using novel animal models. The team must win FDA approval before going forward with the planned Phase I cellular therapy trial of MSC/BDNF brain infusion in patients, with the goal of restoring the health of neurons that have been damaged by the mutant htt protein.

In parallel with the pre-clinical animal model testing, Dr. Wheelock's team plans to enroll a group of up to 40 early-stage HD patients in a pre-cellular therapy observational study. Participants in the observational study will be candidates for the future Phase 1 cellular therapy trial. The goal of the observational study will be to establish a clinical, laboratory and neuroimaging baseline and to determine the rate of change in these parameters for each patient prior to the enrollment in the planned cellular therapy trial. Following FDA approval, a 2-year open-label, prospective Phase I study, evaluating the safety of MSC and MSC/BDNF cellular therapy, will be conducted. All patients will receive intrastriatal implantation of cells. The first arm of the study will be bilateral striatal implantation of MSC, with three additional arms to evaluate MSC/BDNF in a dose escalation study at increasing cell doses: 5, 10 and 20 million cells per side. Patients will be treated and evaluated for a 12 month follow-up period.

UC Davis Stem Cell Grant for HD

(Continued from Page 9)

The primary objectives of the proposed Phase I clinical trial will be safety and dose determination. Secondary endpoints for potential effectiveness will be closely evaluated, including stability or slowing in the rate of decline in motor symptoms, cognitive symptoms or functional abilities. Additional secondary endpoints include imaging biomarkers: stability of or slowing in the rate of striatal neuron loss as measured by MRI, and demonstration of increased BDNF levels in the spinal fluid. Additional biomarkers will be evaluated.

Dr. Nolta's HD research team is also conducting studies using MSC to deliver gene silencing therapy using small interfering RNA molecules (RNAi). They published a study this year demonstrating the ability of MSC/RNAi to reduce the expression of mutant huntingtin protein in cell models. The team plans to proceed with safety testing in animal models in hopes of progressing to a future Phase 1 trial of MSC/RNAi in HD patients.

"People are hopeful, truly hopeful for the first time," explained HD patient advocate Judy Roberson who spoke at the July 2012 CIRM meeting. "This is a nightmarish, cruel disease in every way but now, thanks to CIRM, we are turning the dream of a stem cell therapy trial into a reality. Research means hope for people with this disease, but research costs money. CIRM has given us all hope."

Many Ways **to Give**

There are many ways for you to make a contribution to help HDSA improve the lives of people with Huntington's disease and their families.

- **Make a one-time Donation or a Tribute/ Memorial Gift to honor a friend or relative or the memory of a loved one:** Please visit our website, ***www.hdsa.org*** and click on the **"Donate" icon in the upper left hand corner of the page.** This will take you to a secure page where you can make a **direct donation** to HDSA.

  Or you can use the donation envelope located in this newsletter.

- **Donate Appreciated Stock and/or Mutual Funds:** Earn a charitable tax deduction for the full fair market value of the gift while you lower your capital gains taxes.
  - For information on how to make a stock or mutual fund donation please call 1-800-345-HDSA (4372), extension 235

- **Establish a Family Fund:** Join with friends and relatives and pool your resources to honor your family or remember a loved one and make your donated dollars work harder than you could individually.
  - For information on how to establish a Family Fund please call1-800-345-HDSA (4372), extension 235

- **Make a Planned Gift:** Join the **HDSA Heritage Club:**
  - Remember HDSA in your Will or Estate Plans
  - Establish a HDSA **Charitable Remainder Annuity Trust,** Charitable Lead Trust, **Charitable Remainder Trust, Charitable Remainder Unitrust**
  - Name HDSA as a beneficiary of your retirement plan
  - Name HDSA as a beneficiary of your life insurance policy
  - For information on making a planned gift to HDSA please call 1-800-345-HDSA (4372), extension 235

- **Work Place Giving**
  - **Matching Gifts:** Your employer or organization may be part of the HDSA Program, which can double your donation.
  - A list of participants is available on our website. If your employer is not part of this program, we would be happy to help enroll your company or organization.
  - **United Way/Community Health Charities/ Combined Federal Campaign:** Giving at work through payroll deductions to support HDSA is simple and there are many convenient ways to contribute. Check to see if your employer participates in any of these workplace giving programs.
  - To donate through the Combined Federal Campaign (CFC) designate your contribution to the Huntington's Disease Society of America, CFC ID # 11238

- **Become a Corporate Partner:** Businesses of all sizes can help bring us closer to the day when there will be the last generation with HD.
  - Give a cash or grant donation
  - Join an event: Participate or become a sponsor of the hundreds of HDSA events around the country, such as our Team Hope Walks or Celebration of Hope Galas.
  - Workplace Giving: Encourage employee giving through payroll deductions and show your employees that you support their philanthropic efforts by contributing a company match of their gift.

- **Donate Your Vehicle:** Call toll free 888-HDSA-151/888-437-2151 or e-mail ***donations@charitableautoresources.com*** to speak to an HDSA Vehicle Donation Representative. Our representative will schedule a pickup that's convenient for you, and provide you with confirmation of your donation.
  - Or visit our website, ***www.hdsa.org*** and click on the **"How You Can Help"** icon to donate your vehicle online. Select the Vehicle donation page, which will take you to a secure page where you can choose to make an online vehicle donation to HDSA.

Please visit our website regularly and browse the **HDSA Marketplace.** Purchasing a Care2Cure Bracelet or Necklace, amaryllis plant, golf polo shirt and other merchandise makes a difference – and helps us build awareness at the same time.

Huntington's Disease
Society of America
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