HUMAN BRAINS

HUMAN BRAINS: PRESERVING THE BRAIN
FORUM ON NEURODEGENERATIVE DISEASES

Milan, 15 September 2022 – "Preserving the Brain," a forum on neurodegenerative diseases, is the fourth phase of "Human Brains," Fondazione Prada's neuroscience project. Realized in collaboration with thirteen of the most relevant international neuroscience institutes and universities, "Human Brains: Preserving the Brain – Forum on Neurodegenerative Diseases" comprises an exhibition (16 September – 10 October 2022) and a conference (6 – 7 October 2022) at Fondazione Prada's Milan premises and a series of online workshop (19 September – 4 October 2022) organized by the research centers.

The international institutes involved in "Preserving the Brain" are: Harvard Medical School, Brigham and Women's Hospital, Ann Romney Center for Neurological Diseases, Boston, United States; Hôpital de la Pitié-Salpêtrière, Sorbonne University AP-HP, Neurology department and Paris Brain Institute, Paris, France; UniSR – Università Vita-Salute San Raffaele, Milan, Italy; Juntendo University Hospital, Neurology Department, Tokyo, Japan; Karolinska Institutet, Stockholm, Sweden; German Center for Neurodegenerative Diseases (DZNE) within the Helmholtz Association, Bonn, Germany; Max Planck Institute of Neurobiology, Munich, Germany; Montreal Neurological Institute-Hospital, McGill University, Canada; Tianjin Medical University General Hospital, Neurology Department, Tianjin, China; UCSF Weill Institute for Neurosciences, University of California San Francisco, San Francisco, United States; University College London, United Kingdom; Weizmann Institute of Science, Revohot, Israel; Yale School of Medicine, New Haven, United States.

"Human Brains" is the result of an in-depth research process undertaken by Fondazione Prada in 2018 in the field of neuroscience. The project has been driven by a deep interest to understand the human brain, the complexity of its functions, and its centrality to human history. The program has been developed by Fondazione Prada together with a scientific board chaired by neurologist Giancarlo Comi and composed of cognitive neurologist Jubin Abutalebi, philosopher Massimo Cacciari, science journalist Viviana Kasam, curator Udo Kittelmann, neurologist and neurophysiologist Letizia Leocani, neurolinguist Andrea Moro, and cognitive neurologist Daniela Perani.

"Human Brains" employs a multidisciplinary approach that brings together neurobiology, philosophy, psychology, neurochemistry, linguistics, artificial intelligence, and robotics. The human brain is examined in the plural—as expressed by the title—to underline its intrinsic complexity and the irreducible singularity of each individual. The project's first stage was the online conference "Culture and Consciousness." Held in November 2020, it focused on the study of consciousness, the brain's highest and most complex function. The second chapter, titled "Conversations," was based on a series of video talks from international scientists, philosophers and researchers between September 2021 and April 2022. The third phase, the

exhibition "It Begins with an Idea," focused on the history of brain studies, is currently on view in Venice until 27 November 2022.

"Preserving the Brain" aims to stimulate an open and critical exchange between international scientists and experts on neurodegenerative diseases, such as Alzheimer's, Parkinson's, Amyotrophic lateral sclerosis and Multiple sclerosis, which are widely spread and as yet incurable. The forum participants include researchers, patient associations, and representatives of health care institutions and the pharmaceutical and biotechnology industries. They debate the current state of knowledge of these diseases and the tools currently used to fight them, while also seeking to identify lacunae in the search for possible therapies, and jointly defining priorities and strategies to sustain scientific research.

As stated by Miuccia Prada, President of Fondazione Prada, "For us, this phase of the project is particularly significant as it permits a closer understanding of scientific research's impact on our everyday lives, and in particular on the discovery of possible cures and treatments. 'Preserving the Brain' also demonstrates how critical collaboration and sharing of knowledge are within the scientific community. This first international forum could become recurring and allow the organizations we are associated with to communicate with a wider audience, as they would like to, and Fondazione Prada to contribute tangibly to neuroscientific research."

As underlined by Giancarlo Comi, President of the "Human Brains" scientific board, "The exhibition and conference that comprise the 'Preserving the Brain' project have been developed with the goal of finding a common strategy to protect the brain from neurodegenerative diseases. New technological developments have markedly increased knowledge of the biology that underlies these diseases, and potential targets for new treatments are taking shape."

Exhibition | 16 September – 10 October 2022

The exhibition is on view from 16 September to 10 October 2022 in the Podium, the space at the center of Fondazione Prada's Milan venue. Conceived by the New York studio 2x4, the exhibition design is divided into fourteen sections supervised by the research centers, and a common central area that will encourage dialogue and exchanges between the thirteen institutes. Each section examines a specific research process on neurodegenerative diseases employing video presentations, technological objects and instruments, scientific texts, and visual materials. This project aims to explore the complexity of scientific research by retracing the stages from identifying therapeutic targets to the different phases in the validation of new therapies to the availability of a drug for the patient. Special attention is given to personalized medicine, a new approach that recognizes the uniqueness of the

individual and treats the person suffering from a disease rather than a disease manifested in a person, thus optimizing the use of drugs and the treatment monitoring.

Workshops | 19 September – 4 October 2022

From 19 September to 4 October 2022, institutions participating in the project give a series of eleven online workshops that are available to the public via streaming at humanbrains.fondazioneprada.org. Each meeting enables the assessment of a specific aspect in the search for new treatments for neurodegenerative diseases.

Conference | 6 – 7 October 2022

The pivotal moment of "Preserving the Brain" is the scientific conference held on 6 and 7 October 2022, at Fondazione Prada's Cinema in Milan. Giancarlo Comi has conceived this initiative in dialogue with the thirteen research institutes involved in the project. This initiative is addressed to researchers and universities participating in the project and representatives of prominent institutions in the health sector. The conference is also streamed and visible to all on the online platform: humanbrains.fondazioneprada.org.

Each day is divided into four thematic sessions, each one featuring three lectures and an open discussion between the scientists and researchers. The first day ends with a keynote and a working dinner. The second day is completed by two additional keynotes and a round table in which scholars, technology experts, representatives from the pharmaceutical industry and patient advocacy associations discuss future challenges in developing new therapies. The speakers explore the subject of neurodegenerative diseases from different perspectives, such as genetic implications and molecular mechanisms, clinical trials, and possible drug treatments.

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FORUM ON NEURODEGENERATVE DISEASES

DESCRIPTIONS OF THE EXHIBITION SECTIONS

1.
Karolinska Institutet, Stockholm, Sweden
TECHNOLOGY IN THE STUDY OF NEUROLOGICAL DISEASES: FROM A SINGLE CELL TO A WHOLE-BRAIN

The first observations regarding neurological pathologies date back to the mid-19th century by the father of what is today considered modern neurology, Jean-Martin Charcot (France, 1825). Back then no indication could offer the slightest hint to neurologists like Charcot about the future revolutionary development of technologies capable of unveiling the activation status of genes at single-cell level (single-cell sequencing, a technology pioneered at Karolinska Institutet), visualizing the location of a single molecule in single cells in tissues (RNA in situ hybridization) or the combination of computer-generated magnetic fields and radio waves to recreated images of organs inside the human body (magnetic resonance imaging).

The application of these technologies in neuroscience has spearheaded the understanding of the human brain in health and in disease. The images presented by Karolinska Institutet intend to guide visitors across a fascinating and eye-stimulating walk from a single cell to the complex brain through the lens of these revolutionary technologies.

2.
Yale School of Medicine, New Haven, United States
SINGLE CELL SEQUENCING IN NEURODEGENERATIVE DISORDERS

Immune Cells in the Multiple Sclerosis Nervous System

Multiple Sclerosis (MS) is a genetically mediated autoimmune disease. Over the past half-century, we have gone from not having an understanding of the disease's underlying cause to a good working model that has been validated by clinical trials, such that early treatment can have a major effect in stopping disease flare-ups. Specifically, depleting B cells also has a dramatic effect on stopping attacks in early disease where the treatment is given to a patient every 6 months. In this regard, we are now engaged in clinical trials using B cell depletion at the very earliest stages of disease before there are any clinical manifestations. The genetic bases for MS have been elucidated and we have identified 233 common genetic variants, each with a small effect on disease risk, but that together lead to the disease. The majority of these common variants are controlling immune function and together contribute to a lower threshold for activating the immune system.

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Single cell RNA sequencing is an important advance that allows us to deeply characterize the immune system without bias in order to investigate and interrogate each individual immune cell. Details of the technique are presented in the video. We extract spinal fluid from a patient using a lumbar puncture—which means inserting a needle between the vertebrae and collecting the fluid. Once extracted, the spinal fluid is rushed to the lab where it is spun down to collect the cells, and then brought to the 10x machine. We encapsulate each single T cell into a functionalized gel bead that is bar coded and mix them with enzymes and oils to create single cell microdroplets or GEMS. We then perform a chemical reaction to amplify the nucleic acid that codes for proteins that define each cell type to learn their function. The elegance of the technology is that we can identify cellular subtypes and rare cells with little bias giving a big picture of the biology underlying the disease.

This technique has elucidated new pathways underlying the cause of MS. There is still much to learn. While we have a good working model for early, relapsing remitting MS, we have little insight into the progressive phase of the disease. We also don't yet know whether very early treatment with B cell depletion prevents evolution to the progressive form of MS. Our examination of spinal fluid with these powerful new single cell technologies has revealed previously unknown pathways found in the infiltrating immune cells that are causing the disease.

3.
Montreal Neurological Institute-Hospital, McGill University, Montreal, Canada
ROLE OF GLIAL CELLS IN NEURODEGENERATIVE DISORDERS

Glial cells encompass the non-neuronal cells of the brain and spinal cord, deriving their name from the Greek word for "glue" based on the 19th-century concept of them comprising material that held the brain together. Pioneering histological work, driven by advances in techniques to stain brain tissues, showed that "glia" were comprised of distinct cells type that included astrocytes ("star cells" – Lenhossék 1893) and the "3rd element" oligodendrocytes and microglia (Del Hortega 1921; Penfield 1924). Subsequent advances in physiology–, tissue culture–, and molecular biology–based techniques have shown the functional properties of these cells under homeostatic and pathologic conditions.

Astrocytes and oligodendrocytes share a developmental lineage with neurons, all being derived from neural stem cells. Notable astrocyte functions include providing nutrients to neurons, maintaining integrity of the blood brain barrier by extending their foot processes to end on endothelial cells, regulating concentrations of neurotransmitters and electrolytes that impact on the function of neuronal synapses and axons, and providing clearance of material from the brain (glymphatics). Oligodendrocytes ensure efficient electrical conduction through the wrapping of their myelin membranes around axons and by providing trophic support to neurons.

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Microglia are derived from the embryonic yolk sack, sharing features with bone marrow derived macrophages; they are the primary phagocytic cells of the brain required for clearance of dead cells and debris and for pruning of synapses. Both microglia and astrocytes are sources of molecules usually associated with the immune system, allowing them to regulate functional properties of infiltrating immune cells during the course of neuroinflammatory disorders and to directly mediate tissue injury or conversely contribute to repair. These functions are dependent on their state of activation with the latter determined by their interaction with their microenvironment and by molecules reaching the brain from the systemic compartment (e.g. from the gut microbiome). An important communication network exists between the microglia and astrocytes.

Further included amongst glial cells are NG2 progenitor cells found within the brain parenchyma and ependymal cell that line the ventricles. NG2 glial cells are recognized as progenitors for myelinating oligodendrocytes and for their interactions with neuronal synapses. Ependymal cells contribute to the integrity of the brain/cerebrospinal fluid barrier.

Understanding the dynamic properties of all these glial populations under physiologic and pathologic conditions provides the opportunity to target these for therapeutic purposes aimed at neuroprotection and repair. Genome wide screening association studies (GWAS) have identified genes linked to glial cells as underlying or contributing to a wide range of "neurodegenerative disorders." The advent of techniques to derive each of these cell types from inducible pluripotent stem cells (ipscs) generated from blood cells or fibroblasts from such individuals and "controls" can be used to assess effects of modulating expression of disease-relevant genes and response to candidate therapeutic agents.

4.
Weizmann Institute of Science, Rehovot, Israel
MODELING ALZHEIMER'S DISEASE USING HUMAN BRAIN ORGANOIDS

Alzheimer's disease (AD) is a heterogeneous neurodegenerative disorder that results in the most common form of dementia. We generated mutations in presenilin 1 (*PSEN1*) and tau (*MAPT*) using genome editing. *PSEN1* and *MAPT* are genes that are mutated in cases of familial AD (fAD) and frontal-temporal dementia. Multiple brain regions are impacted in AD, including the hippocampus. Neuropathology and dysfunction in long-term potentiation have been described in the hippocampus and are associated with cognitive deficits. Furthermore, there are accumulating data indicating a strong involvement of the immune system, and in particular the immune resident cells in the brain, the microglia. In addition, over the last few years a strong correlation between metabolic changes and neurodegeneration has been suggested; however, this has yet to be thoroughly investigated.

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To better understand and identify early changes in fAD at the cell biology level we used both region specific and complex human brain organoids. Pioneering research from the lab of the late Yoshiki Sasai demonstrated that stem cells can recapitulate several features of organogenesis, including cell differentiation, spatial patterning, and morphogenesis, and has successfully generated organoids resembling different brain regions and retina. Subsequent research from Lancaster and Knoblich demonstrated that it is possible to obtain a mixed regional identity using a relatively simple media. This field has increased dramatically over the last few years with multiple protocols. Scientists can now generate connections between different brain regions by fusing structures known as "assembloids," thus mimicking a higher organization level, which may prove critical in modeling diseases. There have also been advances in regards to generating functional networks. Multiple studies have characterized the cell repertoire and diversity in organoids obtained from different protocols using single-cell (SC) analyses. Nevertheless, the understanding of brain organoid proteomics and metabolomics is still rudimentary. Our project aims to generate diverse brain organoid models that will include the hippocampus, in which microglia, the innate immune cells of the brain, will be included. The presence of immune cells is of great importance since aberrant inflammation in the CNS has been implicated as a major player in the pathogenesis of human neurodegenerative disease. We will further harness advances in genome editing and mutate *PSEN1* and *MAPT* leading to the generation of a series of isogenic pluripotent cell lines. The mitochondria will be tagged with dual purpose tags that will enable imaging and biochemical purification in a cell-type specific manner. During recent years there have been important advances that enable biochemical purification of mitochondria for proteomics and metabolomics analyses.

Our study focuses on characterizing the pathophysiology of early stages of genetic models of fAD and on mitochondria using hippocampal brain organoids combined with microglia and analysis using live-imaging, mitochondria health parameters, proteomics, metabolomics, and transcriptomics. These studies have the potential to reveal early biomarkers and identify possible therapeutic targets.

5.

Juntendo University Hospital, Neurology Department, Tokyo, Japan

PARKINSON'S DISEASE: A WHOLE BODY DISORDER

Parkinson's disease (PD) is the most common movement disorder.
The number of patients is around 15 million in the world. The incidence increases with age, suggesting that PD will continue to increase in the aging society.

PD has been clinically characterized by four main motor symptoms, rest tremor, akinesia, postural imbalance, and rigidity which are mainly caused by the loss of dopaminergic neurons in substantia nigra. During the disease, patients also show a broad range of non-motor symptoms

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including constipation, anxiety, insomnia, impulse control disorder, and dementia that reflect pathological changes spreading in the entire body. The pathological hallmark of PD is alpha-synuclein-containing neuronal aggregates named Lewy bodies and Lewy neurites.

Currently, no treatment can achieve complete remission, however, a considerable number of medications that target dopaminergic dysfunction are available to attenuate the symptoms.

6.
Max Planck Institute of Neurobiology, Munich, Germany
MULTIPLE SCLEROSIS: THE GUT-BRAIN CONNECTION

Multiple Sclerosis (MS) is not a rare disease: it afflicts an estimated 2M persons worldwide, mostly in the Western world, with an increasing tendency. The underlying lesions (plaques) are distributed throughout the central nervous system, presenting inflammatory infiltrations and confluent zones of myelin destruction.

The drivers of the pathogenesis are brain-specific immune cells (CD4+ T cells), which are activated in the peripheral immune system, break through the tight blood-brain barrier, interact with local tissue cells to ultimately destroy myelin structures, that normally insulate electrically active neuronal processes, axons.

Peripheral activation is the critical event sparking MS pathogenesis. Very unexpectedly, at least part of these processes happen in the intestine – far away from the central nervous system. It appears that certain bacterial components of the gut microbiota stimulate myelin-recognizing T cells, which sit quiescently in the healthy immune system, to become fit to attack the brain tissues.

This presentation will examine the nature of the relevant gut microbes and the mechanisms how they arm the cognate T cells to become pathogenic. Ultimately, the question of potentially new therapeutic strategies will be discussed.

7.
Hôpital de la Pitié-Salpêtrière, Sorbonne University AP-HP, Neurology Department and Paris Brain Institute, Paris, France
REMYELINATION BASIC STUDIES AND IMAGING OF REMYELINATION

Promoting Repair in Multiple Sclerosis: the Next Challenge for Patients' Care

Affecting approximately 2,8 millions patients worldwide, and beginning in most cases between the age of 25 and 35, Multiple Sclerosis is considered one of the major cause of acquired disability in the young adult. The disease is driven by complementary components, that include:

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I) inflammation, involving different types of immune cells infiltrating the central nervous system; II) demyelination (destruction of the myelin sheath, which surrounds the neuronal processes named axons, leading to reduced conduction velocity and increased vulnerability);
III) neurodegeneration (loss or irreversible damage of neuronal cell bodies and axons). The respective contribution of each mechanism depends on the age of the patient, the disease duration, and the type of disease evolution: whereas focal inflammation predominates within Multiple Sclerosis plaques that are disseminated in the relapsing remitting forms of the disease, diffuse neurodegeneration is mostly the hallmark of the progressive phases. Symptoms (motor, sensory, visual symptoms, gait disorders, bladder dysfunction, cognitive deficit, among others) differ between individuals and their severity varies.

Major progresses were made within the last decades regarding the development of immunotherapies, which drastically reduce the inflammatory component of the disease, and consequently the relapse rate. These therapies, however, insufficiently improve the disability accrual and the progressive phase of the disease, which is related to progressive accumulation of irreversible neuronal damage, mostly independent of bouts of inflammation. A key strategy to counteract neurodegeneration is to achieve neuroprotection by enhancing myelin regeneration, hence restoring nerve conduction and metabolic support to the axon.

In this context, research in recent years has led to a better understanding of the cells in charge of remyelination in the central nervous system. These “repairing” cells are either immature oligodendroglial cells (oligodendrocyte precursors or progenitors) which persist in the adult central nervous system or mature oligodendrocytes (these are the myelinating cells of the central nervous system, that may survive the demyelinating insult and contribute to repair). Experimental research using complementary models of demyelination/remyelination recently allowed to unravel the main molecular mechanisms underlying repair, and consequently to identify potential therapeutic targets, paving the way for translation to humans. From these initial promising forays, further problems have emerged, including the identification of an optimal design for repair clinical trials, and of outcomes measures to quantify repair. The development of reliable imaging biomarkers assessing the efficacy of emerging promyelinating drugs is therefore an urgent need, and imaging methods applying magnetic resonance imaging (MRI) but also Positron Emission Tomography (PET, a molecular imaging technology that takes advantage of ligands that specifically bind to myelin), are actively developed.

These new methods should transform the landscape of therapeutic trials targeting central nervous system repair. Several promising studies translating efficacious pro-remyelination therapies to people with Multiple Sclerosis are currently on the way.

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8.
German Center for Neurodegenerative Diseases (DZNE) within the Helmholtz Association, Bonn, Germany
MEMORY DRIVEN COMPUTING IN NEURODEGENERATIVE DISEASES

"The Machine" –– a Prototype
New computer architecture for fast and energy-saving processing of huge amounts of data

The exhibit is part of the first prototype of "The Machine," a completely new computer architecture developed by Hewlett Packard Enterprise (HPE). Most current computers are based on the so-called von Neumann architecture, which places the processor at the center of the computer. However, the traditional computer infrastructure fails when it comes to processing enormously large amounts of data. More and more memory and energy are needed to process data. As a result, large amounts of data must nowadays be distributed over very large computer systems. Such distribution causes a considerable amount of computing time, which becomes prohibitive when analyzing biomedical data.

"The Machine" focuses on memory to store, compute and process large amounts of data. It is also referred to as "memory-driven computing" or memory-centric computing. The exhibit is a so-called node of a 40-node prototype computer system for memory-centric computing. Each node has 4 terabytes (TB) of memory, which has been combined into a total of 160 TB of unified memory. The 160 TB of memory is equivalent to approximately 250,000 CDs. Currently, this is the largest pool of memory within a single computer system. "The Machine" thus allows for more efficient processing and a modular architecture. The processors are hidden under heat distributors, but the many memory bars are clearly visible. Research into neurodegenerative diseases is becoming increasingly data-driven. Microscopes and MRI machines deliver higher-resolution images, genomics data is collected and clinical and population studies provide a data avalanche that outpaces the growth of computational resources. This new type of computer is becoming a "gamechanger" as it allows to process data more easily in parallel. Working with genomics data often involves a series of tools that exchange data using files, but this I/O centric approach can be replaced using memory-driven computing. Here, tools can work more efficiently together on data in memory. Traditionally, memory was considered a scarce resource and a limiting factor for algorithm design, but now this can be overcome.

The DZNE has processed genomics up to 100 times faster by changing the representation of a reference genome in memory so that it can be accessed more efficiently. Not only has this saved 60% of energy, but it also allows to study the molecular processes within a cell at higher resolution. More complex data sets were often downsampled, effectively losing resolution during analysis. Using high-memory computer hardware, DZNE can analyze single-cell data sets with millions of cells to understand changes in gene expressions and cellular processes

during on-set and progression of dementia in patients in comparison to healthy controls.

The processing of large data sets in other domains, e.g. high resolution imagery, can often be portioned to multiple machines that need to synchronize because they do not share state. With memory-driven computing, a shared state limits overheads and reduces the needed programming, giving the researcher more time to focus on their research question. In conclusion, memory-driven computing allows the more efficient processing and analysis of large data sets to give researchers at DZNE faster calculation times and more time to focus on research than software development.

9.
Tianjin Medical University General Hospital, Neurology Department, Tianjin, China
NEUROIMAGING OF DEMENTIA

MRI and PET scans in patients with Alzheimer's Disease

Alzheimer's Disease (AD) is the most common cause of dementia in the elderly with a neurodegenerative continuum that includes a long period in the preclinical phase without any symptoms and an early clinical phase, which is named Mild Cognitive Impairment (MCI) or the prodromal stage, and finally a progressive phase of dementia.

Structural Magnetic Resonance Imaging (MRI) has been wildly used to identify patients with AD and distinguish them from those with other causes of cognitive impairment, such as vascular dementia. Furthermore, arterial spin labeling (ASL), which is a non-invasive MRI technique to quantitatively measure cerebral Blood Flow (CBF) using arterial blood water as an endogenous tracer, has been increasingly investigated in AD, because it has been suggested that changes in CBF plays an important role in the pathogenesis and progression of neurodegenerative diseases, even independent of their core neuropathology.

In the past decades, Positron Emission Tomography (PET) neuroimaging techniques played an important role and have been recommended to use in clinical diagnosis of dementia with appropriate tracers to assess pathophysiology in vivo in neurodegenerative conditions. Compared with indirect measuring abnormal molecular expressions in the cerebrospinal fluid, PET scans are noninvasive and able to reflect topographical
distribution of protein deposition or metabolic changes.

In conclusion, patients with AD had characterized patterns of brain atrophy, cerebral hypoperfusion and hypometabolism. In addition to structural scans, ASL MRI (especially processed with multivariate analysis) may provide
a promising biomarker for AD diagnosis and monitoring on a prospective single-subject basis in the future. Currently, amyloid and tau depositions

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and metabolic pattern measured with PET scans are useful in early diagnosis and differential diagnosis in patients with cognitive impairment.

10.
UniSR – Università Vita-Salute San Raffaele, Milan, Italy
PET AND BODY FLUID BIOMARKERS IN NEURODEGENERATIVE DISEASES

Biomarkers have a double role: to contribute revealing potential therapeutic targets and to diagnose and to monitor the evolution of diseases and their response to treatment. In neurodegenerative diseases, neuroimaging techniques and body fluid biomarkers provide fundamental information on the ongoing brain damage and the underlying pathophysiological mechanisms. The contribution of positron emission tomography (PET), a structural and functional technique, and of serum and spinal fluid neurofilaments are here presented, highlighting the results of some studies performed at San Raffaele Hospital.

11.
UCSF Weill Institute for Neurosciences, University of California San Francisco, San Francisco, United States
NEUROSCAPE: BRIDGING THE GAP BETWEEN NEUROSCIENCE AND TECHNOLOGY

Neuroscape is a translational neuroscience center at the University of California, San Francisco, engaged in technology creation and scientific research to better assess and optimize brain function of both healthy and impaired individuals.

Neuroscape engages in multiple areas of neuroscience research, including studies of attention, memory, perception, neuroplasticity, cognitive development, brain aging, non-invasive brain stimulation, neurofeedback, and psychedelics. Our research studies are supported by Neuroscape's technological innovations in interactive media and video games, virtual reality and multisensory integration, mobile technology and remote trial platforms, multimodal biosensing and advanced neuroimaging, machine learning and signal processing. Results from our studies are applied to advance novel approaches that better assess and improve cognition in healthy populations—children to seniors—as well as individuals with a broad range of medical conditions, such as Attention Deficit Hyperactivity Disorder, Depression, Multiple Sclerosis, and Alzheimer's Disease.

12.
UniSR – Università Vita-Salute San Raffaele, Milan, Italy
NEUROMODULATION TO TREAT NEURODEGENERATION

Neuronal and synaptic loss occur since our early life and continue throughout aging. In a sense, aging in itself is a neurodegenerative condition. In some individuals, owing to genetic repertoire

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and expression and their interaction with the environment, accelerated or specific patterns of neurodegeneration occur, leading to what is considered a pathological condition, particularly if the consequences of such neurodegenerative processes become clinically evident. However, the behavioural and cognitive consequences of neurodegenerative processes can vary remarkably among individuals and within the same person in different life circumstances and stages, owing to differences in brain reserve, cognitive reserve and coping/adaptation strategies.

Among the different factors influencing the consequences of neurodegenerative processes, or even neurodegeneration itself, a fundamental role is played by neuroplasticity. This term is widely used to include all phenomena occurring in neuronal function and structure in response to activity, including learning and practice, or to injuries. Variability in plastic mechanisms also accounts for differences among individuals exposed to similar events, from learning opportunities to brain diseases. Therefore, one strategy to prevent neurodegenerative processes, or to counteract their consequences, involves favouring positive plasticity, as opposed to detrimental plasticity occurring when neuronal changes lead to unfavourable results. These refer to active promotion of behaviours leading to neuronal survival and connectivity, such as cognitive and motor training, but also to the use of physical stimuli that can modulate neuronal activity itself, from the delivery of natural visual, auditory or somatosensory inputs to neuromodulation interventions. Nowadays, technological advancements allow non-invasive neuromodulation approaches, such as, or transcranial magnetic or direct current stimulation. These methodologies, acting on plasticity mechanisms in both directions—e.g. increasing or decreasing neuronal activity—may be used to improve or restore the ideal balance of excitatory and inhibitory activity within the target brain circuits. As neuromodulation itself acts on the same plasticity mechanisms induced by natural sensory stimulation or motor and cognitive behaviours occurring for example during learning and training, it has been hypothesized—and demonstrated in several clinical and research conditions—that the ideal strategy to optimize results is to combine all these methods for synergistic effects. However, much needs to be discovered about the best combinations depending on the individual condition and preferences. Finally, as prevention in general is much more effective than any treatment, much needs to be achieved not only in the fields of medical research, but also in ethical discussions about the opportunity to potentiate apparently not (yet) impaired neuronal functions.

13.
Harvard Medical School, Brigham and Women's Hospital, Ann Romney Center for Neurologic Diseases, Boston, United States
TRANSLATION OF SCIENTIFIC DISCOVERY TO PATIENT CARE

The induction of tolerance is a major goal of immunotherapy. Investigations over nearly 30 years have shown that anti-CD3 monoclonal antibodies (mAbs), a fully human anti-CD3 monoclonal antibody (mAb), modulates the immune system and prevents inflammation by

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inducing regulatory T cells. Several studies in animals showed that Foralumab ameliorates diseases including Multiple Sclerosis (MS), diabetes, arthritis inflammatory bowel diseases, and lupus. Foralumab use was shown to be safe in humans and benefit people with mild COVID-19. Currently, two MS patients are being treated at Brigham and Women's Hospital, Boston MA, with intranasal foralumab under Expanded Access with signs of clinical benefit. The strategy to induce oral tolerance by anti-CD3 mAb represents an exciting and novel avenue for treatment of autoimmune diseases due to the very good safety profile and the high variety of potential applications.

14.
University College London, London, United Kingdom
TRANSLATIONAL CLINICAL RESEARCH

Neurodegenerative diseases such as Alzheimer's disease cause the brain and nervous system to become progressively damaged over time. This eventually leads to dementia, disability and death.

We are living in times where extremely promising breakthroughs are being made in scientific laboratories, both to understand what goes wrong in the brain and to develop treatments that might slow or even begin to reverse the damage that causes dementia.

However, these scientific advances have little real-life meaning unless they can be translated into benefit for people suffering with these conditions. Translational research is the process of taking a discovery or drug from the laboratory and into the patient. It is a process that requires skills and contributions from a wide range of people: vision and leadership from clinical scientists; scientific rigor, precision and patience from those designing and executing the clinical drug trials; commitment and bravery from patients, nurses and doctors who might be receiving or delivering a new drug that
has never before been tested in humans.

At UCL this research is carried out in a highly specialist unit that bridges the worlds of clinical neurology and highly experimental neuroscience. The Center exists within and is supported by the hospital, but is run by specialist clinical academic research staff.

In this exhibition, we confront the viewers with a question. We challenge them to consider whether they would participate themselves in a clinical drug trial for a new drug that is being tested in humans for the very first time. Through a series of photos and a video installation we explain the critical importance of clinical translational research and explain who and what is involved.

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PROGRAM OF THE ONLINE WORKSHOPS

19 September 2022, 10 – 12 AM (UTC-4) | 4 – 6 PM (CEST)
Yale School of Medicine, New Haven, United States
Neuroimmunology of Multiple sclerosis

Overview of the Cause of Multiple sclerosis | David Hafler
Astrocyte and the Neuropathology of Multiple sclerosis | David Pitt
Loss of Immune regulation in Multiple sclerosis | Tomokazu Sumida
New Clinical Trials in Multiple sclerosis | Erin Longbrake
Moderator: David Hafler

There has been tremendous progress in developing working models underlying the cause of early, relapsing remitting Multiple sclerosis (MS). In this workshop, we will begin by presenting an overview of this model, where genetics interacts with environmental factors leading to activated, myelin reactive T cells mediating autoimmune inflammation in the white matter. We will then explore the detailed neuropathology of MS and how astrocytic/microglial interactions drive neurodegeneration. How the environment induces dysregulated immune system allowing activation of autoreactive T cells will be followed by an overview of new clinical trials in MS.

20 September 2022, 10 – 12 AM (UTC-4) | 4 – 6 PM (CEST)
Montreal Neurological Institute-Hospital, McGill University, Montreal, Canada
How Open Science is Changing Personalized Medicine in Neurodegenerative Diseases and Disorders

Welcome and introduction | Stefano Stifani
The C-BIG Platform: A Novel Open Science Combined Patient Registry and Multi-Modal Repository | Jason Karamchandani
The Neuro's Early Drug Discovery Unit: Opening up discovery through collaboration and outreach | Tom Durcan
Open Science in the works: a deeper understanding of Parkinson's Disease immunology | Jo-Anne Stratton
Leveraging Open Science to advance gene discovery in neurodegenerative diseases | Sali Farhan

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Closing remarks | Annabel Seyller
Moderator: Stefano Stifani

Open Science is the concept of sharing data, information, tools, and research results, and eliminating barriers to collaboration. This workshop will offer the opportunity to hear how Open Science is helping accelerate the discovery of new treatments and cures at the Montreal Neurological Institute-Hospital, the first academic institution in the world to fully embrace Open Science practices. Presentations will describe how open initiatives and collaborations are accelerating the scientific process towards understanding, and developing effective new treatments for neurodegenerative diseases and disorders.

22 September 2022, 1 – 4 PM (CEST)
Karolinska Institutet, Stockholm, Sweden
Demyelination & remyelination in neurodegeneration: From molecule to clinical application

Opening speech | Ole Petter Ottersen
The CSF shuttle: from the choroid plexus to brain parenchyma and back | Ana Falcao
Molecular markers of Multiple Sclerosis development and progression | Maja Jagodic
Fibrotic scarring—a determinant factor for the Central nervous system regeneration | Christian Göritz
In vivo imaging and quantification of demyelination and remyelination | Tobias Granberg
Moderators: Fredrik Piehl, Gonçalo Castelo-Branco, Chiara Starvaggi Cucuzza

The aim of this workshop is to highlight the latest findings in nerve and myelin cell biology research in the context of multiple sclerosis such as the role of the choroid plexus and cerebrospinal fluid movement in disease and in relation to drug delivery (Dr Ana Falcão, Karolinska Institutet & University of Minho), the use of non-protein biomarkers and epigenetic changes to resolve disease pathways and possible drug targets (Professor Maja Jagodic, Karolinska Institutet), exploration of the function of blood vessel-associated cells in brain damage and reparation (Associate Professor Christian Göritz, Karolinska Institutet), and development and clinical application of neuroimaging techniques to track disease processes and therapeutic effects (Associate Professor Tobias Granberg, Karolinska Institutet). The scientific and clinical context with future perspectives will be discussed in a round table led by Professor Gonçalo Castelo-Branco, Dr Chiara Starvaggi Cucuzza and Professor Fredrik Piehl.

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23 September 2022, 3 – 5 PM (CEST)
UniSR – Università Vita-Salute San Raffaele, Milan, Italy
PET molecular imaging in "brain neurodegenerative diseases"

Introducing and chairing | Daniela Perani
Assessment of Alzheimer's disease pathology in the living human brain | Rik Ossenkoppele
Evaluating the brain reserve effect on neurodegeneration | Gael Chetelat
How artificial intelligence can support our investigation of the brain | Valentina Garibotto
Moderator: Daniela Perani

Positron emission tomography (PET) is a well-recognized and unique tool for the in vivo assessment of brain metabolism, neurotransmission changes and protein load, and novel PET techniques are emerging for the study of molecular alterations. The availability of PET neuroimaging for the assessment of brain function, biology, and neuropathology has opened new venues in research, diagnostic design, and in the conduction of new clinical trials. Appropriate use of PET tools is crucial in supporting a prompt diagnosis and in evaluating drug targets aiming to slow down or prevent dementia. This workshop critically addresses the role of PET imaging in different neurodegenerative spectrum of diseases, highlighting the strengths and weaknesses, with special emphasis on methodological challenges and future applications.

26 September 2022, 9 – 11 AM (CEST)
Max Planck Institute of Neurobiology, Munich, Germany
Multiple Sclerosis: The Gut-Brain Connection

Introduction: The gut-brain axis in MS | Hartmut Wekerle
Autoimmune T cell traffic gut to brain | Naoto Kawakami
Microbiota and human disease | Amira Metwaly
MS twin study | Lisa Gerdes
Microbiota and MS - From model to human MS | Anneli Peters
Moderator: Hartmut Wekerle

Multiple sclerosis (MS) is an inflammatory condition of brain and spinal cord, which leads to destruction of the myelin substance, and by this causes a diversity of neurological deficiencies. This research explores the earliest onset, the trigger of multiple sclerosis. It has revealed that intestinal bacteria – the microbiome – is changed in people with MS, and that in experimental model animals, such bacteria can spark disease resembling human MS. To identify the disease-triggering microbes and to reveal their pathogenic activities, we use a special transfer system, transplanting gut material from people with MS (and, for comparison healthy people) to germfree mice with a tendency to develop "MS". The human donors are

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"identical" twins, with one twin with and the other without MS. The recipient animals are transgenic mice, which develop disease in the presence of special gut bacteria, but not on the germfree state. Using this system, we shall present first clues on the nature of MS-causing microbiota.

27 September 2022, 7.30 – 9.30 PM (UTC+9) | 12.30 – 2.30 PM (CEST)
Juntendo University Hospital, Neurology Department, Tokyo, Japan
Parkinson's disease from pathophysiology to treatment

Two hundred years' history of Parkinson's disease | Nobutaka Hattori
The accumulation and propagation of alpha synuclein in synucleinopathies | Taku Hatano
Novel biomarkers for detection of early Parkinson's disease and its progression | Shinji Saiki
Small extracellular vescicles matter in the development of Parkinson's disease | Taiji Tsunemi
Moderator: Taiji Tsunemi

Parkinson's disease is an intractable disorder, which affects more than 8 million people in the world. This workshop covers the recent studies which have unraveled the disease mechanism to pave the way for new treatment options. Prof. Hattori will start with an overview of the history of Parkinson's disease. Then Dr. Hatano will present alpha synuclein propagation theory, which is now considered an essential part of disease development. Dr. Saiki will explain recent advances in biomarkers, which enable us to detect the disease in the early stage. Finally, Dr. Tsunemi will discuss exosomes, which will significantly contribute to the disease progression.

27 September 2022, 12 – 2 PM (UTC-4) | 6 – 8 PM (CEST)
Harvard Medical School, Brigham and Women's Hospital, Ann Romney Center for Neurological Diseases, Boston, United States
Innate immunity in neurodegenerative diseases

Astrocytes | Francisco Quintana
Microglia | Oleg Butovsky
Nasal anti-CD3 treatment of neurodegenerative diseases | Rafael Machado Rezende
Protollin treatment of Alzheimer's disease | Howard Weiner

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28 September 2022, 9 – 11 AM (UTC-7) | 6 – 8 PM (CEST)
UCSF Weill Institute for Neurosciences, University of California San Francisco, San Francisco, United States
Monitoring Neurodegeneration in Multiple Sclerosis

Welcome and Introduction | Steve Hauser
Visualizing Neurodegeneration in Real-time: A BRIDGE to the Clinic | Riley Bove
Stepping into the Future – Remote Monitoring as a Signal for MS Progression | Valerie Block
Spinal Cord Atrophy Precedes and Predicts Progressive MS and Silent Disability Worsening | Roland Henry
Antibody Targets in Multiple Sclerosis | Michael Wilson
Linking Microbes and Diet to Neurodegeneration and Clinical Outcomes: Massive Data Integration with a Knowledge Graph | Sergio Baranzini
Speaker Roundtable Discussion | Steve Hauser
Moderator: Steve Hauser

The UCSF Weill Institute for Neurosciences, in connection with the "Preserving the Brain" conference organized by Fondazione Prada, is pleased to host "Monitoring Neurodegeneration in Multiple Sclerosis." This local workshop will feature a series of brief talks highlighting recent advances in monitoring MS disease progression and exploring how new tools may contribute to improved clinical outcomes. The session will conclude with a moderated roundtable discussion to consider current challenges and opportunities for continued progress in treating patients with multiple sclerosis.

29 September 2022, 7.30 – 9.30 PM (UTC+8) | 1.30 - 3.30 PM (CEST)
Tianjin Medical University General Hospital, Neurology Department, Tianjin, China
Neuroimaging in dementia

Opening | Nan Zhang
Neuroimaging in familial frontotemporal lobar degeneration | Qin Chen
Beijing Aging Brain Rejuvenation Initiative (BABRI): Revealing Neuroimaging Indicators | Jun Wang
Cerebral blood flow pattern measured with Arterial Spin Labeling in subcortical ischemic vascular dementia | Mengya Xing
Discussion | Nan Zhang
Moderator: Nan Zhang

Currently, neuroimaging is the primary method to identify the structural and functional changes of human brain in either aging or diseased conditions in vivo. Clinically routine MRI scan has been suggested to support the diagnosis of different types of dementia in corresponding criteria, such as Alzheimer's disease (AD), behavioral variant of

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frontotemporal dementia, primary progressive aphasia, and vascular dementia, according to brain atrophy or cerebrovascular lesions. Recent neuroimaging findings in persons with dementia, in particular AD, subcortical ischemic vascular dementia, and frontotemporal lobar degeneration, will be presented and discussed in this workshop.

30 September 2022, 10 AM – 12 PM (CEST)
German Center for Neurodegenerative Diseases (DZNE) within the Helmholtz Association, Bonn, Germany
Inflammation and immunological responses in neurodegenerative diseases

Lessons learned from prion diseases | Adriano Aguzzi
Sterile inflammation in neurodegenerative disease | Michael Heneka
Anti-neuronal autoantibodies in neurodegenerative dementia | Harald Pruess
Inflammation in white matter aging and its contribution to neurodegeneration | Mika Simons

Neurodegenerative diseases as well as other diseases of the nervous system are complex disorders involving both local and systemic mechanisms. Recent research strongly suggests that the immune system is involved in the development of neurodegenerative brain diseases. Both innate and acquired immunity play very important roles from the early stages to disease progression. In this session, speakers will address both fundamental and clinical aspects of the immune response in neurodegenerative diseases.

4 October 2022, 2 – 4 PM (CEST)
Hôpital de la Pitié-Salpêtrière, Sorbonne University AP-HP, Neurology Department and Paris Brain Institute, Paris, France
Sleep in neurodegenerative diseases

Role of sleep in cognition and fatigue | Thomas Andrillon
Multiple sclerosis and sleep | Anne Laure Dubessy
Parkinson's disease and sleep | Isabelle Arnulf
Dementia and sleep | Géraldine Rauchs

The human brain has a major weakness: it is prone to fatigue. Recent data indicates that cognitive fatigue corresponds to the intrusion of local sleep-like patterns of neural activity in the awake brain. Plus, nighttime sleep consolidates memory. The brain damages in neurological disorders (Alzheimer disease, Parkinson's disease, and Multiple sclerosis) impacts sleep and vigilance, affecting the patient's quality of life. Conversely, altered sleep may worsen cognitive disturbances and contribute to neurodegeneration.

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HUMAN BRAINS: PRESERVING THE BRAIN
FORUM ON NEURODEGENERATIVE DISEASES

SCIENTIFIC PROGRAM OF THE CONFERENCE

6 OCTOBER 2022

8.30 – 9.00 AM | Opening
Miuccia Prada (President of Fondazione Prada)
Giancarlo Comi (President of the "Human Brains" scientific board)

Introduction
Claudio Bassetti (Past President of the European Academy of Neurology)

9.00 – 10.30 AM | Session 1
Neurodegenerative diseases: epidemiology and risk factors
Fabrizio Tagliavini (Fondazione IRCCS Istituto Neurologico Carlo Besta, Milan)

9.00 – 9.20 AM | Lecture 1
Introductory remarks Kristine Yaffe (UCSF, San Francisco)

9.20 – 9.40 AM | Lecture 2
Aging and risks for dementias Monique Breteler (DZNE, Bonn)

9.40 – 10.00 AM | Lecture 3
Modifiable risks Alberto Ascherio (Harvard Medical School, Boston)

10.00 – 10.30 AM | Open discussion
Microbioma Hartmuth Wekerle (Max Planck, Munich)
Microbioma Sergio Baranzini (UCSF, San Francisco)

11.00 AM – 12.30 PM | Session 2
Genetic and Epigenetic
Sergio Baranzini (UCSF, San Francisco)

11.00 – 11.20 AM | Lecture 1
Dementias John Hardy (UCL, London)

11.20 – 11.40 AM | Lecture 2

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Movement disorders Alexis Brice (ICM, Paris)

11.40 AM – 12.00 PM | Lecture 3
Multiple sclerosis Jorge Oksenberg (UCSF, San Francisco)

12.00 – 12.30 PM | Open discussion
Parkinson disease Thomas Gasser (DZNE, Bonn)
Dystonias Taiji Tsunemi (Juntendo University, Tokyo)

2.00 – 3.30 PM | Session 3
Molecular and cellular mechanisms in ageing and neurodegenerative diseases 1
Pierluigi Nicotera (DZNE, Bonn)

2.00 – 2.20 PM | Lecture 1
Proteostasis mechanisms in aging and neurodegenerative diseases Rick Morimoto (Rice Institute for Biomedical Research, Chicago)

2.20 – 2.40 PM | Lecture 2
Mitochondria Nils-Göran Larsson (Karolinska Institutet, Stockholm)

2.40 – 3.00 PM | Lecture 3
Brain metabolism Jens Bruning (Max Planck Institute, Munich)

3.00 – 3.30 PM | Open discussion
Synaptic dysfunction Monica Di Luca (Università degli Studi di Milano, Milan)
Autophagy – Parkinson disease mitophagy Helen Plun Favreau (UCL, London)

4.00 – 5.30 PM | Session 4
Molecular mechanisms in ageing and neurodegenerative diseases 2
Fredrik Piehl (Karolinska Institutet, Stockholm)

4.00 – 4.20 PM | Lecture 1
Acquired immunity Michal Schwartz (Weizmann Institute, Rehovot)

4.20 – 4.40 PM | Lecture 2
Innate immunity Katerina Akassoglou (UCSF, San Francisco)

4.40 – 5.00 PM | Lecture 3

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Proteinopathies Mathias Jucker (DZNE, Bonn)

5.00 – 5.30 PM | Open discussion
Role of glial cells Jack Antel (McGill University, Canada)
The lesson of age-related macular degeneration Brian Hafler (Yale, New Haven)

5.30 – 6.00 PM | Keynote 1
Pathology of neurodegenerative disease. The basis for the understanding of disease mechanism
Hans Lassmann (Center for Brain Research at the Medical University, Wien)

7.30 – 10.00 PM | Working dinner
Preserving the Brain: a global campaign on modifiable risk factors
Elena Moro (CHU Grenoble)
Giancarlo Comi (President of the "Human Brains" scientific board)

Burden of neurodegenerative diseases Alberto Ascherio (Harvard Medical School, Boston)
Environmental factors Carlo Ferrarese (Università degli Studi di Milano, Milan)
Microbiome and food Sergio Baranzini (UCSF, San Francisco), Gonzalo Torres (CUNY School of Medicine, New York)
Physical exercise Letizia Leocani (UniSR, Milan)
Smoking Tomas Olsson (Karolinska Institutet, Stockholm)
Sleep Luigi Ferini Strambi (IRCCS Ospedale San Raffaele, Milan)

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8.30 – 9.00 AM | Keynote 2
Neurodegenerative disorders and sterile inflammation. Microglia
Michael Heneka (Luxembourg Centre for Systems Biomedicine – LCSB)

9.00 – 10.30 AM | Session 5
Clinical outcomes in phase II-III Clinical Trials
Giovanni Frisoni (IRCCS Fatebenefratelli, Brescia)

9.00 – 9.20 AM | Lecture 1
Dementia Jeffrey Cummings (University of Nevada, Las Vegas)

9.20 – 9.40 AM | Lecture 2
Targeting the glucocerebrosidase pathway. A paradigm for slowing Parkinson's disease
Tony Schapira (UCL, London)

9.40 – 10.00 AM | Lecture 3
Multiple sclerosis Xavier Montalban (Multiple Sclerosis Centre of Catalonia – Cemcat, Barcelona)

10.00 – 10.30 AM | Open discussion
Amyotrophic lateral sclerosis (ALS) Vincenzo Silani (Università degli Studi di Milano, Milan)
Fronto-temporal dementia Nan Zhang (Tianjin Medical University, Tianjin)

11.00 – 12.30 AM | Session 6
Biomarkers in phase II-III Clinical Trials in neurodegenerative diseases
Philip Scheltens (University Medical Center, Amsterdam)

11.00 – 11.20 AM | Lecture 1
Magnetic resonance imaging (MRI) Giovanni Frisoni (IRCCS Fatebenefratelli, Brescia)

11.20 – 11.40 AM | Lecture 2
Positron emission tomography (PET) Daniela Perani (UniSR, Milan)

11.40 AM – 12.00 PM | Lecture 3
Body fluids Henrik Zetterberg (UCL, London)

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12.00 – 12.30 PM | Open discussion
Bodyfluids biomarkers in MS Hans Peter Hartung (Department of Neurology Heinrich-Heine-Universität Düsseldorf, Germany)
Biomarkers in NMOSD and MOGAD Fu-Dong Shi (Tianjin Medical University, Tianjin)

12.30 – 1.00 PM | Keynote 3
Clinical trials design to accelerate therapy
Randall Bateman (St. Louis University, Washington)

2.00 – 3.30 PM | Session 7
Pharmacological treatment
David Hafler (Yale, New Haven)

2.00 – 2.20 PM | Lecture 1
Dementias Cath Mummery (UCL, London)

2.20 – 2.40 PM | Lecture 2
Parkinson's disease Per Svenningsson (Karolinska Institutet, Stockholm)

2.40 – 3.00 PM | Lecture 3
Multiple Sclerosis Stephen Hauser (USCF, San Francisco)

3.00 – 3.30 PM | Open discussion
Amyotrophic lateral sclerosis (ALS) Adriano Chiò (Università degli Studi di Torino, Turin)
Huntington disease Alexandra Durr (ICM, Paris)

4.00 – 5.30 PM | Session 8
Further topics
Alberto Albanese (Humanitas Research Hospital, Milan)

4.00 – 4.20 PM | Lecture 1
Gene therapy Luigi Naldini (San Raffaele Hospital, Milan)

4.20 – 4.40 PM | Lecture 2
The impact of cell therapies in neurodegenerative disorders. Progressive multiple sclerosis and beyond Stefano Pluchino (Cambridge Biosciences Campus, UK)

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4.40 – 5.00 PM | Lecture 3
Rehabilitation – Neuromodulation Letizia Leocani (UniSR, Milan)

5.00 – 5.30 PM | Open discussion
Brain stimulation in Amyotrophic Lateral Sclerosis Vincenzo di Lazzaro (Università Campus Bio-medico, Rome)
DBS in Parkinson Hagai Bergman (Institute of Medical Research, Israel)

5.30 – 6.45 PM | Round table
Together to accelerate therapy for neurodegenerative diseases
Giancarlo Comi (President of the "Human Brains" scientific board)
Pierluigi Nicotera (DZNE, Bonn)

Mario Alberto Battaglia (FISM), Shibeshish Belachev (Biogen), Anna Chiara Carozza (CNR Italy), Carlo Caltagirone (Institute of Research), Tim Coetzee (National MS Society), Frédéric Destrebecq (Brain Council), Maximilian Schuier (Janssen), Alessandro di Rocco (Parkinson League New York), Lucia Faccio (Safinnova Partners), Lice Ghilardi (FRESCO Foundation), John Lehr (Parkinson's Foundation), Chiara Liberati (Axxam), Niels Plath (Biotech MUNA), Marco Salvetti (Industry Forum PMSA), Jan Schadrack (Roche), Davorka Tomic (Merck), Florian Von Raison (Novartis), George Vradenburg (US against Alzheimer organization)

6.45 – 7.00 PM | Concluding remarks and future actions