Huntington Disease Society India

HDSI INTERNATIONAL CONFERENCE 2025 2nd edition

23 & 24 August 2025
NIMHANS Convention Center

Conference Summary

20 Sessions

Keynote Lectures, Panel Discussions, Personal Narratives, Scientific Presentations, Demonstration Sessions, Book Launch, Cultural Programs and Networking Sessions

~80 Attendees

Researchers, clinicians, lived experience individuals, family members, students, policymakers, government representatives

TOPICS OF DISCUSSION

- Science & Research on Huntington's Disease
- Diagnosis, Care, and Management
- Lived Experience, Community Voices & Support Systems
- Awareness, Advocacy, Culture & Wellness

CONFERENCE QUOTES

"Families affected by Huntington's Disease face an enormous burden without adequate medical or social support. Recognition in the national rare disease framework, along with specialised clinics and a patient registry, will be a lifeline for thousands of families in India"

Venkateswara Rao Koushik, Chairperson, HDSI

Inauguration

Dr. Pratima Murthy, Director, NIMHANS, welcomed the HDSI International Conference and reaffirmed commitment to advancing HD science and care.

Dr. K VijayRaghavan, Former PSA to the PM and DBT Secretary, outlined genetics history, rapid progress, and Bengaluru's research ecosystem.

Dr. Rajani Parthasarathy, Deputy Director of Mental Health, Karnataka, discussed the state task force on dementia, HD, and neurological disorders.

Dr. PS Mathuranath, Professor of Neurology, NIMHANS, stressed the need to bridge science and society.

Dr. Suvarna Alladi, Professor of Neurology, NIMHANS, called for breaking silos and accelerating therapy development.

Dr. Pramod Pal, Professor of Neurology, NIMHANS, highlighted tailored therapies for HD and urged informed caution.

Dr. Sanjeev Jain, Emeritus Professor, NIMHANS and HDSI Founding Member, reflected on establishing the Molecular Genetics Lab and its journey.

Session Highlights

Mechanisms of Huntington protein transmission in the brain via two proteins

Dr. S Subramaniam
Assoc. Professor, FAU, USA

How Huntington Disease May Spread by Two Brain Proteins Working Together

Huntington disease is a serious brain disorder that affects movement, thinking, and behavior. It is caused by a harmful form of a protein called Huntingtin, which has an unusually long stretch of repeated genetic code (CAG repeats). The transfer of this deadly protein from one brain cell to another is one way that this disease gets worse over time.

The precise mechanism is still unknown, even though researchers have observed this spread in lab models and individuals with HD. It is known that tunneling nanotubes (TNTs), which are microscopic cell-to-cell links, are responsible for this.

Dr Subramaniam mentioned that their study has shown a crucial interaction between two proteins that propel this process: Rhes and Slc4a7. Rhes aids in the formation of TNTs and is mostly located around the brain impacted by HD. They found that it has a close relationship with the protein Slc4a7, which controls the acidity of cells. TNT production decreased and the dangerous mHTT protein decreased intercellular spread when Slc4a7 was blocked. Importantly, the distribution of the harmful Huntingtin protein in the brain was significantly less in mice lacking in Slc4a7.

These findings provide a new target for therapeutics that could delay or stop the progression of HD by revealing how Rhes and Slc4a7 work together to enhance disease propagation.

Session Highlights

Understanding HD

Dr. Vikram Holla
Assoc. Professor, Dept of Neurology, NIMHANS

Dr Meera Purushottam
Senior Consultant, MGL, NIMHANS

Dr. Vikram Holla explained the basics of Huntington's disease (HD) by breaking it down into clear, accessible ideas. He described HD as an inherited, progressive brain disorder caused by a genetic change that leads to the gradual damage of neurons. He explained how the condition affects movement, thinking, and mental health—often beginning in adulthood—and emphasised that HD is not just a movement disorder, but one that deeply impacts individuals as well as their families.

Dr Meera Purushottam outlined the basics of Huntington's disease (HD) using the idea of the cellular cookbook of life to explain its genetic and cellular underpinnings. She went on to show a global map of HD prevalence, underscoring why tracking prevalence is critical for understanding disease burden, identifying regional gaps, and guiding research and care, especially in India and briefly shared preliminary data from ongoing studies. In an important announcement, she called on clinicians and researchers across the country to come together to form an **Indian HD research consortium,** encouraging collaboration and shared access to patient samples. She concluded by stressing the urgent need for a national HD registry as the backbone for coordinated research, long-term follow-up, and improved care for people living with HD in India.

Session Highlights

From adversity to strength – stories of hope and courage

Paramjit Oberoi
Caregiver to 3 generations of HD patients

Rashmi E
Caregiver

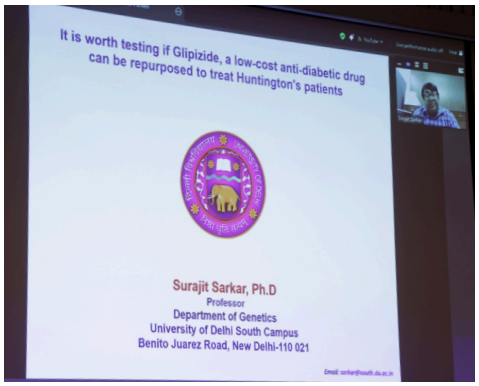
“I'm glad my husband had a car crash!”
With this startling statement, Paramjit Oberoi offered a philosophical and deeply poignant caregiver’s perspective to reflect on acceptance, and finding meaning in unexpected life turns. Her narrative invited the audience to sit with discomfort and rethink ideas of loss, resilience, and care.

Rashmi E. spoke with remarkable openness and calm, echoing the belief that what cannot be cured must be endured. Through her reflections, she highlighted quiet strength, emotional steadiness, and offered a grounded and compassionate lens on endurance and caregiving.

Repurposing Medicines - Is it worth testing Glipizide, a low-cost anti-diabetic drug to treat HD

Dr Surajit Sarkar

Asst. Professor, Department of Genetics, University of Delhi



Prof. Surajit Sarkar of the Department of Genetics at the University of Delhi joined the online session to deliver a talk about the unexpected effects of the anti-diabetic drug Glipizide in ameliorating neurotoxicity in Huntington-like diseases.

Prof. Sarkar began by explaining how the Huntingtin gene can be cloned into Drosophila to model Huntington's disease in fruit flies, thereby circumventing ethical issues with research in human participants and also providing significant benefits in ease of manipulation and drug testing.

He emphasised the significance of the insulin signalling cascade by enumerating its vital roles in development, growth, metabolism, and brain function, as well as its surprising role in reversing neurodegeneration.

He elaborated on the similarities between the pathways downstream of the insulin signalling pathway in Drosophila and humans like cell proliferation, glucose metabolism and protein synthesis drawing particular attention to the growth pathway which can potentially halt neurodegeneration. He further expounded on the importance of the insulin-mediated growth pathway by showing that blocking this pathway prevents insulin-mediated rescue of neurodegeneration in poly-Q expansion disorders like Huntington's disease.

Given the salient role of insulin in stalling neurodegeneration in Huntington-like diseases, Prof. Sarkar narrated how his team tested anti-diabetic drugs for their ability to restrict the pathogenesis of Huntington's disease of which Glipizide, emerged as a promising candidate. He showed that Glipizide, via the insulin cascade, reduces the number of inclusion bodies (a key hallmark of the disease) in the brains of humanized flies. He also mentioned that the treated flies had better locomotion and flight while also showing improved cognition and better transcription efficiency.

Underscoring the impact of this study, Prof. Sarkar revealed that the paper discussing these findings had sparked the interest of other scientists too, becoming the basis for several other studies.

He concluded by highlighting Glipizide's clinical potential as a readily available 2nd generation anti-diabetic drug which stimulates secretion of insulin, promotes insulin sensitivity and increases insulin receptivity and, most importantly, it does not lead to adverse side-effects in patients without Type-2 diabetes making it an ideal candidate for clinical trials such as those Prof. Sarkar and his colleagues are poised to conduct.

Genetic counseling and the family planning dilemma

Dr. Meenakshi Bhat

Faculty, Centre for Human Genetics, Bangalore

How to achieve a healthy baby if family is at risk?

Huntington's disease (HD) is caused by the CAG expansion in HTT gene. If an individual is positive for HD, then in each pregnancy there is 50% chance for the baby to get the disease. Women transfer the expanded allele in a stable manner compared to men. Hence parent of origin is important. Most of the family with HD will have family history, it is only 1-3% have de novo mutations.

Modalities of testing

1. Direct Diagnostic Testing – individual who is showing the signs of movement disorder, who has repeated falls, symptoms of HD will be directly sent for genetic testing.

2. Pre-symptomatic Testing – If someone visits the clinic asking for genetic testing because many in their family have HD, testing will not be done directly. International guidelines will be followed before testing the individual. Individual will be informed about the outcomes of the results, marriage prospects if not married, insurance services, employment choices.

3. Pre-natal Testing and Counselling - Someone who already knows that family members have HD but wants to have a healthy child

a)Prenatal testing at 11 weeks of pregnancy – Chorionic villus sampling (CVS)

A needle is put in through the womb of the pregnant mother and takes a tiny bit of tissue from the placenta. Once the tissue is taken out, genetic testing is conducted. If the baby has an expanded gene and invariably ends up with this disease in distant future, the choices about what parents would do will be discussed far in advance.

b) Amniocentesis - 15 weeks of pregnancy onwards

The needle is passed with the help of ultrasound and 15ml of fluid is sucked out which has the cells shed from the skin of the baby and these cells containing DNA will be tested.

These tests are known as definitive tests. Ethical issues are general for both families and clinicians in these scenarios. Questions such as should we stop the pregnancy for a late onset disorder, whether any treatments will be available by then and will we regret the decision, may arise.

Indirect testing: When a parent does not want to know their status but wants a healthy child indirect testing is done. This method tracks HD gene through family.

Pre-implantation diagnosis: Sperm and egg from the couple will be used to generate embryos using in vitro fertilisation technique. 1-2 cells from the embryo will be taken and genetic testing will be performed. The status is known for only couple of cells in the whole embryo. Healthy embryo will be transferred to the women, although it is not a completely healthy embryo if the cells are mosaic.

Cell free DNA in non-invasive screening test: Foetal DNA circulating in the maternal circulation will be subjected to DNA testing

Session Highlights

Living with HD – Overcoming challenges and a wishlist for a better life

Archana, Noyon and Sankesh
HD heroes

Archana shared her story through a deeply personal wishlist for a better life: to be seen for who she truly is; to have access to compassionate telemedicine from doctors; and to receive timely, dignified palliative care for Huntington's disease (HD).

Sankesh shared his personal journey of living with Huntington's disease and spoke about his book, Whispers of Resilience: The Boy Who Would Not Break.

Noyon shared the story of his father, who lives with Huntington's disease, and spoke more broadly about the experiences of other families affected by HD in Bangladesh, offering a compassionate perspective.

Session Highlights

Therapeutic Choreography based on Odissi dance style for Chorea

Daksha Mashruwala and Namrata Mehta
Odissi Teacher and her senior disciple

Dance as therapy for HD.

Advocacy - Why it matters and examples from around the world

Phyllis Foxworth
Manager of Advocacy, Huntington's Disease Society of America (HDSA)

Michaela Winkelmann
International Development, International Huntington's Association (IHA)

HDSA officials Phyllis Foxworth and Joyce Sereno emphasised the power of doing any small thing—raising your voice, creating awareness, and speaking up wherever you can, from everyday spaces like parking lots to the highest levels of policymaking.

From the International Huntington Association (IHA), Michaela Winkelmann shared examples of successful advocacy campaigns from Germany, illustrating how even a single voice can spark meaningful change.

Targeting mutant Huntingtin phosphorylation as therapy for HD

Dr. Ravi Vijayvargia

Asst. Professor, Dept of Biochemistry, Faculty of Science, M.S. University of Baroda

Huntington's disease (HD) is an autosomal dominant neurodegenerative disorder caused by CAG repeat expansion in the HTT gene, leading to polyglutamine expansion in the Huntingtin protein. The misfolded protein aggregates and produces both toxic gain of function and partial loss of function, causing selective neuronal death in the striatum and cortex. Clinically, this results in motor, cognitive, and psychiatric symptoms, along with peripheral effects such as cardiomyopathy, immune dysregulation, and testicular atrophy.

In this study, a fragment model of Huntingtin was used to mimic late-stage disease where N-terminal toxic fragments accumulate. The focus was on phosphorylation of Huntingtin at serine 13 and 16, as previous work suggested this modification can protect against toxicity. Kinetin, a plant hormone metabolized into kinetin triphosphate, was tested because it can act as a phosphate donor via CK2 to promote Huntingtin phosphorylation. Mutant Huntingtin fragments aggregated rapidly into puncta, reducing cell viability, but kinetin treatment significantly reduced aggregation in pre-, co-, and post-treatment conditions. Western blotting confirmed decreased insoluble aggregates and increased soluble protein after kinetin treatment. Importantly, kinetin rescued transcription of PGC1α, NRF1, and BDNF, which are crucial for mitochondrial function and neuronal survival. It also reduced mitochondrial ROS levels and normalized ATP production, indicating restoration of mitochondrial health. ER stress markers, which were elevated in mutant-expressing cells, were brought back to normal levels by kinetin.

Most critically, cell viability, which dropped to ~20% in untreated mutant cells, was preserved near normal levels with kinetin treatment. Overall, the study demonstrates that kinetin-mediated phosphorylation at serine 13/16 reduces aggregation, corrects multiple cellular dysfunctions, and restores survival, highlighting kinetin as a potential therapeutic approach for HD.

Organellar crosstalk linked to HD Pathology

Dr. Shuvadeep Maity

Asst. Professor, Dept of Biological Sciences, BITS Pilani - Hyderabad

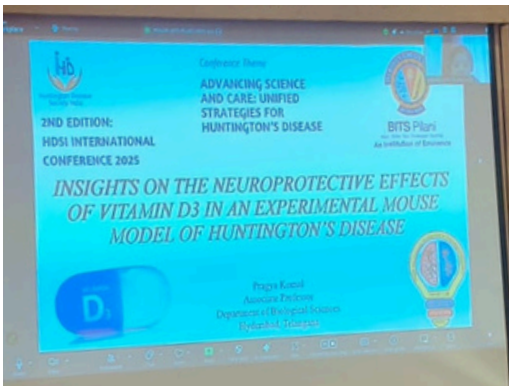
Prof. Shuvadeep Maity from BITS-Pilani talked about link between mitochondrial dynamics and HD pathology. He started with brief introduction about genetic basis of HD focusing on how HD mutation was discovered along with explaining consequences of this mutation. He placed mitochondria at the center of discussion and talked about its involvement in multiple important cellular processes such as energy metabolism, calcium balance and apoptosis.

Talking about mitochondrial dynamics, he focused on how mitochondrial fusion and fission processes form elongated and fragmented mitochondria respectively and the importance of balance between these processes in maintenance of mitochondrial function and cellular homeostasis. Imbalance between these processes is implicated in multiple neurodegenerative conditions. He discussed that in HD, alteration in expression and activity of fusion and fission proteins favors mitochondrial fission over fusion leading to excessive mitochondrial fragmentation and impaired mitochondrial quality control mechanisms. Hence, any therapy that favors mitochondrial fusion could counterbalance excessive fission process and could alleviate disease condition. In this direction, his lab screened around 40 small compounds in HTT-Q74 and HTT-Q23 expressing neurons in vitro and found 5 compounds that could restore fragmented mitochondrial phenotypes. Further, his lab found that peroxin expression is reduced in 3-Nitropropionic acid model of HD in mice, which is rescued by 4-Phenylbutyric acid. These findings are important as mitochondrial dysfunction is found in multiple neurodegenerative conditions, and these compounds might also be useful in those conditions.

Insights on the neuroprotective effects on vitamin D3 in an experimental mouse model

Dr Pragya Komal

Assoc. Professor, Dept of Biological Sciences, BITS Pilani - Hyderabad



Prof. Pragya Komal is an Associate Professor in the Department of Biological Sciences at BITS Pilani. She talked about the neuroprotective effects of vitamin D3 in the context of Huntington's disease (HD). She began her talk by discussing HD pathology, focusing on synaptic alterations, neuroinflammation, and redox alterations. She also talked about the structure, functions, and physiological effects of vitamin D3 in healthy conditions.

Moreover, she briefly discussed the pleiotropic effects of vitamin D3 in brain-related disorders, where it regulates neuroplasticity, neuroprotection, and neuroinflammation. Multiple studies suggest that vitamin D3 deficiency can be a risk factor for the development of several age-related neurological disorders. Furthermore, evidence suggests that serum vitamin D3 levels are low in Alzheimer's, Parkinson's, and Huntington's disease, and that supplementation has been found to improve cognitive and memory functions. However, the mechanisms behind its beneficial effects are not known.

Prof. Pragya utilised HD mice as a model to address this question. Her lab found that vitamin D3 administration reversed motor dysfunction in HD mice (3-Nitropropionic acid model). She explained that these benefits were brought about by an increase in neurotrophic factors such as brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), and vitamin D receptor (VDR), as well as antioxidant markers like catalase and glutathione peroxidase.

Since vitamin D3 deficiency is common in many neurodegenerative diseases and its supplementation enhances protective pathways, vitamin D3 can hold promise in slowing down these conditions.

Types of Drug and Clinical Trials for HD and rare disease

Dr Taslimarif Saiyed

Director, Center for Cellular and Molecular Platforms (C-CAMP) at NCBS

1. Brief Introduction about C-CAMP: Dr. Saiyed introduced the Centre for Cellular and Molecular Platforms (C-CAMP), which is part of the Bangalore Life Science Cluster along with NCBS, InStem, and TIGS. C-CAMP functions as one of India's foremost innovation and translational hubs in biotechnology and life sciences. It has nurtured startups, facilitated high-impact research, and enabled translational efforts that bridge laboratory discoveries with therapeutic applications.

2. Current Research on Huntington's Disease (HD) Treatment: The talk highlighted emerging therapeutic strategies being explored globally and within India to address Huntington's disease (HD), a devastating neurodegenerative disorder. Key approaches included:

- Gene Editing Approaches
- Cell Replacement Therapy
- Improving Neuronal Health
- HTT Protein Lowering Therapies

3. Advances in Innovative Therapy in India for Other Disorders: Dr. Saiyed then placed HD research in a broader context, showing how similar gene-, cell-, and molecule-based therapeutic strategies are being applied to other disorders, with India emerging as a key player in this translational ecosystem.

- Ophthalmology (Eye Stem Therapies): Stem-cell and regenerative interventions are under development for age-related macular degeneration and retinitis pigmentosa, two leading causes of vision loss.

- Neurodegeneration (Beyond HD): Neural precursor cells (NPCs), derived from pluripotent stem cells, offer potential for disease modelling and therapy. Early work with intranasal delivery of PSC-derived NPCs in Parkinson's disease rat models underscored innovative delivery strategies being investigated.

- Duchenne Muscular Dystrophy (DMD): Hanugen Therapeutics (Bengaluru) is pioneering genetic solutions such as exon-skipping and gene-editing approaches to slow disease progression. Peptris Technologies, leveraging AI-driven drug discovery, has partnered with Revio Therapeutics to advance PEPR124 (RT001), a repurposed therapy with mutation-agnostic potential that has shown promising preclinical efficacy. This asset is positioned for Phase 2 development and orphan drug designation, with global commercialisation plans in progress.

- Vitiligo: Ahammune Therapeutics is developing small-molecule therapies targeting deregulated cellular pathways that drive melanocyte destruction, with the goal of restoring skin pigmentation in this chronic, relapsing autoimmune condition.

- Rare Diseases: Startups like Aten Porus are venturing into the orphan drug space, addressing a profound unmet need for therapies in conditions that are individually rare but collectively affect millions worldwide.

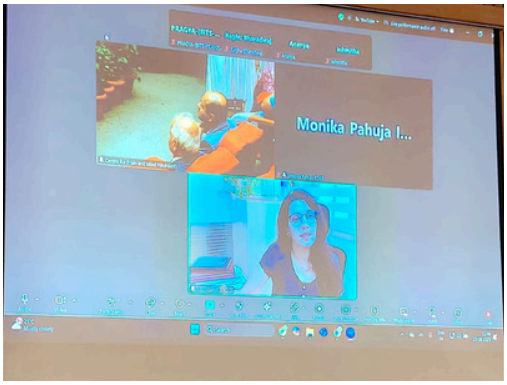
In conclusion, Dr. Saiyed's talk showcased how C-CAMP and Indian biotech startups are accelerating therapeutic innovation across multiple domains - from DMD, to stem-cell based regenerative therapies in neurodegeneration and ophthalmology, to AI-driven discovery platforms for rare diseases and autoimmune conditions like vitiligo.

Funding pathways and tracking rare diseases in India

Dr Monika Pahuja
Scientist E, Indian Council of Medical Research

Dr Amlin Shukla
Scientist E, Delivery Research, Indian Council of Medical Research

Dr Pahuja and Dr Shukla discussed funding opportunities and grants that can support the initiation of disease registries and early-stage research.



Building on this, Dr Shukla highlighted national efforts to track rare diseases, noting that an estimated 6–8% of India's population is affected.

Session Highlights

Managing HD at home and other approaches, followed by Q&A

Dr. Prasanna Menon
Founder & Director, Dr Menon's Palliative Care, Mumbai

Dr Menon from Mumbai explained the role of palliative care in HD and shared stories about how how timely and appropriate palliative care can be.

Yoga for strength and balance, breathing and wellbeing

Dr Hemant Bhargav
HOD, Dept of Integrative Medicine, NIMHANS

Dr. Selva Ganapathy
Senior Physiotherapist, Gait Lab at NIMHANS

Dr Bhargav highlighted how yoga can support people living with HD by improving balance, strength, breathing, and neuroplasticity, using simple demonstrations to illustrate key practices.

Dr Ganapathy talked about the importance of physiotherapy and gait.

A practical guide to living better with HD

Dr Nitish Kamble
Addl. Professor, Dept of Neurology, NIMHANS

Dr BK Yamini
Addl. Professor, Dept of Speech Pathology and Audiology, NIMHANS

Dr Sheeba Vasu
Assoc. Professor, JNCASR

The talk by Dr B K Yamini, an expert in speech and audiology, highlighted common impairments seen in patients with Huntington's disease at various stages of the condition. These included impairments in speech as well as swallowing. She provided suggestions to improve communication between patients and caregivers and suggested alternate means of communication that are not reliant on verbalisation, such as signs and writing pads. She emphasised the need to repeat, rephrase, and simplify messages by using keywords judiciously. To elicit yes/no responses, questions were simplified. She also suggested rehabilitative and compensatory swallowing strategies.

Dr Nitish Kamble, Professor of Neurology, argued that multidisciplinary expertise is involved in patient care, including clinicians from neurology and psychiatry, as well as physiotherapists, counsellors, and dietitians. He pointed out that strategies must cater to both patients and caregivers due to the prolonged and often debilitating nature of the condition, which can affect multiple family members. He advised physical activities including gentle exercises and yoga, occupational therapy, and installation of adaptive devices for fall prevention. Nutritional strategies, including frequent small meals with high nutritive value, were suggested. Healthy fats, protein-rich foods, and full-fat dairy products may be provided. Creating a structured daily and weekly schedule was suggested to help patients maintain a sense of control. Seeking support by connecting with family and neighbourhood networks was also recognised as a positive emotional and practical aid.

Dr Sheeba Vasu emphasised the importance of the circadian clock and its healthy functioning. She explained that the body's internal clock regulates many physiological and cognitive functions, making its health critical for overall wellbeing. She highlighted research using animal models that showed improvements in motor function and metabolic outcomes when subjects were placed on strictly timed feeding protocols. Given that circadian clock organisation is conserved across species, such behavioural and lifestyle interventions may help slow the progression of Huntington's disease.

Session Highlights

Mental & emotional wellbeing for patients and caregivers

Dr Sanjeev Jain
Psychiatrist, NIMHANS; HDSI

Dr Adesh Agarwal
Asst. Professor, AIIMS Deoghar, Jharkhand

Dr Adesh Agarwal spoke about mental health needs and care for HD patients and caregivers

Dr Sanjeev Jain and Dr Adesh Agarwal also addressed questions about mental health interventions for HD community, highlighting available support through the Tele-MANAS mental health helpline.

Juvenile HD - finding a future and finding strength in numbers through outreach

Jenna Heilman
Executive Director, Huntington Disease Youth Organization (HDYO)

Paramjit Oberoi
Founder of the charity Sheenam's Wish, UK

Jenna talked about the idea of "Big A" and "little a" advocacy, highlighting how both large-scale action and everyday efforts matter and emphasising the power of even one voice in creating change.

Paramjit Oberoi shared the inspiration behind founding the charity Sheenam's Wish, reflecting on how personal experience can be transformed into purpose-driven support for others.

Gallery

Gallery

Huntington Disease Society India

HDSI VISION, MISSION, CORE VALUES & SLOGAN

Huntington Disease Society India (HDSI) is a nonprofit organisation striving to bring hope to people affected by Huntington's disease in India.

Vision: To create a supportive community where individuals and families affected by HD can find hope, resources, and care.

Mission: To improve the quality of life for those affected by Huntington's disease through support, education, and advocacy.

Core Values: Striving to facilitate living with dignity for HD families, patient-centric programmes, and collaboration and partnership.

Slogan: Hide no more, Defeat HD

HDSI is trying to solve what problem?

Awareness: HD is relatively unknown to many healthcare professionals in India. HDSI aims to raise awareness among the medical fraternity and policymakers through workshops, awareness events, and other initiatives.

Right to Live with Dignity: People with HD are often shamed and ostracised by society. HDSI aims to provide a voice for families affected by HD and enable them to live with dignity until death by empowering families to make informed decisions, involving them in social events, and promoting supportive communication.

Huntington Disease Society India

Provide Resources: HD predominantly affects people in midlife. Over time, as the disease progresses, patients lose cognitive and motor functions, often resulting in job loss. Patients may become fully dependent on caregivers, and families may lose financial stability and harmony. HDSI aims to provide financial assistance, palliative care (through government hospitals), and genetic counselling for families affected by HD.

Facilitate Research: Currently, there is limited research on Huntington's disease in India. HDSI aims to work with the government to create a national registry of patients with HD, collaborate with international organisations and pharmaceutical companies for drug discovery, and support participation in clinical trials.

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Huntington Disease Society India

I stand with Huntington Disease families

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