



TOWARD A CURE

A R E S E A R C H U P D A T E

*Stem Cells, Biomarkers, Mitochondria, mRNA, Dopamine Signaling, and NMDA Receptors
Mark New Grant and Fellowship Projects for 2005!*

More Than \$7 Million Donor Dollars Dedicated to HD Research Programs in 2005

This year promises to be a banner year for HDSA funded research. From stem cell research to messenger RNA, HDSA Grant and Fellowship recipients are testing the limits of what we know about HD and some researchers may even change some very basic thoughts about this disease.

The HDSA Grants and Fellowship Program provides researchers with vital funds that allow them to study different aspects of HD and to gather data that is needed to obtain new support from larger funding agencies such as the National Institutes of Health. Grant and Fellow awards also encourage a new generation of researchers to look at HD as a unique opportunity to *make a difference* and they, in turn, bring fresh, new and innovative ideas to the research table for consideration and exploration. As one researcher noted, "the opportunity to meet people and actually understand the human consequences of our work is very important."

For 2005, HDSA is pleased to announce that more than \$1 million has been dedicated to fund new grant and fellowship proposals from some of the most

prestigious research facilities in the world. Each year, HDSA receives in excess of 50 requests to fund projects for the limited number of grants or fellowships that HDSA can fund. How can we fund more of the research that may hold the key to treating this disease? As in the past, the answer lies with you. *Your support* remains essential if we are to move forward rapidly in all areas of HD research – basic, translational, drug discovery and clinical trials – that *will* yield effective treatments and a cure. Your past generosity has brought us to where we are today. Not only have your dollars moved research forward at an astounding rate, for what is considered an orphan and rare disease, but you have also led investigators to conclude that HD is a *model* for other neurodegenerative disorders and that the answers that are found for HD may break new ground for Parkinson's and Alzheimer's disease patients.

Much has come out of the HDSA research program and we expect even greater results in the future. Your support has helped us to set a standard by which only the best of the best is currently funded. We hope that you find



the descriptions of the new Grant and Fellowship projects interesting and enlightening. We ask that you consider making a gift in support of this vital and important program that introduces young investigators to Huntington's Disease research while fostering a spirit of originality and creativity in tackling the many pathways of HD. With your continued help, *we can make this the last generation with HD.*

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NOTES FROM THE LAB

By James Gusella, Ph.D
Chair, HDSA Medical and
Scientific Advisory Committee



In June, the Huntington's Disease Society of America will host its 20th Annual Convention in Atlanta Georgia. I urge each of you to attend this very important three day meeting that brings together our HD families with researchers, clinicians, social workers and friends to learn more about exciting advances in the care and cure of HD.

This year's Research Forum promises to be even better than before. Just about one year ago, HDSA identified two areas in HD research that would complement the basic research being conducted by HDSA and expand the pipeline to drug discovery and clinical trials. To that end, HDSA formed strategic partnerships with CHDI (drug discovery) and the Huntington Project (clinical trials). Each brings new talent to the HD pipeline and supports the work begun by HDSA Grant and Fellowship recipients and HDSA Coalition for the Cure investigators. Both CHDI and the Huntington Project will play an integral role in this year's Research Forum and I know that, if you are able to attend, you will leave with the knowledge that HDSA is truly driving the research process and that your dollars are being well spent in support of this continuum of research.

We know that our families want treatments and answers *now*. We understand the frustration that those living at risk and those affected by HD feel, and we are moving ahead as swiftly as we can to bring effective therapies from the laboratory bench to your bedside. That is one reason why HDSA sought to partner with and fund CHDI and Huntington Project. Together, we can move research forward in a logical and

progressive manner to clinical trials that will yield treatments while we continue to search for those elusive pieces of the HD puzzle that are needed to find answers for this disease.

And finding the answers is important – for those with HD, their children and their grandchildren. And it is also important for so many who are affected by other neurological diseases. It is estimated that by the end of this decade, one in every four Americans will be affected by a neurological disease. That means more than *75 million people!* If we can find the answers to HD then we may play a pivotal role in answering the questions for more wide spread neurological diseases like Parkinson's and Alzheimer's.

The cure is out there and with our partners, CHDI and the Huntington Project, we can move rapidly to take advantage of every new advancement that is made both in basic research and in the arena of drug discovery and translational research which, in turn, will lead us to viable treatments for our HD families—treatments that are needed *now*.

So much is going on in HD research today. In addition to financially supporting the efforts of CHDI, the Huntington Project, and the HDSA Coalition for the Cure, HDSA has also awarded Grants and Fellowships totaling more than \$1 million to projects that are studying a range of "hot" topics in research today including early neural stem cell research, preliminary study of the role of mitochondria, fundamental process of aggregation, genetic modifiers, and basic RNA. Many of these research areas are also studied, in greater depth, by one or more of the HDSA Coalition for the Cure teams. You can

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TOWARD A CURE

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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 appearance of advertising, or the mention of commercial articles available for sale in articles published in this newsletter, is not an HDSA, Inc. guarantee or endorsement of the product or the claims made for the product by the manufacturer. Statements and opinions expressed in articles are not necessarily those of HDSA, Inc.

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 Huntington's Disease.

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.



Huntington's Disease
Society of America

HDSA Grants and Fellowships for 2005

New HDSA Grants

Roger Albin, M.D.

University of Michigan

■ *NR2B Mediated Toxicity in Murine HD*

This research project will explore the mechanisms of nerve cell death/dysfunction in HD by crossing HD mouse models with other mouse models that carry known abnormalities in pathways thought to be involved in nerve cell death/dysfunction. Past hypotheses have focused on the idea that increased signaling through NMDA receptors coupled with abnormalities of nerve cell energy production contribute to nerve cell death/dysfunction in HD. Defining the mechanisms of nerve cell death/dysfunction in HD may point the way towards potential treatments.

Sarah Augood, Ph.D.

Massachusetts General Hospital

Ruth Luthi-Carter, Ph.D.

Swiss Federal Institute of Technology

■ *mRNA/Cell Changes in Human Caudate Neuron*

Transcription has become an important target for HD therapeutics. In order to identify and correct underlying transcriptional deficiencies present in the human HD caudate, early mRNA changes that are specific to neurons and independent of cell death must be found. Extending earlier work that looked at regional striatal mRNA expression, this study will now pinpoint these early changes by harvesting neuron-specific mRNA samples using laser-capture microdissection of medium spiny striatal neurons (MSN) from human HD brains.

Ilya Bezprozvanny, Ph.D.

University of Texas Southwestern Medical Center

■ *Dopamine Signaling in HD*

Selective neurodegeneration of striatal medium spiny neurons (MSN) occurs in the brains of individuals affected by HD. Earlier research has identified a connection between glutamate signaling and MSN degeneration in HD. This study will now look at dopamine signaling and MSN degeneration in order to identify potential pharmacological blockers that might prove useful in therapies for HD.

Janet Dubinsky, Ph.D.

University of Minnesota

■ *NMR Spectroscopy of HD Mice*

This study will use powerful magnets to measure the amounts of certain cellular chemicals (metabolites) that are involved in energy production. This information will

be used to determine whether energy production changes prior to or as a consequence of aggregate formation, as well as whether energy production changes prior to or as a consequence of actual neuronal cell death. Researchers will use mouse models of HD to compare change in certain metabolites during disease progression with the appearance of mutant huntingtin aggregation and markers of cell death.

Jonathan Fox, BSc, BVSc., Ph.D.

Massachusetts General Hospital

■ *Lactate Dehydrogenase Dysfunction in HD*

Neurological symptoms in HD result from dysfunction and death of brain cells (neurons) caused by insufficient energy within the cell. This energetic insufficiency could result from decreased production of, or increased requirements for, chemical energy in the form of ATP. ATP is produced by mitochondria, the cells' energy "power house." This study will determine whether abnormal activity of an enzyme known as lactate dehydrogenase (LDH), which is involved in the production of chemical energy, contributes to brain energetic insufficiency in HD. LDH is already known to play an important part in the production of chemical energy in the brain under normal conditions. During this process, neurons take up lactate and convert it to ATP in a biochemical cascade that requires LDH. Abnormal utilization of lactate by neurons occurs in human HD. Thus, this study will first try to understand the relevance and causes of decreased LDH activity and then try to determine whether increasing LDH activity in animal and cell models of HD is protective.

Second Year HDSA Grants

Daniel Cullen, BSc., Ph.D.

J. David Gladstone Institutes

■ *The Role of Huntingtin in Synaptic Vesicle Recycling and Its Altered Function in HD*

It is suspected that HD may result partly from an alteration of normal huntingtin function. In particular, Cullen hypothesizes that huntingtin functions as a regulator of synaptic vesicles (SV), which are essential components of nerve cells, and that this function is disrupted in HD. To test the theory, he will monitor SV regulation in nerve cells resembling those of HD patients to determine if there are any differences between those cells and normal nerve cells. Using protein identification technology, he also hopes to identify other proteins that are part of the huntingtin protein complex in order to better understand the actual role that huntingtin plays in HD.

Eric Kmiec, Ph.D.

University of Delaware

■ *Oligo Disruption of Htt Aggregates*

The aggregation (clumping) of mutant huntingtin causes the formation of inclusion bodies which may contribute to HD pathogenesis. A number of pharmaceuticals are being screened to determine their ability to inhibit or reverse huntingtin aggregation (Htt) and/or inclusion body formation. Novel synthetic DNA molecules called oligonucleotides can work to prevent the Htt aggregation process. Using cell based and biochemical assays, researchers have found a class of oligonucleotides that can largely reduce aggregate and inclusion body formation. This study aims to define the most active form of oligonucleotide and evaluate potential therapeutic agents through screening assays. The most active form will then be prepared for analyses in animal models of HD.

Thung-S. Lai, Ph.D.

Duke University Medical Center

■ *Tissue Transglutaminase and Polyglutamine Aggregation*

The hallmark of HD is the presence of insoluble inclusions that contain aggregated, expanded polyglutamine and other proteins. It is unknown whether insoluble inclusions are an effect or a cause of the disease. However, to find effective treatments or a cure for HD, it is important that researchers understand how insoluble inclusions are formed at the molecular level. It has been hypothesized that soluble high molecular weight (HMW) complexes are small and scattered. Researchers recently discovered an enzyme known as tissue transglutaminase (TTG) that promotes the formation of soluble HMW complexes that may contribute to neurotoxicity in HD. Therefore, this study will attempt to turn off the expression of this enzyme, TTG, and to identify its functional domain so a strategy can be designed to control the formation of soluble HMW complexes within the cell.

Anne Messer, Ph.D.

NYS Department of Health/Health Research Inc.

■ *Intrabody Testing in HD Fly Models*

Previous research has shown that antibodies can bind to very specific proteins and this has led to several clinical trials for cancer and infectious diseases. In HD research, scientists have been able to bind a specific antibody fragment to a region adjacent to the mutated part of the toxic HD protein, huntingtin, so that the mutant huntingtin acts more normally inside cells and brain tissue. In this study, researchers will conduct additional tests in fly models of HD to determine which properties of the antibody fragments are most important in reversing the disease. They will then use these either directly as drugs or indirectly to validate drug targets.

Kasturi Puranam, Ph.D.

Duke University Medical Center

■ *Polyglutamine Expansion and Mitochondrial Dysfunction in HD*

HD is caused by a polyglutamine (polyQ) expansion but the mechanism by which expanded PolyQ causes neuronal death is not clear. Collaborating with HD researcher Dr. Panov and HDSA Coalition for the Cure investigator Dr. Greenamyre, the investigator has shown in preliminary data that the addition of polyQ protein to normal mitochondria causes a similar toxic effect on oxidative phosphorylation as is found in HD. This study will attempt to define the defect in mitochondrial function caused by proteins with expanded PolyQ domains in order to identify novel therapeutic targets to block HD pathogenesis.

Ashwini Rao, EdD, OTR

Columbia University

■ *Is Motor Variability a Reliable Biomarker in Presymptomatic HD?*

Recently, motor variability has been identified as a reliable marker in patients with HD. Motor variability increases with disease progression and correlates well with UHDRS scores and CAG repeat length. Using highly precise measurement techniques that provide accurate and objective measures of fingertip forces during object grasping, researchers will look at the variability of kinematics during reaching movements, and spatial and temporal variability during walking, in order to assess motor impairments associated with HD.

Hoon Ryu, Ph.D.

Boston University School of Medicine

■ *Role of Mitochondrial Protein Kinase: A Signaling Pathway in HD*

Mitochondria are the “power houses” of the cells and the source of more than 80% of the reactive oxygen species generated in the neurons. Neuronal toxins and stress that block mitochondrial functions can cause excessive neuronal damage and cell death by the dysregulation of oxy-radicals. Mitochondria dysfunction in HD may be directly associated with the dysfunction of a mitochondrial signaling pathway via an enzyme known as protein kinase A (PKA) and cAMP response element binding protein (CREB). This study will attempt to identify the relevance of the mitochondrial signaling pathway in HD and to provide therapeutic approaches to correct the dysfunction.



New HDSA Fellowships

Wado Akamatsu, M.D., Ph.D.

University of Toronto

■ *Establishment of a Novel Stem Cell Therapy for HD*

In conjunction with Dr. Derek van der Kooy, also at the University of Toronto, these researchers will use neural stem cells found in the adult brain in two HD mouse models (HDSA Coalition for the Cure investigators Michael Hayden's YAC mouse and Gillian Bates' R6/2 mouse) to self-repair damage to brain tissue caused by HD through the use of growth factors and neuroprotectants. The three objectives for this study can be found under the fellowship review of Dr. van der Kooy listed below under Second Year HDSA Fellowships.

Guang-Ho Cha, Ph.D.

Baylor College of Medicine

■ *Genetic Modifiers of Huntingtin Induced Neurodegeneration in Drosophila*

During the last few years, a growing number of neurodegenerative diseases have been identified that have an expansion of polyglutamine tracts as their underlying cause. However, scientists still have a limited understanding of the potentially various mechanisms of disease pathogenesis triggered by these expansions. This study will use the *Drosophila* (fruit fly) model of HD to try to clarify what goes wrong during neurodegeneration and how it can be stopped or at least retarded. The *Drosophila* model was chosen because of its versatility in high throughput genetic screens, its small genome which is fully sequenced and mapped, and its relatively short life span. The goal of this study is to discover the genes, pathways and molecular mechanisms involved in HD pathogenesis and to find effective therapeutic approaches for the treatment of HD. The genes identified in these genetic screens may also be relevant to research aimed at treating other neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. Finally, the genes and the corresponding proteins identified in this study may also provide valuable targets for future drug development for therapy in HD.

Catherine Cowan, BSc., Ph.D.

University of British Columbia

■ *Huntingtin and NMDA Receptor Function*

Recent studies have provided clues to how abnormal huntingtin causes neurons to die but much is still unknown about the actual process. Huntingtin alters activity of an ion channel protein on the neuronal cell surface known as NMDAR which functions to send signals between neurons. Overstimulation of NMDAR can cause neuronal death, and abnormal huntingtin promotes this overstimulation. This research study will look at the mechanisms by which abnormal huntingtin alters NMDAR function using

the YAC transgenic mouse model generated by HDSA Coalition for the Cure investigator, Michael Hayden. If scientists can understand how abnormal huntingtin causes neurons to die, effective therapies may be developed to halt or delay neuronal cell death.

Veronique Hermann, BSc, Ph.D.

University of Kentucky

■ *Structural Characterization of Exon-1 of Huntingtin*

HD is caused by an expanded polyglutamine sequence that is known to aggregate and cause neuronal cell death. The goal of this research project is to find a new route to HD therapy by preventing polyglutamine aggregation. If a drug could be designed to bind to this region of the huntingtin protein (encoded by exon-1), aggregation could be prevented, thus providing a route to disease therapy. To create a viable drug design strategy, more needs to be known about the structural properties of Exon-1 of huntingtin. Exon-1 was previously thought to be unstructured but new evidence shows defined structures that could be used as targets in drug design that would serve as part of an integrated therapy program.

Martin Lemay, Ph.D.

University of Montreal Health Center

■ *Changes in Voluntary Movement in HD*

The symptoms of HD progress slowly over a period of years. To test new drugs and therapies more rapidly, clinicians will need to detect small changes in brain function well before symptom changes can easily be detected. Movement anomalies are present years before onset and qualitative measures of changes in these anomalies may provide a good measure for short-term progression. This study will test the potential of sensitive motor measures to detect changes over short intervals of 6-12 months in presymptomatic and early HD and will compare these changes to those observed in clinical symptoms and in brain scans.

Jieya Shao, Ph.D.

University of California, San Francisco

■ *Regulatory Role of P160 ROCK Pathway and Actin Cytoskeleton in HD*

Previous research has found that a protein kinase, P160ROCK, helps to regulate huntingtin aggregation by modifying an actin-binding protein known as cofilin. Using a *Drosophila* (fruit fly) model of HD, this research project will study how the P160ROCK pathway might regulate HD pathogenesis by modulating an interaction between huntingtin and actin. Further studies will map and mutate the actin-binding design of huntingtin in order to test whether there is an association between actin and the regulation of huntingtin aggregation.

Second Year HDSA Fellowships

Nicholas Allen, BSc., Ph.D.

Cardiff University

■ ***Creation of a Human Model for Huntington's Disease by Gene Targeting of the HD Locus in Human Embryonic Stem Cells***

Although transgenic animal models have proven invaluable in testing theories about HD and the disease process, there is still a need to use human cell models. The development of sophisticated human cell models not only will enable researchers to further study the mutant HD protein in a human cellular context, it will also provide an ideal cell source with which to screen the actions of potential novel therapies. A suitable human cell model that recreates the HD mutation can now be made using a method known as "gene targeting" in human embryonic stem cells, which have the ability to differentiate into all cell types of the body including neurons.

Yoon Cho, Ph.D.

University of Bordeaux, France

■ ***In Vivo Electrophysiological and Behavioral Studies of R6/2 Transgenic Mice: A Mouse Model of HD***

This project will look at the extent to which the functioning of *in vivo* striatal cells is altered in the well characterized R6/2 transgenic mouse. Dr. Cho will compare the cell activity of these HD mice with their control littermates (who do not have the HD gene) in order to determine what cellular properties correlate to sensory, motor and behavioral alterations that occur in the R6/2 mice. The results should provide neurophysiological measures that could be used to test potential therapeutic treatments for HD.

Derek van der Kooy, Ph.D.

University of Toronto

■ ***Growth Factor Mediated Control of Neural Stem Cell Proliferation and Differentiation: Potential for Treatment of HD***

This project investigates the possibility of using stem cells to replace brain cells lost in HD. In order to stimulate the growth of stem cells in the brain, the researchers will administer growth factors and neuroprotectants to brain tissue. They will then use transgenic mouse models of HD to study the ability of the stem cells to replace damaged cells. There are three parts to the research study:

1. localize and characterize adult mouse neural stem and progenitor cells from normal and transgenic mouse lines.
2. test the ability of specific growth factors to stimulate the growth and replenishment of lost cells
3. perform behavioral tests to assess the success of growth factor infusions in recovering the damage produced in HD.

HDSA HAS MOVED

As of May 7, 2005, the National Office of the Huntington's Disease Society of America moved to 505 Eighth Avenue, Suite 902, New York, NY 10018. Please direct all correspondence to our new address. HDSA's toll free telephone number, National Office phone number, and fax remain unchanged. Please be sure to place the handy magnet enclosed with this mailing of *Toward a Cure* in a convenient place to remind you of our new address.

The Huntington's Disease Society of America would like to thank Newmark and Company Real Estate, Eric Gural, Allen Gurewich and Barbara Siegal for their generosity which allowed HDSA to move into newly renovated space at no cost to HDSA.

HDSA would also like to thank Castaneda Architects, SL Green, Pitkin Carpet, Whitestar Construction, Dullanski Electrical Supply, Cool Breeze HVAC and Excel Security for their participation in completing the new National Office.

IMPORTANT NOTE:

The Marker — Corrections

HDSA wishes to extend its apologies for misinforming readers about the true cause of Doug Marr's passing. Mr. Marr succumbed to cancer and not HD. Mr. Marr's first wife died from complications associated with HD and it was due to her that Mr. Marr became involved in HDSA. We sincerely regret any pain we may have inadvertently caused the Marr family.

Please note that the correct contact information for the social worker at the HDSA Center of Excellence at Indiana University is:
Phone: 866-488-0008 or 317-271-0624
Email: info@hdsaindiana.org

Please do not use any contact information included in The Marker for the social worker, Leo Rafail. HDSA apologizes for any inconvenience this may have caused.

HDSA Dedicates New HDSA Centers of Excellence at the University of South Florida and Indiana University

As reported in the November 2004 issue of *Toward a Cure*, HDSA designated four new HDSA Centers of Excellence that would begin serving our HD families and allied healthcare professionals beginning in January 2005. The four new HDSA Centers of Excellence were named at Indiana University in Indianapolis, Rush University Medical Center in Chicago, University of South Florida in Tampa and the University of California at Los Angeles.

HDSA is pleased to report that dedication ceremonies have been held at both the HDSA Center of Excellence at the University of South Florida (March 14) and Indiana University (May 5). Both events were well attended by university and clinic staff, local dignitaries and HD family members from the area.

HDSA wishes to thank the Development and Center staff at the University of South Florida, Dr. Stephen Klasko, Vice President of Health Sciences and Dean of the College of Medicine and Dr. Peter Dunne, Chair of the Department of Neurology as well as Dr. Juan Sanchez-Ramos, Director of the HDSA Center of Excellence, Marci McCall, clinic coordinator and Karen Karle, social worker for their support and enthusiastic welcome.

HDSA also wishes to thank Indiana University and

Drs. D. Craig Brater, Dean of the School of Medicine, Kenneth Cornetta, head of the Medical and Molecular Genetics Department, Mark L. Dyken, founder of the Neurology Outpatient Center, P. Michael Conneally, distinguished professor of Genetics and Neurology, and Tatiana Foroud, division head of Hereditary Genomics for their kind remarks and participation in the dedication ceremony that took place on May 5. HDSA also wishes to congratulate Drs. Joanne Wojcieszek and Kimberly Quaid, the new co-directors of the HDSA Center of Excellence and to recognize the HDSA Center Staff, Patti Day, Leo Rafail and Dr. Rod O'Brien.

HDSA will soon be traveling to Chicago (August 11) to formally dedicate the new HDSA Center of Excellence at Rush University Medical Center. We hope that our HD families and friends, residing in the metro Chicago area, will join us at this memorable event. HDSA will also soon be scheduling the dedication ceremony at the University of California at Los Angeles. For more information about either of these events, please contact. HDSAinfo@hdsa.org. Information will also be available on the new HDSA website which will formally launch at the 20th Annual HDSA Convention in late June.

HDSA Centers of Excellence can be found at the following well known medical facilities. For more information, please go to the HDSA national web site at www.hdsa.org (click on "Getting Help" and then "Centers of Excellence") or call HDSA at 800-345-HDSA.

New England HDSA Center of Excellence at Massachusetts General Hospital
Tel: 617-724-2227

HDSA Center of Excellence at University of Rochester
Tel: 585-341-7500

HDSA Center of Excellence at Columbia Health Sciences/NYS Psychiatric Institute
Tel: 212-305-9172

George G. Powell HDSA Center of Excellence at North Shore University Hospital
Tel: 516-562-2401

HDSA Center of Excellence at Johns Hopkins Univ/Hospital
Tel: 410-955-2398

HDSA Center of Excellence at University of Virginia
Tel: 434-924-2665

HDSA Center of Excellence at Emory University
Tel: 404-728-4957

HDSA Center of Excellence at University of South Florida
Tel: 813-974-6022

HDSA Center of Excellence at University of Alabama
Tel: 205-939-6280

HDSA Center of Excellence at Ohio State University
Tel: 614-688-3675

HDSA Center of Excellence at Washington University School of Medicine
Tel: 314-362-3741

HDSA Center of Excellence at Indiana University
Tel: 317-274-6949 or 866-488-0008

HDSA Center of Excellence at Rush University Medical Center
Tel: 312-563-2900

HDSA Center of Excellence at University of Iowa Hospitals and Clinics
Tel: 319-353-4307

HDSA Center of Excellence at Hennepin County Medical Center
Tel: 612-873-2515

HDSA Center of Excellence at Colorado Neurological Institute
Tel: 303-762-6674

HDSA Center of Excellence at Baylor College of Medicine
Tel: 713-798-7438

HDSA Center of Excellence at University of Washington
Tel: 206-616-2135

HDSA Center of Excellence at University of California, Davis Medical Center
Tel: 916-734-6278

HDSA Center of Excellence at University of California, Los Angeles
Tel: 310-794-1225

HDSA Center of Excellence at University of California, San Diego
Tel: 858-622-5854

Notes from Lab

CONTINUED

read more about the work of the HDSA Coalition for the Cure in the most recent issue of *The Marker* magazine.

As we look towards the 20th Annual HDSA Convention, we should stop for a moment to reflect on the many advances that have been made with your help. Today we are so much closer to understanding the disease process, so much closer to developing clinical trials that will lead to effective treatments. We have come so far in such a short time. Today, we are within reach.

I look forward to seeing each of you in Atlanta.

Drive for the Cure BMW Sweepstakes

There is still time to purchase your 2005 Drive for the Cure BMW Sweepstakes tickets. For every two that you purchase at \$100 each, you receive a third one absolutely free! This annual event is one of HDSA's most popular with winners drawn each year during the Convention Awards Dinner and Gala. While winners need not be present to win, imagine the excitement if you were attending HDSA's 20th Annual Convention AND YOU won! All proceeds are used to support the HDSA Research program. For more information about the 2005 Drive for a Cure BMW Sweepstakes, please contact Robert Coffey at coffeyr@hdsa.org or call 800-345-HDSA and ask for extension 10.

HDSA Hosts 20th Annual Convention in Atlanta Georgia

There is still time to register for HDSA's historic 20th Annual Convention which will take place in Atlanta from June 24-26. This exciting three day event will feature the annual Opening Ceremony, HDSA Focus on the Family Care Forum (Planning Along the



Prints Still Available from Noted Artist with Donation to Woody and Marjorie Guthrie Research Fund!

Limited signed edition prints (22" x 28") of Charles Bank Wilson's work *This Land Was Made for You and Me* are still available with a donation of \$300 or more to the Woody and Marjorie Guthrie Research Fund which directly supports the HDSA Coalition for the Cure. These colorful prints feature a prominent image of Woody Guthrie with his guitar set against an Oklahoma plain.

Mr. Banks Wilson is a noted printmaker, artist, and illustrator whose work hangs in major museums and galleries worldwide. As a personal friend of Woody Guthrie, Mr. Banks Wilson decided to use his final commission from the Oklahoma State Senate Historical Preservation Fund to capture the essence of this American icon. As Mr. Banks Wilson is known to quip "I never felt I was good enough to paint Woody but at the age of 86, I thought I might finally be ready."

The original work now hangs in the Rotunda of the Oklahoma State Capitol. Smaller signed prints are also available with a donation of \$100 to \$299 to the Woody and Marjorie Guthrie Research Fund. To obtain your limited signed edition or smaller signed print, please enclose your check, made payable to the Woody and Marjorie Guthrie Research Fund, in the convenient reply envelope included with this newsletter. Please be sure to indicate where the print should be shipped.

HDSA thanks Charles Banks Wilson for his generous gift to HDSA-funded research, and our HDSA Coalition for the Cure investigators also thank everyone who has given so generously to support research.

Continuum of HD), HDSA Research Forum (What's New in HD Research), workshops, sessions and roundtables. To register, just go to the HDSA national web site and click on "HDSA Convention." Complete the registration form and return to HDSA by mail, fax or email. Remember, registering for the convention doesn't guarantee you a room at the Atlanta Hilton. You must make your own hotel arrangements but, when you call the hotel (404) 659-2000, be sure to mention our special convention code (HDS) so you receive the lowest cost possible.

HDSA to Begin Quarterly Electronic and On Line Newsletter Dedicated to the Care and Cure of HD.

HDSA is pleased to announce that beginning in late June a new quarterly electronic and on line newsletter, Care2Cure, will bring information to

our HD families about international and national HD research news, clinical trials, articles about care and alternative treatments including supplements and natural therapies that may positively impact those affected by HD.

Martha Nance, M.D. will serve as the principal editor with regular contributions from HDSA Center of Excellence Directors and staff as well as commentary from HDSA and HD researchers in the field.

Care2Cure will be available on the HDSA national web site (www.hdsa.org) as well as through an exclusive email list serve. If you would like to receive Care2Cure as an email (with attachment), please register by emailing hdsainfo@hdsa.org and be sure to place "care2cure" in the subject line so you are added to the correct email list serve.