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EHDN News

EUROPEAN HUNTINGTON'S DISEASE NETWORK

Joseph Jankovic (Houston) and Bernhard Landwehrmeyer, Chairman of EHDN

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Imprint: Editorial Board of the EHDN Newsletter: Prof. Gillian Bates (King's College London School of Medicine, London, UK), Prof. Jenny Morton (University of Cambridge, Cambridge, UK), Dr. Diana Raffelsbauer (PharmaWrite, Giebelstadt, Germany), Dr. Jenny Townhill (Cardiff University, Cardiff, UK), Christiane Lohkamp (IHA, Stuttgart, Germany), Gabriele Stautner (Artifox Communication Design, Ulm, Germany).

FEATURE ARTICLE

By Diana Raffelsbauer, Freelance Medical Writer, Pharmawrite, Germany

EHDN 2010

The largest meeting ever devoted to Huntington's disease

The **European Huntington's Disease Network** (EHDN) invited its members to attend the 6th EHDN Plenary Meeting (EHDN 2010) in Prague. With more than 650 participants, this was the largest scientific meeting ever dedicated exclusively to Huntington's disease (HD). Why Prague? Besides being a wonderful city, Prague and HD have historical links. In her presentation, **Christiane Lohkamp** (Stuttgart) told us that the patron of Prague is Saint Vitus, after whom HD was initially named (as St. Vitus' dance). The martyr St. Vitus is one of the Fourteen Holy Helpers and is believed to have many healing skills, especially for movement disorders and diseases of the nervous system.

Welcome

The Chairman of EHDN, **Bernhard Landwehrmeyer** (Ulm), welcomed the attendees and encouraged them to actively participate in discussions. To facilitate the participation of the large number of attendees from the Czech Republic and Poland, especially non-healthcare professionals from HD-affected families, simultaneous translations into Czech and Polish were provided. The programme of the meeting, carefully selected by the Organising Committee that was chaired by Joaquim Ferreira (Lisbon), covered a

St. Vitus

wide range of topics. Scientists came ready to share their unpublished data in a bid to accelerate research aimed at finding treatments for HD. The progress in HD research has been tremendous since our last meeting in Lisbon in 2008, especially in the fields of neuroimaging and other assessment tools for premanifest HD, deep brain stimulation, gene silencing, HD mouse models, networks and systems biology. Recent results from these research strands were the highlights of the meeting.

Local host **Jan Roth** (Prague) described how the situation of HD patients and their families in the Czech Republic has improved thanks to the pioneering work of geneticist and founder of the Czech Huntington Association Jana Židovská. This ultimately led to the creation of a multidisciplinary clinical team for HD, now part of the movement disorders centre at Prague's Charles University.
Jiri Hruda (Pardubice) from the Czech Huntington Association addressed the meeting participants as "the fighters of HD". He said that they were impatient for results, thus reiterating the importance of a close relationship between scientists, healthcare professionals and HD families.

Hot topics

As has become customary at EHDN plenary meetings, the conference opened with a 'hot topics' session at which four hot topics were subjected to a lively debate. The first topic considered whether there are HD patients with fewer than 36 CAGs. A repeat of 40 CAGs is a fully penetrant mutation, repeats of 36-39 show incomplete penetrance, but repeats of 35 and less are currently considered to be non-pathogenic. Intermediate alleles between 27 and 35, whilst not causing disease, in some cases, can expand into the mutant range when passed onto the following generation. **Joseph Jankovic** (Houston) presented evidence from clinical practice that individuals with less than 36 CAGs develop HD; however **Arvid Heiberg** (Oslo) argued that this is unlikely to be the case. Approximately 1% of people who develop symptoms that look like HD do not have a CAG expansion. In some cases, they are found to have a mutation in another gene, but in most cases, the cause of the disease is unknown. Intermediate alleles are quite common (more than 4% of the general population) and therefore it is possible that by chance, some people carrying intermediate alleles will develop symptoms that look like HD.

Genetic counselling was the topic addressed by **Marina Frontali** (Rome), who drew the attention of the audience to the complexity of predictive genetic testing for HD, which has psychological, social, legal and ethical implications. The mutation in the HD gene *(HTT)* was identified in 1993, and the guidelines for predictive testing for HD were published in 1994. Together with Gerry Evers-Kiebooms and members of the EHDN Genetic Testing and Counselling Working Group, Frontali has been working on revising and updating these guidelines. Controversial topics include the implications of carrying intermediate alleles, the predictive testing of minors, the disclosure of the size of the expanded CAG repeat, and post-counselling information on prodromal signs / the possibility of participating in clinical trials for premanifest HD gene mutation carriers.

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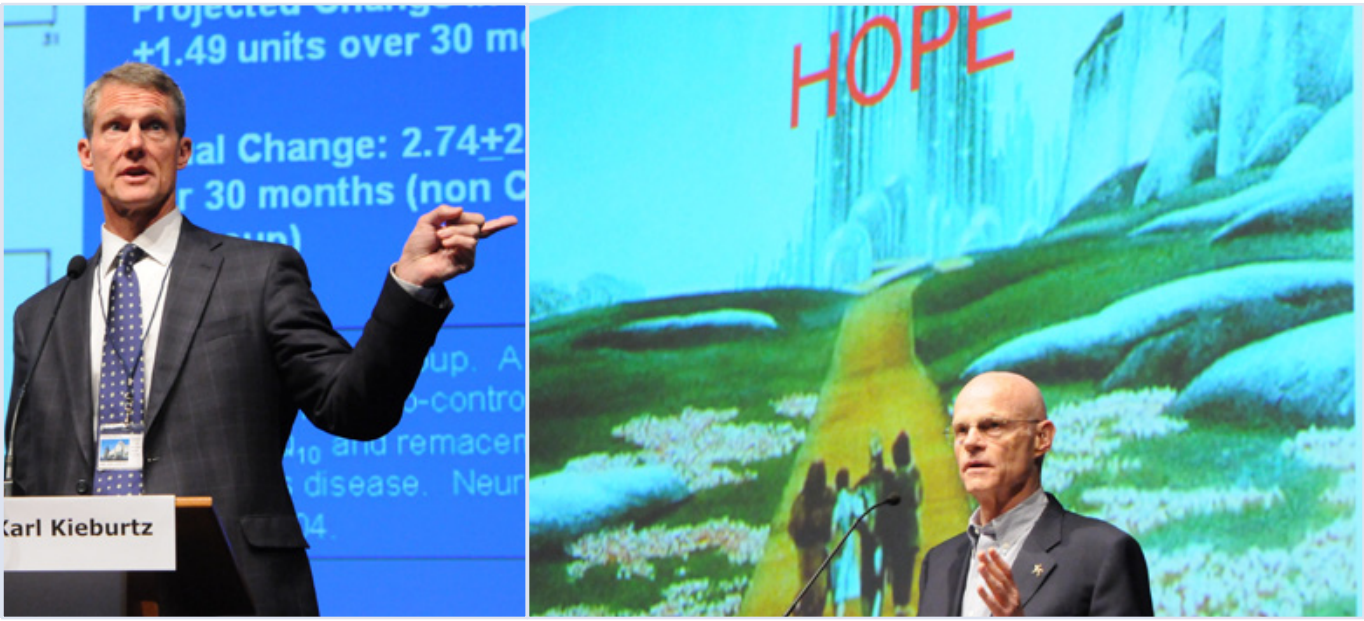


Sheila Simpson (Aberdeen) and **Stephen Smith** (Norfolk) enacted an entertaining and informative 'role play' to highlight the pros and cons of caring for HD patients at home as compared to in a nursing home. They communicated the burden of caring for someone affected by HD, the concerns associated with opting for institutionalised care, and the guilt caused by choosing this option. But they also demonstrated how professional carers could assist family members who are struggling to make this decision. They expressed the needs of HD families for specialised community-based clinical support in the form of nursing-at-home programmes that allow people to live at home for as long as possible.

While HD families are eagerly awaiting clinical trials, EHDN 2010 dedicated a hot topic session to the question of whether we are ready to measure a disease-modifying effect in HD. Based on examples from both observational and interventional trials that have been conducted in HD, **Karl Kieburtz** (Rochester) claimed that the tools are already available, e.g. UHDRS[1] (-TFC[2] and -TMS[3]) and MMSE[4], although these are only sensitive in manifest patients. The changes over time on these scales vary between individuals in the natural history of the disease and are expected to be smaller in treated patients. **Dan van Kammen** (Princeton) argued that our assessment tools are not sensitive enough for detecting clinical improvements. He highlighted the critical parameters of trial design: parallel versus delayed start, study population (inclusion and exclusion criteria), choice of endpoints, dose, trial duration, time point of visits, etc. "It is clear that we have to understand the biology of HD better as well as how the target or mechanism of action of the compound relates to the disease biology and how to optimally execute disease-modifying trials", said van Kammen.

1 Unified Huntington's Disease Rating Scale
2 Total Functional Capacity
3 Total Motor Score
4 Mini-Mental State Examination

Ongoing HD clinical trials

Whilst the discussion on whether we are able to measure disease modification in HD continues, exciting results from studies using potential treatments for HD were presented. **Lars Wojtecki** (Düsseldorf) and **Jan Vesper** (Düsseldorf) showed preliminary results from a pilot trial of deep brain stimulation (DBS) in HD patients that was conceived within the EHDN Surgical Approaches Working Group. The procedure consists of implanting electrodes in the area of the brain known as the globus pallidus (GP) and stimulating neurones by electrical impulses using a device placed under the skin. This technique has been successfully used in patients with Parkinson's disease and dystonia. The group led by Prof. Vesper has studied two different DBS schemes: stimulation of the internal GP followed by the external GP each for 6 weeks, or vice versa. Preliminary data from the first four patients of a total of six have shown that the procedure is safe with no surgical complications and no cognitive decline. Effects on motor performance are promising, but variable, differing between individuals, the stimulation sites used and the motor domain affected (dystonia vs. chorea vs. hypokinesia). Importantly, all patients in the study have subjectively reported improvements in daily living. Long-term effects have yet to be determined, and more sensitive assessment tools need to

be employed to measure differences in the various motor domains. A multi-centre trial involving 40 HD patients with assessments in the motor, cognitive, behavioural, functional and quality of life domains is currently under development.

Joakim Tedroff (Stockholm) gave an overview of the MermaiHD study, a phase III, double-blinded, placebo-controlled trial of the dopamine stabiliser pridopidine (also known as ACR16 or Huntexil®), which used a motor scale adapted from UHDRS-TMS as the primary endpoint. Pridopidine at a dose of 90 mg daily was shown to improve voluntary movements, eye movements and dystonia, but not chorea. As shown for the dystonia scores, "the effect size seems to increase over time, which is compatible with a disease-modifying effect", noted Tedroff. Patients with higher CAG repeat numbers are likely to benefit most from pridopidine, as the drug seems to reduce the correlation between the size of the CAG repeat and the rate of disease progression. The statistical analysis of the trial has been replicated independently by EHDN. Further information as to the efficacy of pridopidine will emerge from the ongoing open label expansion phase and from the HART study in North America.

Preparing for clinical trials

The subtle but recognisable undertone of nearly all presentations was the preparation for disease-modifying clinical trials, either through the use of cell and animal models, networks and systems biology, or by developing more sensitive tools to assess clinically HD onset and progression.

What is in the drug pipeline?

Ignacio Munoz-Sanjuan (Los Angeles) presented the strategies for the development of disease-modifying therapeutics that are being pursued by the CHDI Foundation. In cooperation with external collaborators, these comprise a wide variety of programmes, e.g. the strategies to reduce levels of mutant huntingtin (HTT) and the modulation of key mechanisms of HD pathogenesis, such as protein misfolding. Over 100 therapeutic agents have been tested in animal models, some of which are close to becoming an 'investigational new drug' and entering phase I clinical trials. Examples of promising candidate drugs are inhibitors of histone deacetylase 4. Following on from the genetic data generated by Michal Mielcarek (London) and Gillian Bates (London), the CHDI Foundation expects to have a molecule for proof-of-concept in HD animal models by the end of 2010.

Four presentations addressed the development of molecular strategies for silencing the *HTT* gene by destroying the messenger RNA from which the huntingtin protein is made. The two different approaches to this problem employ either antisense oligonucleotides (ASOs) or small interfering RNA (siRNA) molecules. In collaboration with ISIS Pharmaceuticals, **Don Cleveland** (San Diego) has designed ASOs that reduce the levels of both the mutant and normal forms of HTT and which, when delivered into the ventricles of the rodent brain, have beneficial effects in models of HD. **Jeff Carroll** (Boston) described initial encouraging attempts to develop ASOs that would target only the mutant *HTT* allele. Using a similar strategy, **Neil Aronin** (Boston) showed that it is possible to design siRNAs that will silence only the mutant *HTT* allele

by targeting single nucleotide polymorphisms (SNPs), i.e. sites where nucleotides differ between the mutant and normal alleles. **Dinah Sah** (Cambridge, USA) presented a programme for designing and screening siRNA molecules, improving their selectivity and potency, and delivering them into the human brain. The main challenges of this technology are still to understand the deleterious effects that may occur from reducing HTT levels and how these therapeutics can effectively be delivered to the brain.

Robin Meray (Cambridge, USA) presented a new strategy to harness the potential of a natural process called autophagy to enhance the degradation of misfolded and toxic proteins like mutant HTT. One of her molecules has been shown to promote clearance of toxic proteins and improve cognitive deficits in mouse models of Alzheimer's disease and Parkinson's disease. In collaboration with the CHDI Foundation and Gillian Bates, the molecule is currently being tested in mouse models of HD. Phase I clinical trials in healthy volunteers and early-stage Alzheimer's patients have so far shown that the compound is safe and well-tolerated.

Mahmoud Pouladi (Vancouver) has been testing a new lithium formulation (a low-dose microemulsion) in mouse models of HD. In treated animals, the compound rescued motor deficits, striatal atrophy, loss of medium spiny neurones and mutant HTT aggregation. At the molecular level, lithium normalised caspase-6 activity in the striatum and increased the expression of BDNF[5], a neurotrophic factor that promotes neuronal survival. Although lithium has been long used to treat bipolar disorders, the safety and efficacy of the new preparation in HD patients have yet to be established.

New assessment tools

Many research projects have focused on the development of new assessment tools for premanifest HD gene mutation carriers and early-stage HD patients. For instance, the FuRST-pHD[6] programme was established by the CHDI Foundation in order to develop a sensitive, reliable and valid rating scale for measuring symptoms and their functional impact in premanifest HD. **Ken Evans** (Toronto) described the programme as an iterative, multiple-cycle process that incorporates inputs from patients, clinicians and raters, clinical site staff, experts from different working groups as well as data from the available literature. The process involves fractionating core symptom domains into single items, developing interview questions and testing them in the target population. Evans stressed the need to listen to patients in order to be guided to relevant aspects of the disease. The launch of the FuRST-pHD Scale version 1.0 is expected in 2011. **Hugh Rickards** (Birmingham) gave a concrete example on how to improve rating scales in premanifest individuals. The task requires not only the dissection of a broad concept like 'apathy' into the different symptoms that people may present, but also necessitates devising and posing the right questions in order to obtain the most precise information. Rickards observed that clustering of symptoms on the UHDRS Apathy Item results in

5 Brain-derived neurotrophic factor

6 Functional Rating Scale Development Task Force for pre-Huntington's Disease

a low sensitivity for premanifest HD, but that the use of single sub-domains may be more sensitive. For instance, he found that lack of energy is the best performing item in premanifest HD and may be a good measure across the spectrum of HD. **Mark Guttmann** (Markham) outlined the next steps of the FuRST-pHD programme. He touched upon current diagnostic criteria and proposed a paradigm shift from onset/event-based to spectrum/process-based diagnosis, which encompasses the very early changes in brain volume, cognitive impairments, behavioural and motor abnormalities.

Hans-Peter Müller (Ulm) has been working with Jan Kassubek (Ulm) to devise new methods to optimise the analysis and post-processing of DTI[7] brain images of HD premanifest and manifest individuals. He developed quality control checks able to detect disturbed/corrupted measures (e.g. caused by movement of the subject during scanning) and to reduce noise in the dataset. The overall goal is to define MRI[8]-based parameters to be used in clinical trials as surrogate markers of disease progression and measures of drug efficacy. **Rachael Scahill** (London) discussed the potential usefulness of different imaging modalities in future clinical trials, particularly the techniques tested in the TRACK-HD study, such as structural MRI (analysis of cortical thickness, whole-brain volume and caudate volume), MRS[9], functional MRI and iron imaging. These techniques have been able to detect changes due to disease progression between premanifest and early HD patients as compared to controls over one year. Importantly, the imaging outcomes from TRACK-HD correlate with some functional outcomes.

Ralf Reilmann (Münster) questioned whether the gold standard for motor assessments in HD, the UHDRS-TMS, is objective, sensitive and reliable enough to be used as outcome measure in clinical trials. He has been developing novel quantitative motor assessments, such as tongue force, grip force, tapping and three-dimensional positioning and orientation assessments. These tools have been tested in TRACK-HD and have been shown to differentiate premanifest and early HD subjects from controls and to correlate with imaging and functional outcomes.

There are good reasons to support the inclusion of cognitive assessments in clinical trials for HD. For instance, cognition is an independent measure that has poor correlation to motor and behavioural performances; it may predict functional capacities in different domains; and it should be included in trials of neurodegenerative disorders at least for the assessment of intervention safety. **Anne-Catherine Bachoud-Lévi** (Créteil) discussed various aspects of choosing and using cognitive measures in clinical trials, including the pitfalls, and 'dos and don'ts'. She reported her recent progress in developing new cognitive tasks (the syntax test, a sentence-picture matching task based on syntax rules) and improving existing ones (statistical analysis and modelling of disease progression based on outcomes of the Stroop word test).

7 Diffusion Tensor Imaging, a Magnetic Resonance Imaging technique
8 Magnetic Resonance Imaging
9 Magnetic Resonance Spectroscopy

Two further presentations addressed the development of specific rating scales for HD. **Jenny De Souza** (Birmingham) presented preliminary findings from a REGISTRY data mining project aiming to explore the sensitivity, specificity and validity of two depression rating scales in HD: the Beck Depression Inventory and the Hamilton Rating Scale for Depression. **Aileen Ho** (Reading) gave an update on the Patient Quality of Life Questionnaire Project, an EHDN seed funded project from the Quality of Life Working Group. This new scale will be used as an optional component in REGISTRY version 3.0.

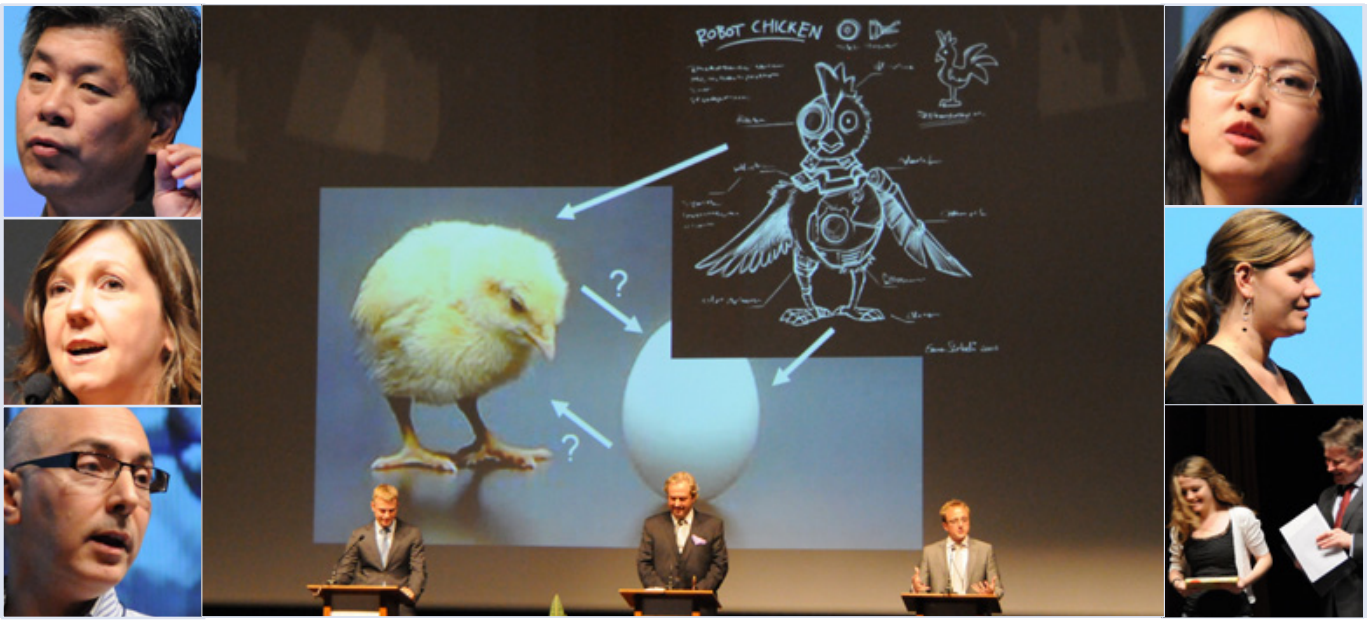
Networks and systems biology

A series of presentations focused on exploiting the potential of biological and clinical networks to identify pathways and targets involved in HD pathogenesis. Keynote speaker **Bin Zhang** (Seattle) explained the approach taken by Sage Bionetworks and noted that many key regulators predicted by network analysis have been validated at later stages. He anticipated that, as the amount of large-scale genomic data increases, biological networks will become more predictive and helpful in drug development and clinical research, as well as in understanding the biology and mechanisms of a disease. **Christian Néri** (Paris) has been exploring the FoxO network, for which he found common nodes between pathways involved in longevity/neuronal survival and HD pathogenesis, which possibly act as genetic modifiers of age of onset. **Erich Wanker** (Berlin) has been also searching for modifiers of age of onset using systems biology. His approach has led to the identification of several single nucleotide polymorphisms which may be involved in modulating disease onset.

John Warner (Princeton) has been developing statistical models that aim to better predict disease progression and inform clinical trial design, a task closely linked to the identification of biomarkers and the assessment of patient performance on different clinical rating scales.

Molecular pathogenesis of HD

Keynote speaker **William Yang** (Los Angeles) has been studying molecular mechanisms of HD pathogenesis in transgenic mouse models. To understand why mutant huntingtin, which is present in all cells of the body, causes selective degeneration of particular neurones, Yang has created conditional mouse models using bacterial artificial chromosomes (BAC) on which the human *HTT* gene can be switched on and off in different cell types. The so-called conditional BACHD mouse model replicates the neuropathological and behavioural symptomatology seen in other mouse models. By switching off mutant *HTT* expression in specific neuronal populations, Yang concluded that HD is in part a non-cell-autonomous disease, and that reducing mutant HTT toxicity in the cortex may reduce toxicity in the striatum and improve symptoms. Yang presented the results of a study in which he showed that serines 13 and 16 in the 17-amino acid-long N terminus of HTT (called NT17) are critical determinants of HD pathogenesis in BACHD mice (see EHDN *News* of June 2010, Article of the Month 06/2010). Mutation of these two amino acids to aspartate (which resembles a phosphorylated serine) can prevent motor and behavioural symptoms, neurodegeneration and HTT aggregation. NT17 plays a role in intracellular trafficking of mutant HTT (cytoplasmic vs. nuclear localisation) and could influence neuronal toxicity. Yang is now



validating the role of NT17 in HD pathogenesis and conducting *in vivo* proteomic studies of HTT fragments to find possible interacting partners. A detailed understanding of the mechanisms of this process may provide potential therapeutic targets for HD.

Rick Morimoto (Chicago) explained that the CAG repeat expansion, which causes the HTT protein to misfold and aggregate, disturbs protein homeostasis. The therapeutic goal is to restore proteostasis and prevent HTT from misfolding. As the cell ages, it gradually loses its ability to recognise and degrade damaged or misfolded proteins. Hence, there are common features between HD and aging, in that cellular proteostasis collapses with the result that the cellular stress response to toxic proteins becomes impaired. Another link between aging and neurodegeneration was presented by **Ruth Luthi-Carter** (Lausanne), who has been studying the role of sirtuins, a class of histone deacetylases, in HD. SIRT1 has been reported as neuroprotective in models of various neurodegenerative diseases when overexpressed. However, there are studies showing that inhibition of Sir2 (a sirtuin ortholog) and of SIRT2 suppresses HD pathogenesis in animal models. Luthi-Carter has found that SIRT2 inhibitors reverse the increased levels of cholesterol elicited by mutant HTT in a neuronal model of HD. Nevertheless, the function of sirtuins *in vivo* and how they may influence each other is complex, and there is still much to be understood regarding sirtuin biology. **Ronald Melki** (Paris) drew an interesting parallel between HD and prion diseases. In cell culture, he has found that HTT aggregates can penetrate cells and induce the aggregation of the host HTT protein. Whether this has any relevance in the context of the whole organism has not been demonstrated.

Nightly news

Repeating the success launched at the 2009 World Congress on Huntington's Disease in Vancouver, NBC News Correspondent **Charles Sabine** (Gloucestershire) presented and moderated the Nightly News session in which reporters **Ed Wild** (London) and **Jeff Carroll** summarised the presentations in plain language for the lay audience. All sessions of the meeting were recorded, and the videos will soon be available on the EHDN Website.

Poster awards

…went to **Aileen Ho** for her pilot study on expanding the response scale of the UHDRS-TFC, **Sofia Hult** (Lund) for her work on psychiatric and metabolic changes in the BACHD mouse model, and **Miranda Say** (London) for detecting visuomotor integration deficits in premanifest and early HD. About 150 posters were presented.

Teaching courses

In a first for EHDN Plenary Meetings, teaching courses were offered on selected topics. **Sarah Tabrizi** (London) and **Oliver Quarrell** (Sheffield) discussed 'how and when to diagnose HD', a controversial topic that has a major impact on the accuracy of the data collected and analysed in natural history studies such as REGISTRY. **Ralf Reilmann** and **Ferdinando Squitieri** (Pozzilli) presented new quantitative tools to assess the motor phenotype of HD in a more sensitive and objective way. Prescribing practices to treat the core symptoms of HD vary across

Europe. **Josef Priller** (Berlin) and **Raymund Roos** (Leiden) covered this topic during their teaching course 'how to treat HD', whereas **David Craufurd** (Manchester) and **Erik van Duijn** (Leiden) focused on the various psychiatric disorders that accompany HD and how to treat them.

Update on EHDN Working Groups

Three new EHDN Working Groups (WGs) were established in 2009 and 2010: Advanced HD WG (lead facilitators: **Sophie Duport** and **Nicholas Stoy**, London), Environmental Modifiers WG (**Martin Delatycki** and **Kaye Trembath**, Melbourne, and **Monica Busse-Morris**, Cardiff) and Young Adults WG (**Michael Orth**, Ulm, and **Ruth Sands,** Liverpool). The aims, needs, ongoing and future projects of each of these were presented by the respective lead facilitators. Progress and recently achieved milestones in three other WGs were also summarised: Biological Modifiers WG (**Christian Néri**, Paris), Neuroprotective Therapy WG (**Joaquim Ferreira,** Lisbon) and Standards of Care WG (**Sheila Simpson** and **Daniela Rae**, Aberdeen). For details, please visit the EHDN Working Group Website.

EHDN Business Meeting

Bernhard Landwehrmeyer summarised the achievements of EHDN since September 2008. The Network has now more than 1,000 members, an increase of 450 in the last two years. There are almost 140 EHDN study sites in 19 countries (including Russia, Singapore and South Korea). Since September 2008, the number of REGISTRY participants has nearly doubled to approximately 6,500 HD patients and premanifest gene carriers. Approximately 120 biosamples are collected from new participants every month. The biosample collection is stored at BioRep in Milan and now contains samples from more than 4,700 individuals. A wealth of clinical and biological data collected at more than 16,000 visits is now available for data mining projects.

The data that have been collected through REGISTRY are being utilised by many studies, for instance to find biomarkers, genetic modifiers of age of onset, as well as to improve assessment tools and rating scales and to provide an evidence base for symptomatic treatments. For details, please visit https://www.euro-hd.net/html/projects/proposals/announcements. To ensure that meaningful and conclusive results can be generated, biological data have to be combined with good-quality clinical data. Therefore, a new questionnaire, the 'HD clinical characteristics and age of onset questionnaire', has been developed to capture detailed information on disease onset and the first symptoms with which someone presents. A teaching video developed by David Craufurd with instructions on how to interview patients to better gather these data will be available soon.

REGISTRY version 3.0 will facilitate and accelerate the development and validation of new explorative assessment tools as optional components or sub-studies. These aim to establish an extended suite of validated assessment options targeted at specific patient groups and stages (e.g. premanifest HD, late stage HD, juvenile HD). The implementation of REGISTRY version 3.0 is one of the immediate goals of EHDN. Over the next year, a major objective of EHDN will also be to increase the number and quality of scientific publications resulting from its activities.

PLoS Currents: Huntington Disease is a new platform for HD scientific publications that is produced by the Public Library of Science (PLoS) with support from the CHDI Foundation. It was launched by **Mark Patterson** (Cambridge, UK) and **Gillian Bates** at this meeting. It is a new open-access publishing initiative for the rapid publication of scientific and clinical data to accelerate HD research. The venture will shorten the time required for publishing a manuscript (this can be up to one year in conventional review processes) to as little as one day after acceptance. The advantages are: i) Articles are judged only on their scientific quality and not a perception of importance, ii) they are rapidly peer reviewed by a panel of experts, iii) they are archived in PubMed Central and citable, and iv) all articles are open access and licensed under the Creative Commons Attribution Licence. Editor Gill Bates said "there is a clear need for a new platform through which data generated by HD researchers can be published. There is an enormous amount of data which never gets to see the light of the day because the publication hurdle is too high" (e.g. studies with negative results). Together with Mike Levine (Los Angeles) and Sarah Tabrizi as journal editors, Bates coordinates the review process by a team of approximately 40 experts representing all areas of HD research.

Bates has been also leading the editorial board of the EHDN *News*, together with Jenny Morton (Cambridge, UK), Diana Raffelsbauer (Giebelstadt), Jenny Townhill (Cardiff), Christiane Lohkamp (Stuttgart) and Gabriele Stautner (Ulm). Now in its 11th edition, the Newsletter has been the main vehicle for informing the members of EHDN about recent activities and research results in the HD field. From 2011 on, the EHDN *News* will appear in January, April, July and October. Besides the EHDN *News*, the Network has published information about HD in the form of frequently asked questions, which have been translated into most European languages.

The management structure of EHDN has been reorganised into four functional groups:

- Clinical Operations (Manager: **Tim McLean**, West Linton): Management of REGISTRY, interventional clinical trials, regulatory and IT provision, and language area coordinators
- Science (Director: **Joaquim Ferreira**): Scientific direction, coordination of working groups, management of research proposals and advice on clinical trials
- Communication (Director to be appointed): public relations, information and education, business development, and EHDN representation
- Administration (Manager: **Jamie Levey**, Paris): financial and legal issues, authorisation and coordination of facilities, membership, human resource, and general administration.

Stephen Dunnett (Cardiff) was reelected for the Executive Committee (EC) and **Christian Néri** became a new EC member. The Scientific and Bioethics Advisory Committee (SBAC) has three new members: **Maria Björkqvist** (Lund), **Åsa Petersen** (Lund) and **Edward Wild** (London). EHDN thanks the members who rotated out for their valuable contribution to the Network: **Joaquim Ferreira** and **Arvid Heiberg** from the EC, **Emilio Di Maria** (Genoa), **Alexandra Dürr** (Paris) and **Joaquim Ferreira** from the SBAC.

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All photos by Gabriele Stautner, Artifox Communication Design, Ulm, Germany

Thank you

… to all of the people who organised this very productive meeting and the wonderful social activities, which included a gala dinner at Prague's Municipal House and guided walking tours. Special thanks are given to **Bernhard Landwehrmeyer**, the local organising committee (**Jan Roth**, **Jiri Hruda** and **Pavla Šašinková**), the programme committee (Chair **Joaquim Ferreira**), Central Coordination (**Jamie Levey**, **Sonja Adam**, **Katrin Barth**, **Sonja Trautmann** and others). Particular thanks go to sponsors **Medivation**, **NeuroSearch**, **Siena Biotech**, and especially the **CHDI Foundation,** for supporting the activities of the Network and having made this event possible.

The meeting was an excellent example of what can be achieved in a collective effort towards developing treatments for HD and improving standards of care for HD patients. With inspiration, luck, hard work and funding, finding disease-modifying treatments for HD is becoming a realistic possibility.

More photos from the EHDN Meeting in Prague can be found here:

http://www.artifox.com/ehdn-prague

See you soon!

Save the dates for the 2011 World Congress on Huntington's Disease (September 11-14, 2011) in Melbourne, Australia, and EHDN 2012 (September 14-16, 2012) in Stockholm, Sweden.

Upcoming Meetings 2010/2011

2010	
Nov 11-14	Huntington Society of Canada National Conference, Edmonton, AB, Canada www.huntingtonsociety.ca/english/content/?page=194
Nov 12	Meeting of the Italian HD Association AICH-ROMA ONLUS and Fondazione Roma Terzo Settore in Rome: "Alla ricerca del senso perduto-Looking for the lost sense" based on the handbook "Hurry up and Wait" by Jimmy Pollard www.aichroma.com/incontropollar/comunicato%20stampa.pdf www.aichroma.com/incontropollar/incontro_pollar.asp
Nov 12-13	1st National Congress on Huntington's Disease, Madrid, Spain
Nov 13-17	40th Annual Meeting of the Society for Neuroscience, San Diego, CA, USA www.sfn.org/am2010/
Nov 30	National Conference on Huntington's Disease, NHS Trust, North Staffordshire Combined Healthcare, Staffordshire, UK
Dec 9-12	7th International Congress on Mental Dysfunctions & Other Non-Motor Features in Parkinson's Disease, Barcelona, Spain www2.kenes.com/mdpd2010/pages/home.aspx

2011	
Jan 28-30	4th European Neurological Conference on Clinical Practices, Lisbon, Portugal www.paragon-conventions.net/enccp2011/
Feb 7-10	6th Annual CHDI Conference on HD Therapeutics, Palm Springs, CA, USA www.chdifoundation.org/
Mar 9-13	10th International Conference on Alzheimer's & Parkinson's Diseases, Barcelona, Spain www2.kenes.com/adpd/Pages/Home.aspx
Mar 12-15	19th EPA European Congress of Psychiatry, Vienna, Austria www2.kenes.com/epa/Pages/home.aspx
Mar 26-29	26th International Conference of Alzheimer's Disease, Toronto, Canada www.adi2011.org/default.aspx?PageID=Home
Apr 22-24	XIII International Meeting of the Polish HDA Members and HD Conference, Warsaw, Poland www.huntington.pl/
June 24-26	26th Annual National Convention of the Huntington's Disease Society of America, Minneapolis, MN, USA www.hdsa.org/national-convention/convention.html
Sept 11-14	5th World Congress on Huntington's Disease, Melbourne, Australia www.worldcongress-hd2011.org/
Nov 12-17	20th World Congress of Neurology, Marrakesh, Morocco www2.kenes.com/wcn/Pages/Home.aspx
Dec 11-14	XIX World Federation of Neurology World Congress on Parkinson's Disease and Related Disorders, Shanghai, China www2.kenes.com/parkinson/Pages/Home.aspx

The Italian HD Association AICH-ROMA ONLUS and the Fondazione Roma Terzo Settore invite you to the meeting 'Alla ricerca del senso perduto – Looking for the lost sense' dedicated to family members and professional carers of HD patients. The meeting is based on the handbook **'Hurry up and Wait' by Jimmy Pollard**. The Italian translation of this book, 'Sbrigati e Aspetta', has been authored by Dr. Gioia Jacopini. Jim Pollard and Giola Jacopini will launch 'Sbrigati e Aspetta' in **Rome on 12th November 2010**.
For more information, please contact Paola Zinzi (paolazinzi@yahoo.com).