INTERNATIONAL CONGRESS ON

NEURODEGENERATIVE DISEASES

September 19-21, 2024

Champalimaud Foundation
Lisbon, Portugal

Image: Dancing Astrocytes

CONGRESS SUMMARY 2024

GOLD SPONSOR:

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FUNDACIÓN REINA SOFÍA
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Fundação Champalimaud
INTERNATIONAL CONGRESS ON
NEURODEGENERATIVE DISEASES
September 19-21, 2024
Champalimaud Foundation
Lisbon, Portugal

INDEX

INTRODUCTION

The purpose of this report is to summarize the activities carried out in the **last edition of the International Congress on Neurodegenerative Diseases**, held on **September 19-21, 2024** in Lisbon, Portugal, at the Champalimaud Foundation, on the occasion of World Alzheimer's Day under the Presidency (and attendance) of H.M. Queen Sofia.

The Congress, a joint effort of Spain and Portugal, was organised by the **Queen Sofia Foundation**, the **CIEN** (Centre for Research in Neurological Diseases) and the **Champalimaud Foundation**, and brought together world-leading researchers in a unique platform to share the latest advances in research and treatment of neurodegenerative diseases. The collaboration between the two countries is reaffirmed at a unique and hopeful time, marked by **precision medicine**, advances in **early detection with biomarkers, experimentation with artificial intelligence for diagnosis or the horizon of new drug therapies.**

World-renowned experts, researchers and health professionals have come together in a scientific event that seeks not only to promote the exchange of knowledge and experiences, but also to promote research and raise awareness in society, areas in which the Queen Sofia Foundation and H.M. Queen Sofia have been working for decades.

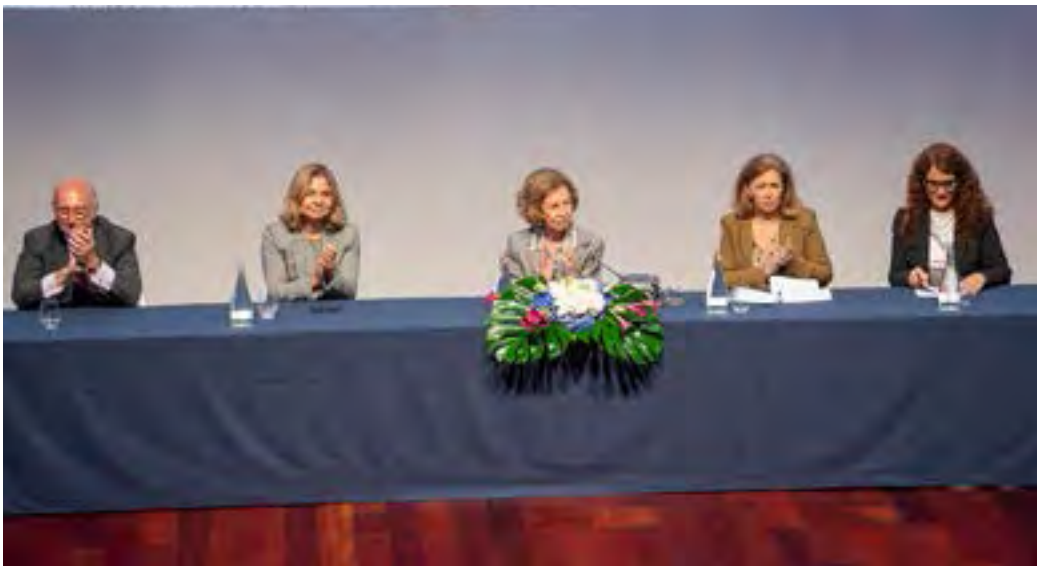
The International Congress on Neurodegenerative Diseases, established as a key event for research professionals in this field, featured a program structured into seven scientific sessions and contributions from more than thirty researchers and representatives of associations from several countries, in an effort to integrate research and care.

Among the most prominent speakers at the congress was **Cath Mummery**, a neurologist at University College London and director of the Dementia Research Centre at the British institution, where she is leading innovative clinical trials for treatments that could modify the course of neurodegenerative diseases such as Alzheimer's disease; **David Wolk**, director of the Penn Alzheimer's Disease Research Center at the University of Pennsylvania, recognised for his pioneering work in research on cognition and ageing, and **Ed Lein**, senior researcher at the Allen Institute for Brain Science at the University of Washington, who contributed his experience in the creation of brain atlases and the use of genomics to better understand these pathologies.

INTRODUCTION

The **scientific program** covered aspects of **early detection, genetic variability, neuroimaging, biomarkers, personalised medicine or non-pharmacological therapies**, and was designed by experts from the Champalimaud Foundation, such as **John Krakauer**, director of the Human Neuroscience Programme; **Joe Paton**, director of Champalimaud Research, and **Marcelo Mendonça**, neurologist and researcher, as well as by **Pascual Sánchez Juan**, scientific director of CIEN.

Several renowned experts from Spain and Portugal presented keynote lectures and participated in debates, including **Joaquim Ferreira**, director of the Laboratory of Clinical Pharmacology and Therapeutics at the University of Lisbon; **Alberto Lleó**, head of Neurology at the Hospital de la Santa Creu i Sant Pau and director of its Memory Unit; and **Agustín Ruiz**, scientific director of the ACE Alzheimer Center Barcelona.



FUNDACIÓN REINA SOFIA

Fundação Champalimaud

INTERNATIONAL CONGRESS ON NEURODEGENERATIVE DISEASES

September 19-21, 2024

Champalimaud Foundation
Lisbon, Portugal

Image: Dancing Astrocytes

Accreditation requested for the International Congress on Neurodegenerative Diseases of the Fundación CIEN with the reference number 57/164553.9/24 to the Commission for the Continuing Training of Health Professions of the Community of Madrid.

Early detection of the pathological components leading to cognitive deterioration

08.30 - 09.00 **REGISTRATION**

09.00 - 11.30 **SCIENTIFIC SESSION I:** NEUROPATHOLOGY

We will start with a neuropathology session proposing that co-pathology is the norm in dementia, and most patients have several components: tau, amyloid, TDP-43, alpha-synuclein

CHAIRPERSON:

ALBERTO RÁBANO
BTCIEN-Fundación CIEN, Madrid. Spain.

SANDRA TOMÉ
KU-Leuven, Belgium.
The impact of TDP-43 pathological synergies in Alzheimer's Disease

GABOR KOVACS
Laboratory Medicine & Pathobiology. University of Toronto. Canada.
Interpretation of mixed pathologies (On-Line)

DIEGO SEPÚLVEDA
UKE Hamburgh. Germany.
New insights in mechanisms for resistant and resilience in familial Alzheimer's disease brains

ED LEIN
Allen Institute, Seattle. USA.
Single cell transcriptomics & disease progression in Alzheimer's Disease

11.30 - 12.00 **COFFEE BREAK & POSTERS**

Early detection of the pathological components leading to cognitive deterioration

SCIENTIFIC SESSION I: NEUROPATHOLOGY

We will start with a neuropathology session proposing that co-pathology is the norm in dementia, and most patients have several components: tau, amyloid, TDP-43, alpha-synuclein

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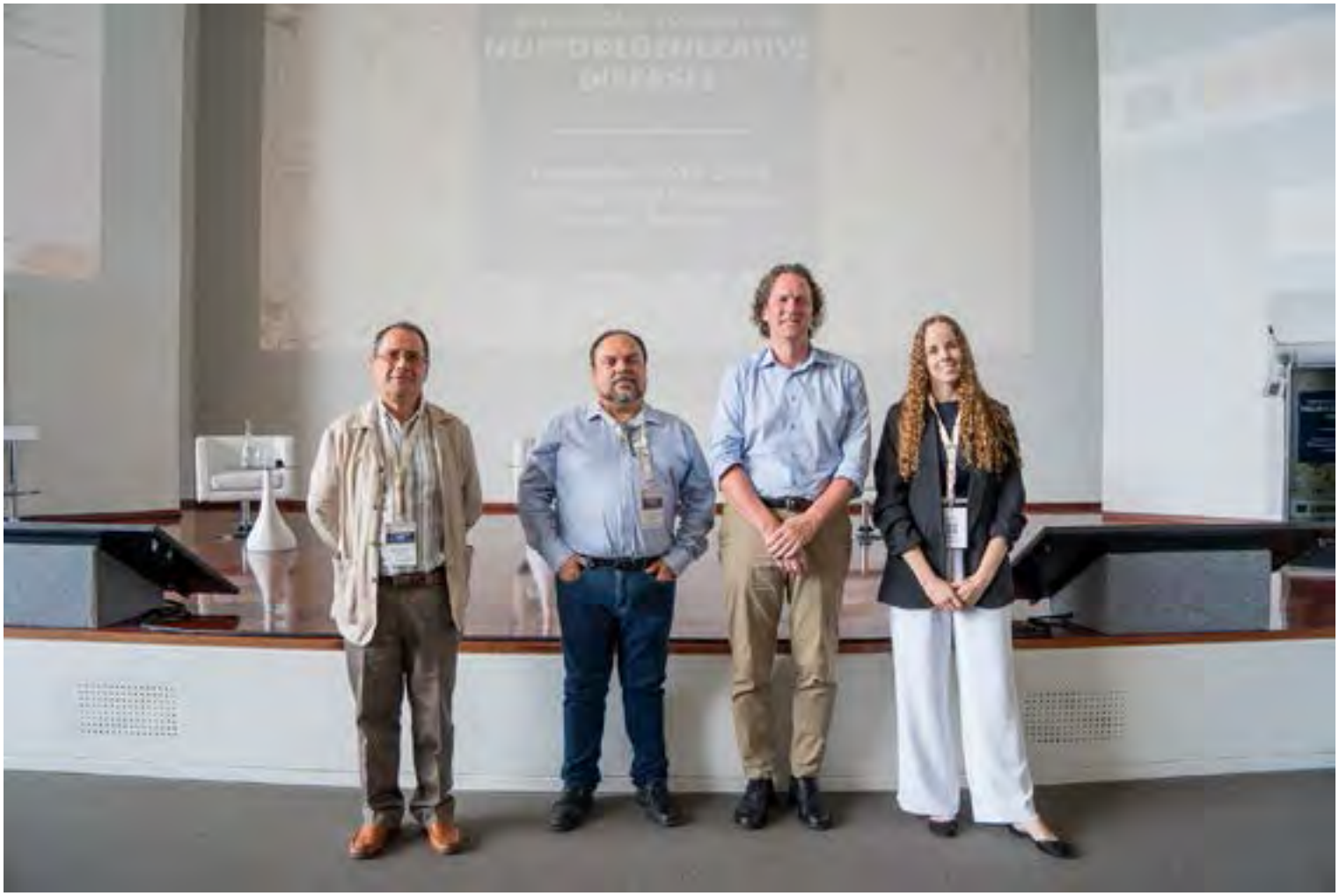
ALBERTO RÁBANO

BTCIEN-Fundación CIEN, Madrid. Spain.



Early detection of the pathological components leading to cognitive deterioration

Sandra O. Tomé
KU Leuven

I am a postdoctoral researcher investigating neuropathology of dementia, at KU Leuven, Belgium. My main research focus is TDP-43 pathology in Alzheimer's Disease, LATE and frontotemporal dementia, using human post-mortem brain tissue and combining histology, multiomics and biophysics. I am also interested in investigating pathological synergies between TDP-43 and different co-pathologies in the context of dementia and how they contribute to neurodegeneration. Ultimately, I aim to improve the neuropathological assessment of age-related pathologies, which may contribute to the development of disease-specific therapeutic strategies.

Early detection of the pathological components leading to cognitive deterioration

Gabor G. Kovacs

University of Toronto

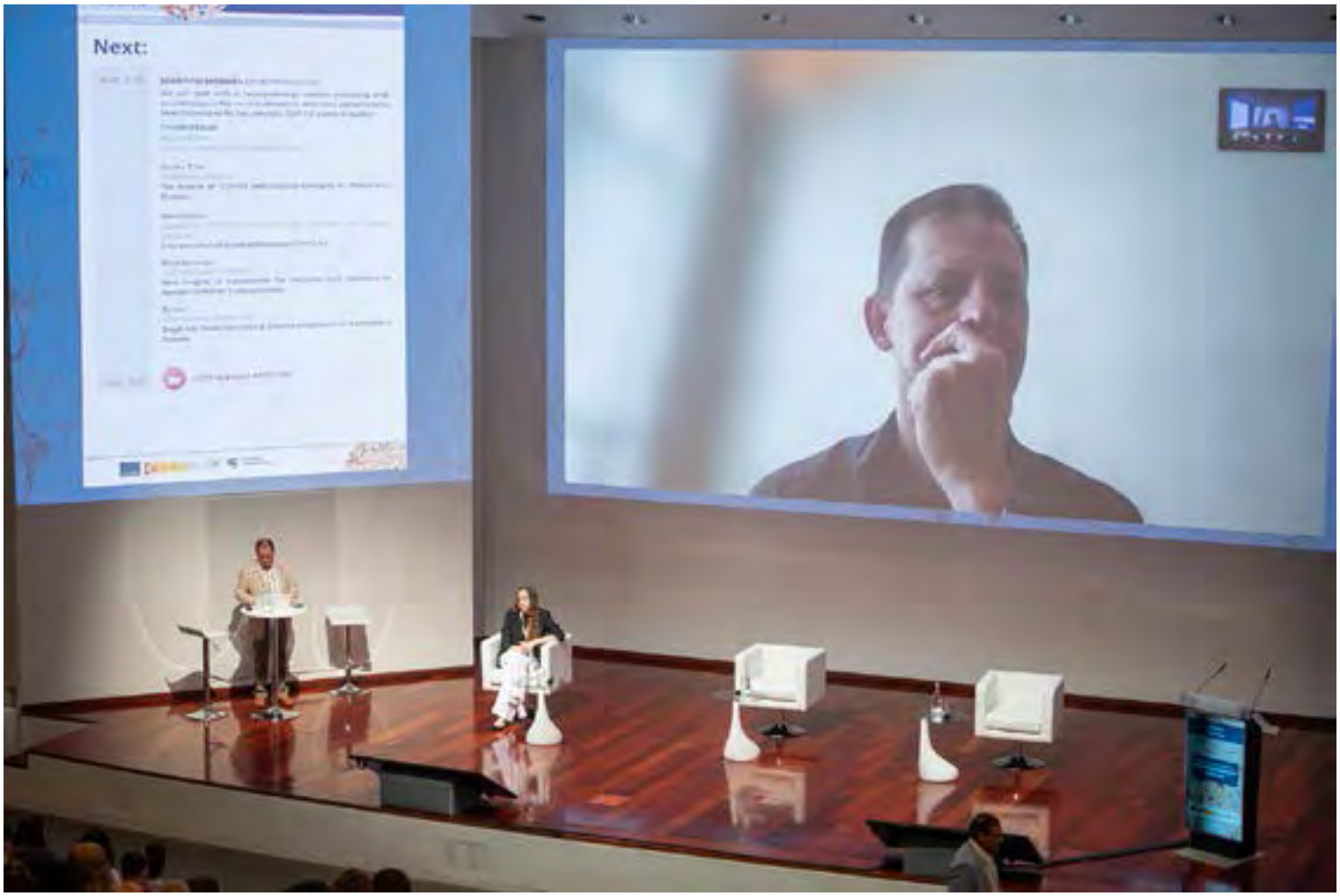
Gabor G. Kovacs MD PhD is Professor of Neuropathology and Neurology at the University of Toronto.

He is Consultant Neuropathologist and Neurologist at the University Health Network (UHN) and a Principal Investigator at the Tanz Centre for Research in Neurodegenerative Disease. Dr. Kovacs is the Co-Director of the Rossy Program for Progressive Supranuclear Palsy Research.

Dr. Kovacs completed his medical training at the Semmelweis University (Budapest, Hungary) where he specialized in Neurology and Neuropathology and obtained a PhD in Neuroscience. From 2004 to 2007, he was the Head of the Department of Neuropathology at the National Institute of Psychiatry and Neurology in Budapest, Hungary. From 2007 to 2019, he was an Associate Professor at the Institute of Neurology at the Medical University of Vienna, Austria. He was the leader of the Hungarian (2004-2019) and Austrian (2011-2019) Reference Center for Human Prion Diseases. Dr. Kovacs has also trained at Indiana University (2007) and University of Pennsylvania (2016 and 2017) as a visiting professor/scholar.

His major research interest is the **neuropathology of neurodegenerative diseases to identify early biomarkers and therapy targets**. His achievements include first descriptions, characterization and pathogenic elucidation of several poorly recognized neurological diseases, including frontotemporal dementia with globular glial inclusions and ageing-related tau-astrogliopathy (ARTAG). He coordinated a study and described the sequential distribution of tau pathology in progressive supranuclear palsy, which allows staging of disease. In addition, Dr. Kovacs has made fundamental descriptions and advances in the pathogenic, genetic, neuropathologic and epidemiologic studies on human tau, alpha-synuclein and prion protein- diseases. He has published more than 380 peer-reviewed papers and edited three books on Neuropathology.

Early detection of the pathological components leading to cognitive deterioration

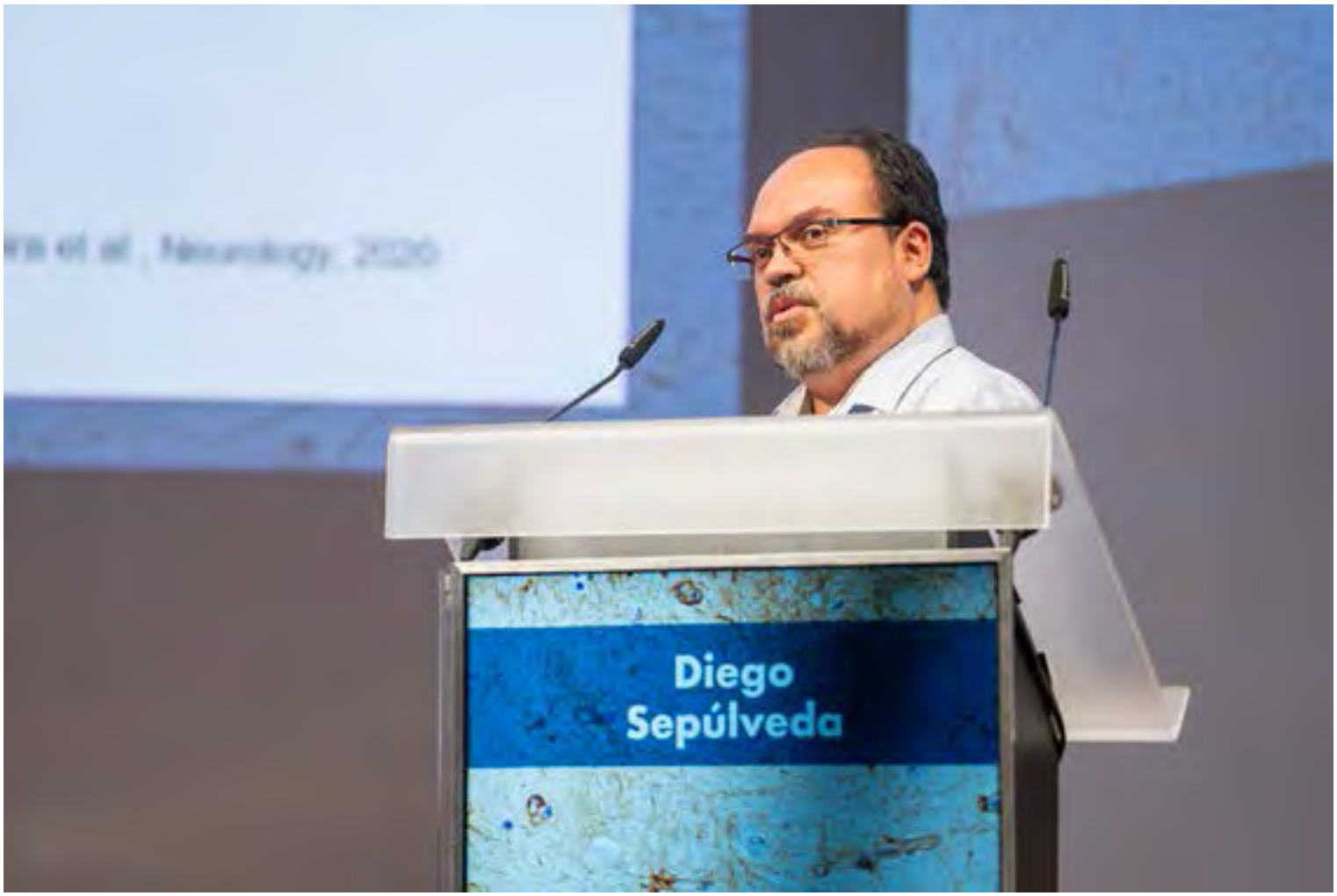


Early detection of the pathological components leading to cognitive deterioration

Diego Sepúlveda
Institute of Neuropathology
UKE Research. Hamburg

Dr. Sepulveda-Falla studied medicine at the University of Antioquia in Medellín, Colombia, and obtained a Doctor of Medicine degree from the University of Hamburg, Germany. He followed with a postdoctoral position at the Institute of Neuropathology of the University Medical Center Hamburg-Eppendorf in the same city, focusing on neuropathology of Alzheimer's disease. In the same institution he became a research group leader, and he is currently the head of the Laboratory of Molecular Neuropathology of Alzheimer's Disease in this center. Dr. Sepulveda-Falla started his interest in Alzheimer's disease as a brain donation assistant, which motivated him to follow a career researching the pathobiology of Alzheimer's disease. Dr. Sepulveda-Falla conducted the identification of the underlying cause of cerebellar symptoms in familial Alzheimer's disease, spotting Ca2+ homeostasis in the mitochondria as a key factor in Purkinje Cells neurodegeneration. Following this clue, he developed cellular and animal models for the exploration of the role for gamma secretase in mitochondria. His group focuses on the characterization of disease heterogeneity in more than 100 brains donated by members of the PSEN1 E280A population, identifying the association between the degree of severity of tau pathology and age of disease onset. His lab is currently using high throughput molecular, and machine learning-assisted neuropathological techniques to perform deep phenotyping of Alzheimer's brains. Recently, Dr. Sepulveda-Falla described the neuropathological findings in two outstanding cases protected against the disease for more than three decades.

Early detection of the pathological components leading to cognitive deterioration



Ed Lein

Allen Institute for Brain Science

Ed Lein is a Senior Investigator at the Allen Institute for Brain Science and an Affiliate Professor in the Departments of Neurological Surgery and Laboratory Medicine and Pathology (DLMP) at the University of Washington. He received a B.S. in biochemistry from Purdue University and a Ph.D. in neurobiology from UC Berkeley, and performed postdoctoral work at the Salk Institute for Biological Studies.

Ed joined the Allen Institute in 2004 and has provided scientific leadership for the creation of large-scale anatomical, cellular and gene expression atlases of the adult and developing mammalian brain as catalytic community resources, including the inaugural Allen Mouse Brain Atlas and a range of developmental and adult human and non-human primate brain atlases. Current research interests involve the use of single cell genomics as a core phenotype to understand brain cellular organization, mammalian conservation and human specificity, define cellular vulnerability in disease, and identify regulatory elements that allow cell type-specific targeting and manipulation.

He leads the Human Cell Types Department, which aims to create comprehensive cell atlases of the human and non-human primate brain, understand what is disrupted in Alzheimer's disease, and create tools for precision genetic targeting of brain cell types as transformative tools for basic neuroscience and gene therapy. He is also a member of the BRAIN Initiative Cell Atlas Network (BICAN), a member of the Organizing Committee of the Human Cell Atlas (HCA), and a CIFAR fellow.

Ed's areas of expertise include developmental neurobiology, structural and cellular neuroanatomy, transcriptomics and epigenomics, comparative neurobiology, and Alzheimer's disease. His research program work encompasses brain cell atlasing, comparative neurobiology, Alzheimer's disease, and gene therapy.

Early detection of the pathological components leading to cognitive deterioration

Early detection of the pathological components leading to cognitive deterioration

COFFEE BREAK & POSTERS

Early detection of the pathological components leading to cognitive deterioration

12.00 - 13.00

SCIENTIFIC SESSION II: GENETIC VARIABILITY

In the following session, we will address how genetic variability is associated with the risk of AD and some of the most frequent co-pathologies (alpha-synuclein, TDP43, vascular...) and how it can potentially be used for diagnostic and/or risk classification. We will also explore brain resilience from a genetic perspective.

CHAIRPERSON:

AGUSTÍN RUIZ

Fundació ACE-CIBERNED, Barcelona. Spain.

ALFREDO RAMÍREZ

Cluster of Excellence for Aging Research. University of Cologne. Germany.

Inflammatory pathways operating at the intersection of ageing and Alzheimer's disease modulate the speed of cognitive decline

HENNE HOLSTEGE

Alzheimer Center Amsterdam. VUmc. Amsterdam UMC. Netherlands.

How genetics help to maintain a young and healthy brain: lessons from cognitively healthy centenarians

13.00 - 15.00

LUNCH & POSTERS

Early detection of the pathological components leading to cognitive deterioration

SCIENTIFIC SESSION II: GENETIC VARIABILITY

In the following session, we will address how genetic variability is associated with the risk of AD and some of the most frequent co-pathologies (alpha-synuclein, TDP43, vascular...) and how it can potentially be used for diagnostic and/or risk classification. We will also explore brain resilience from a genetic perspective.

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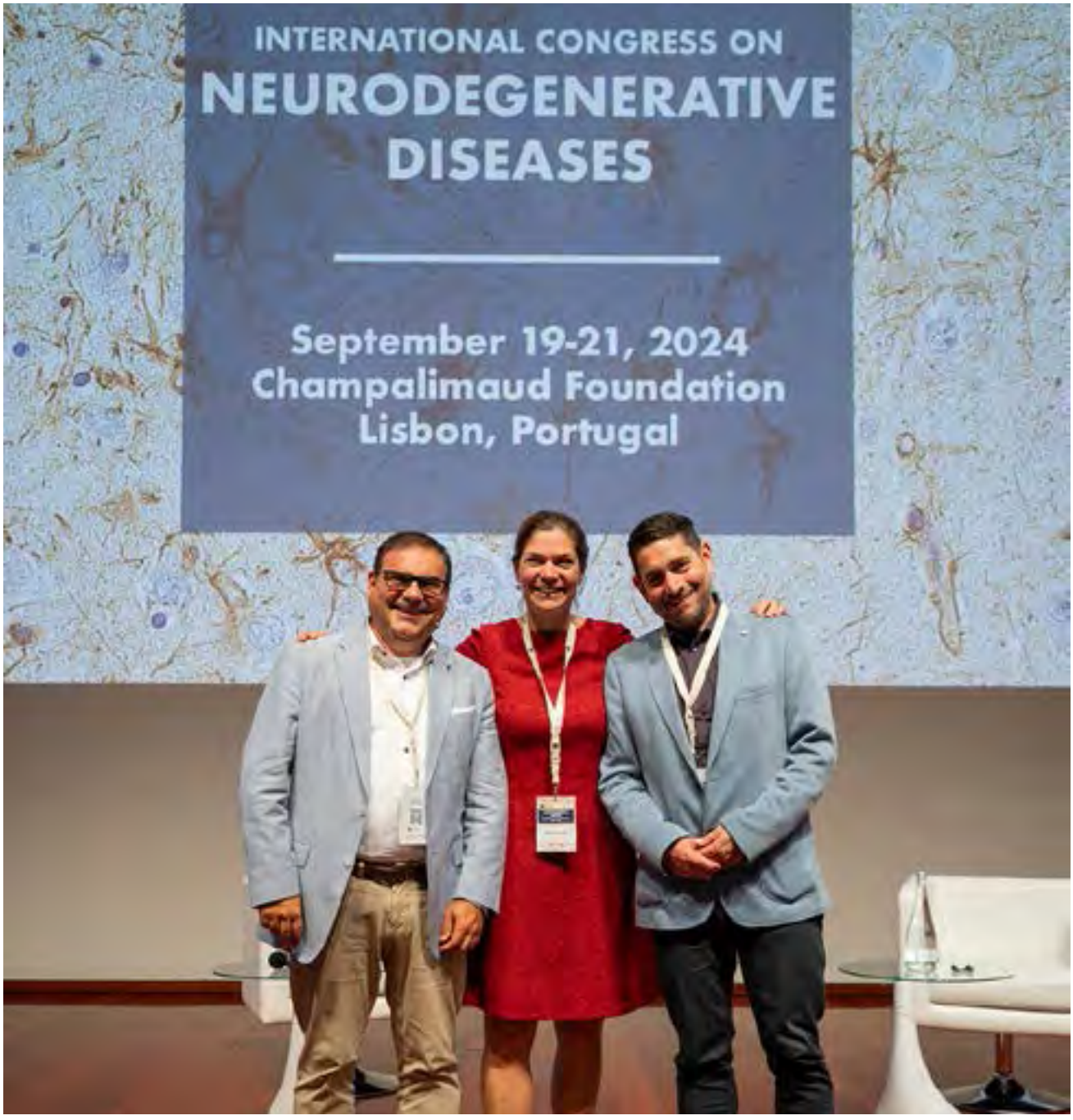


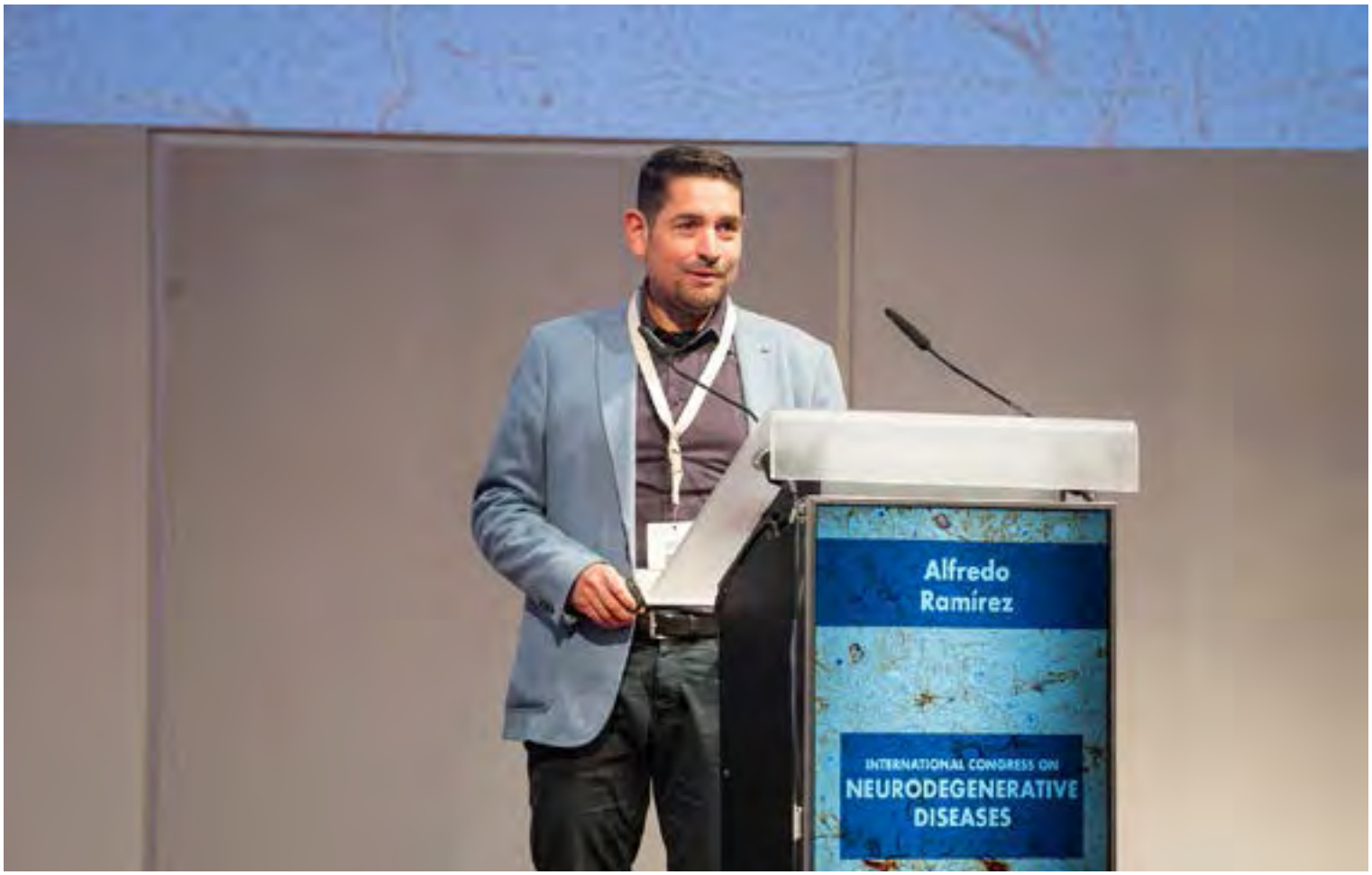
Early detection of the pathological components leading to cognitive deterioration

Alfredo Ramírez Zúñiga
CECAD-University of Cologne

The main objective of Dr. Ramirez's laboratory is the identification of the genetic and epigenetic causes of cognitive disorders such as Alzheimer's disease. His group has created the basis for research on the genetics and epigenetics of prodromal Alzheimer's disease and its progression through the creation of international collaborations. In this context, Dr. Ramirez has collected large genetic and epigenetic data sets in the context of international consortia dedicated to the genetics of neurodegeneration. He is an active member of the largest European consortium in Alzheimer's disease genetics, the "European DNA Bank for deciphering the missing heritability of Alzheimer's disease" (EADB consortium).

In 2016, he started the Neurogenetics and Molecular Psychiatry division in the Department of Psychiatry at the University of Cologne, expanding his research area to the molecular biology of aging as a major risk factor for Alzheimer's disease. His division has been specifically interested in cellular senescence as one of the fundamental processes occurring during aging. In this regard, Dr. Ramirez's team has explored whether the levels of specific proteins in cerebrospinal fluid belonging to the so-called senescence-associated secretory phenotype (SASP) are associated with accelerated biological aging and whether this acceleration influences the progression to Alzheimer's type dementia.

Early detection of the pathological components leading to cognitive deterioration

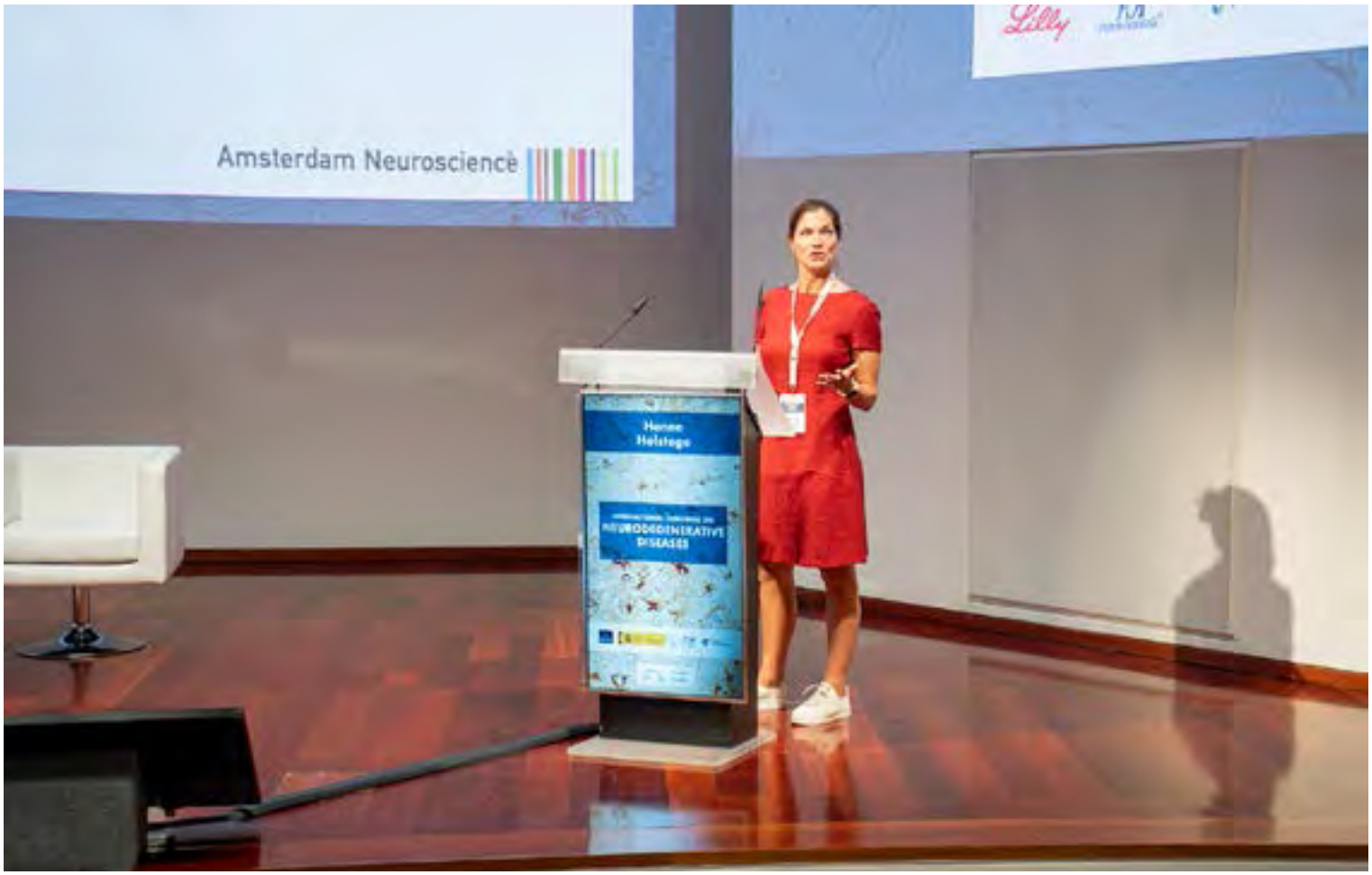


Early detection of the pathological components leading to cognitive deterioration

Henne Holstege
Amsterdam UMC

Henne Holstege majored in biochemistry at the University of Leiden. She spent a year at Harvard University in Boston, where she investigated the molecular mechanisms of satiety, and the influence of genetics on this. She went on to do her PhD at the Netherlands Cancer Institute where she studied the somatic genetic aberrations associated with the development of breast cancer. During her PhD, Dr. Henne Holstege was intrigued by how it is possible that some people manage to reach extreme ages in good health and without cognitive decline. To identify protective genetic and biomolecular factors that associate with the escape of cognitive decline, Holstege set up the 100-plus Study, a cohort study of cognitively healthy centenarians. Currently the cohort includes more than almost 500 centenarians from whom Holstege collects brain tissues, blood samples and faeces samples, in order to investigate genetics, neuropathology, neuropsychology, immunology associated with escaping cognitive decline. While on one hand Holstege's research focus lies on identifying protective molecular constellations that make the centenarians stand out, she realizes that this is tightly linked with the genetics of (early onset) Alzheimer's Disease; both extremes on the same cognitive spectrum. Therefore, her lab is involved in large international collaborative joint analysis of sequencing data thousands of Alzheimer Disease cases and cognitively healthy controls the collected by European ADES and American ADSP consortia. Holstege and collaborators are currently identifying novel genes associated with the increased or decreased risk of AD. For more information see: www.holstegelab.eu

Early detection of the pathological components leading to cognitive deterioration



Early detection of the pathological components leading to cognitive deterioration

LUNCH & POSTERS

Early detection of the pathological components leading to cognitive deterioration

15.00 - 16.30

SCIENTIFIC SESSION III: NEUROIMAGING

We will deepen into neuroimaging techniques for detecting different dementia-related brain alterations reflecting distinct pathologic components.

CHAIRPERSON:

MICHEL GROTHE
Fundación CIEN, Madrid. Spain.

DAVID WOLK
Penn Alzheimer's Disease Research Center. University of Pennsylvania. USA.
Neuroimaging Clues to LATE and Other Common AD Co-Pathologies

JESÚS SILVA
Fundación CIEN, Madrid. Spain.
Hypometabolic signatures of LATE-NC and Lewy Body pathology. Unveiling the heterogeneity of amnestic syndromes

LAURA JONKMAN
Amsterdam Neuroscience - Brain Imaging (VUmc). Amsterdam UMC. Netherlands.
From MRI to microscope: translating neuropathology

16.30 - 17.00

COFFEE BREAK & POSTERS

Early detection of the pathological components leading to cognitive deterioration

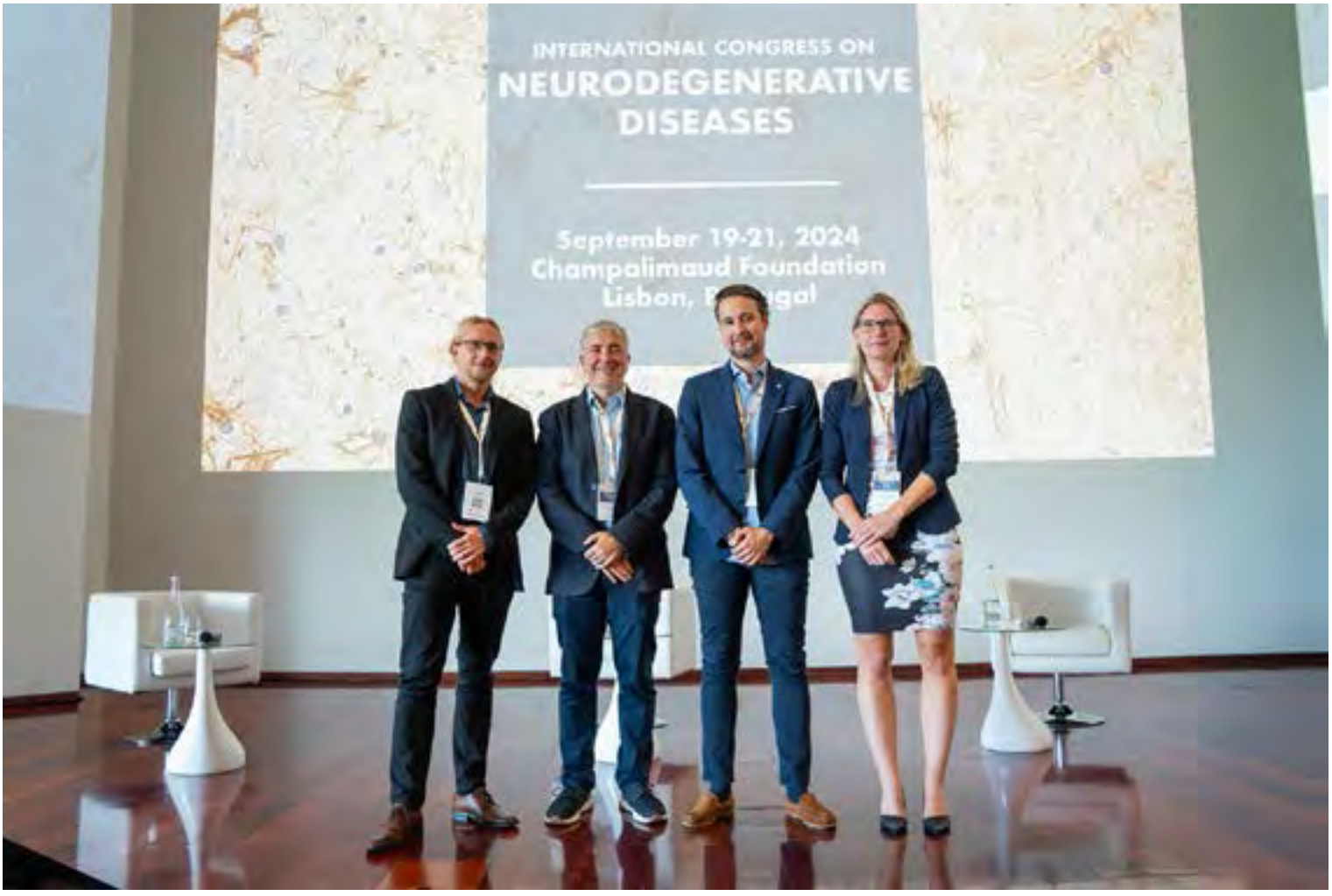
SCIENTIFIC SESSION III: NEUROIMAGING

We will deepen into neuroimaging techniques for detecting different dementia-related brain alterations reflecting distinct pathologic components.

CHAIRPERSON:

MICHEL GROTHE

Fundación CIEN, Madrid. Spain.



Early detection of the pathological components leading to cognitive deterioration

David Wolk

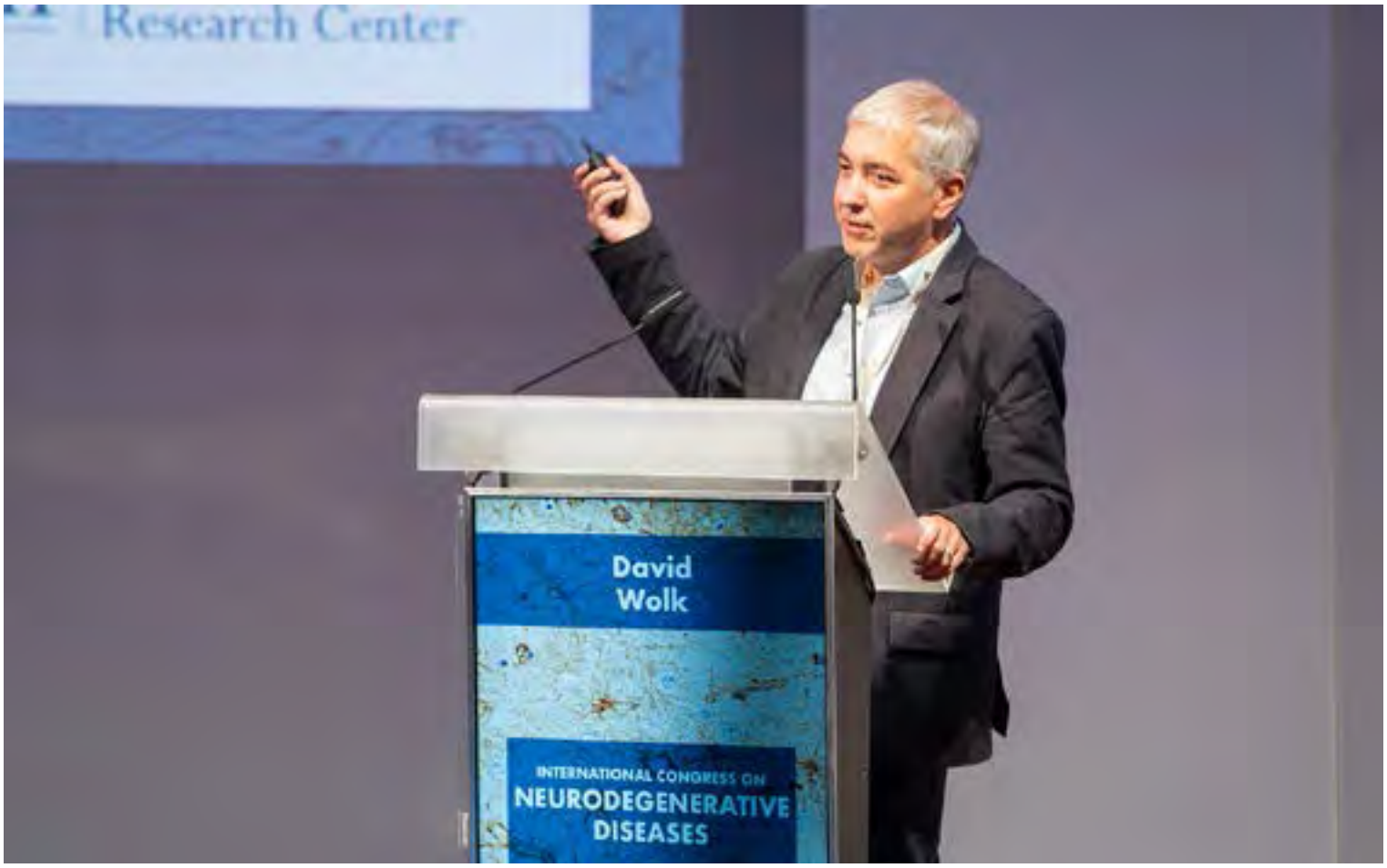
Penn Alzheimer's Disease Research Center
University of Pennsylvania

Dr. David Wolk is Professor of Neurology, Chief of the Division of Cognitive Neurology, Director of the National Institute of Aging funded Penn Alzheimer's Disease Research Center, Co-Director of the Penn Institute on Aging, and Co-Director of the Penn Memory Center.

Dr. Wolk's primary clinical interest has been in the diagnosis and care of individuals with a variety of neurodegenerative conditions. His research has focused on the cognitive neuroscience of memory decline associated with aging and Alzheimer's Disease using techniques including behavioral testing, structural and functional MRI, and FDG and molecular PET imaging. Much of this work is also directed at examining biomarkers, including behavioral and neuroimaging, that differentiate healthy aging from the earliest transition to AD and their potential role in understanding disease mechanisms and incorporation into treatment trials. Another related thread of his work has been to better understand, classify and predict sources of heterogeneity in AD. Dr. Wolk has had sustained NIH support since 2003 and has been the principal or co-investigator on numerous local, national and international studies, including therapeutic trials.

Dr. Wolk completed his medical training at Johns Hopkins University, a Neurology residency at the University of Pennsylvania, and clinical Fellowship training in Cognitive and Behavioral Neurology at Brigham and Women's Hospital/Harvard Medical School; where he also completed a post-doctoral research fellowship studying memory in Alzheimer's Disease. Amongst a number of honors, he is the recipient of the American Academy of Neurology's Norman Geschwind Prize in Behavioral Neurology.

Early detection of the pathological components leading to cognitive deterioration



Jesús Silva-Rodríguez

Fundación CIEN

After completing my formal university training in Physics, in 2012 I started a research career focused on the technical aspects of PET imaging. In 2017, I obtained my PhD (‘Cum Laude' distinction) in Medicine from the University of Santiago de Compostela (Spain). During my PhD I developed SimPET, a novel tool for Monte Carlo simulation currently used by research groups around the world to test the robustness of neuroimaging biomarkers (https://github.com/txusser/simpet). To date, I continue leading the SimPET project as an active national multicenter consortium. As of the time of writing, 2 Ph.D. students are developing their thesis projects using SimPET.

Since the start of my career, I have also been active in the field of clinical and preclinical neuroimaging, as evidenced by my significant scientific publications in the field. These publications showcase my expertise in processing various imaging modalities, including PET, MRI, and SPECT. In 2015 I co-founded Qubiotech Health Intelligence SL (www.qubiotech.com), a startup commercializing neuroimaging quantification software. Serving as the company's CTO from 2017 to 2020, I led a team of 5 developers and successfully brought to market our most successful product, Neurocloud (www.neurocloud.es/en). Neurocloud, which incorporates automated pipelines for analyzing FDG and amyloid PET, MRI T1 and T2/FLAIR images (volumetry and white matter lesion detection), and dopaminergic SPECT, among others, is an approved medical device in the EU and is currently utilized by over 60 client hospitals in Europe. In 2020, I chose to further pursue my passion for research by joining the Molecular Imaging Group at the Health Research Institute of Santiago de Compostela (IDIS) as a postdoctoral researcher, where I led neuroimaging processing tasks in the NeuroAtlantic project (https://www.neuroatlantic.eu/). In January 2022, after being awarded the prestigious "Sara Borrell Fellowship" by the Spanish Ministry of Health, I initiated a promising research line at the Movement Disorders Group in the Institute of Biomedicine of Seville (IBIS), examining the role of Lewy Body (co-)pathology in neurodegeneration patterns and clinical presentations in clinical Alzheimer’s Disease. As of January 2024, I have joined the CIEN Foundation, Reina Sofia Alzheimer Centre, in Madrid as a postdoctoral researcher.

Throughout recent years, I have published several studies on neurodegenerative diseases, including Alzheimer’s Disease, Lewy body disorders, primary age-related tauopathy (PART), and limbic age-related TDP-43 encephalopathy (LATE). I have authored 39 research articles in international peer-reviewed journals, with 10 articles as the first author and 6 as a corresponding or senior author, in some of the leading journals in the field, such as JAMA Neurology, Brain, Movement Disorders, and Alzheimer’s & Dementia. My current H-index is 18, with more than 800 citations. During my career, I have been honored with several awards for my outstanding trajectory in technology transfer, including the “Best Technology Transfer” by the Royal Galician Sciences Academy and the “Barrié de la Maza Award” by the Royal Galician Medicine Academy, among others. I have also supervised and mentored numerous trainees and doctoral students, contributing to the next generation of researchers in the field.

Early detection of the pathological components leading to cognitive deterioration

Early detection of the pathological components leading to cognitive deterioration

Laura Jonkman
Amsterdam UMC

DR. Assistant Professor, Anatomy and neurosciences

PI from 2019-2024

Specialization

MRI, pathology, neurodegeneration

Focus of research

My main research aim is to translate post mortem MRI signatures of neuropathology and neurodegeneration to the clinical setting, in order to facilitate a better interpretation of MRI datasets, and contribute to a better diagnosis, prognosis and patient stratification.

For this, we set up a standardized pipeline including Alzheimer's disease, Parkinson's disease, and other dementia's as well as non-neurological control donors, all undergoing the same 3T in-situ MRI and autopsy protocol with subsequent pathological assessment.

With this approach, my research line is threefold: studying the influence of pathological and neuronal markers on (i) cortical alterations, (ii) specific nuclei and their projections, and (ii) the integrating and segregating brain network.

Early detection of the pathological components leading to cognitive deterioration

Early detection of the pathological components leading to cognitive deterioration

COFFEE BREAK & POSTERS

Early detection of the pathological components leading to cognitive deterioration

17.00 - 18.30

SCIENTIFIC SESSION IV: BIOMARKERS

Discussing the detection of neuropathological components in the periphery using biochemical markers

CHAIRPERSON:

PASCUAL SÁNCHEZ JUAN

Fundación CIEN, Madrid. Spain.

GEMMA SALVADÓ

Clinical Memory Research. Lund University. Sweden.

Current status of fluid biomarkers in research, clinical setting and trials

ANJA SCHNEIDER

Translational Dementia Research. German Center for Neurodegenerative Diseases-DZNE. University of Bonn. Germany.

Extracellular vesicle biomarkers in neurodegenerative diseases

MARC SUÁREZ-CALVET

Barcelona Beta Brain Research Center. Hospital del Mar, Barcelona. Spain.

Blood-based Biomarkers in Cognitively Unimpaired Individuals at-risk of Alzheimer's disease

Early detection of the pathological components leading to cognitive deterioration

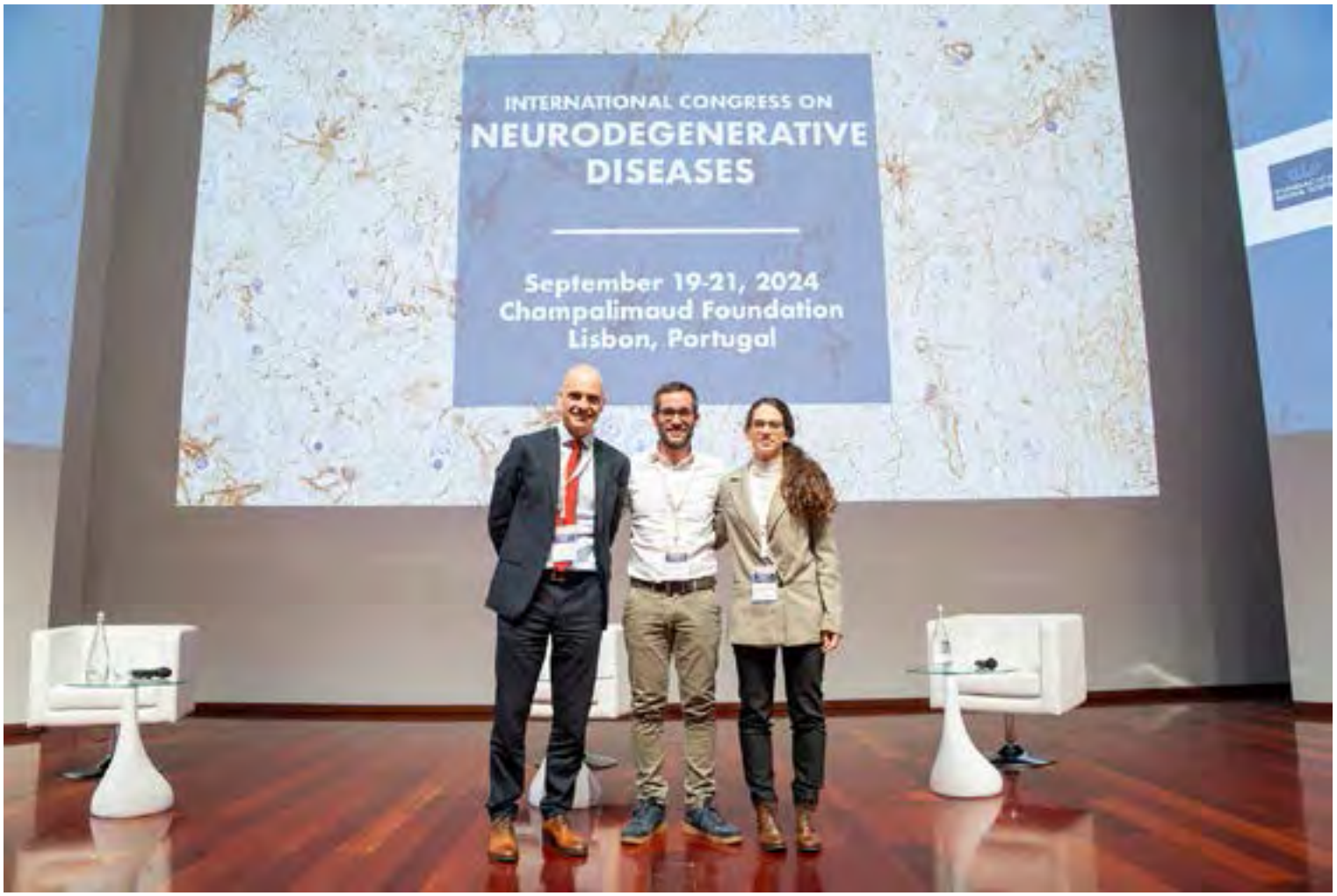
SCIENTIFIC SESSION IV: BIOMARKERS

Discussing the detection of neuropathological components in the periphery using biochemical markers

CHAIRPERSON:

PASCUAL SÁNCHEZ JUAN

Fundación CIEN, Madrid. Spain.



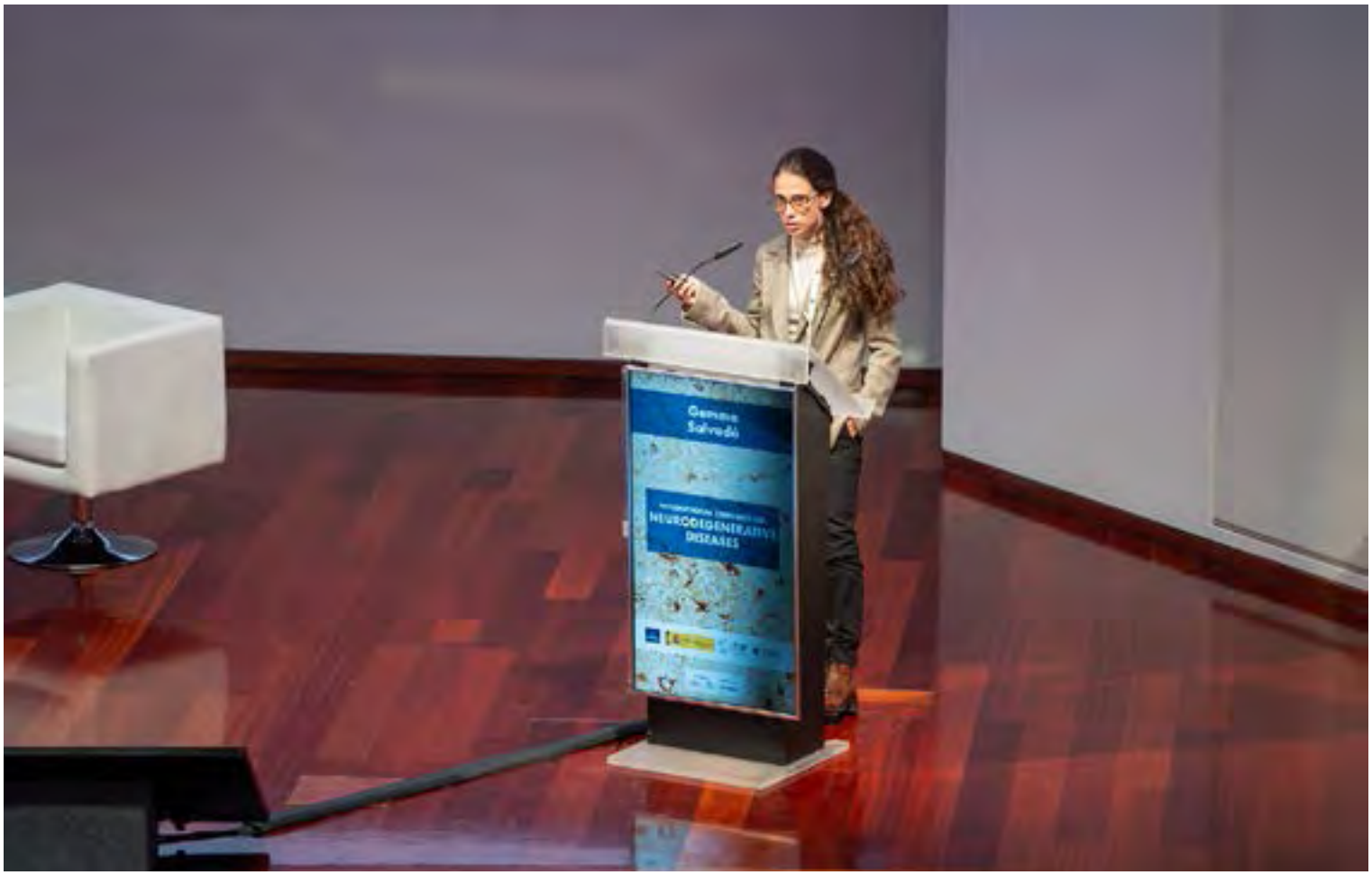
Early detection of the pathological components leading to cognitive deterioration

Gemma Salvadó Blasco
Lund University

Gemma Salvadó, PhD, is an associate researcher at Lund University, Sweden. She was awarded the prestigious Marie Skłodowska-Curie postdoctoral fellowship for her exceptional research contributions. Gemma earned her PhD at the Barcelonaβeta Brain Research Center, Spain, focusing on the preclinical stages of Alzheimer's disease using neuroimaging and fluid biomarkers. Her current research investigates plasma and cerebrospinal fluid biomarkers to understand disease progression.

Gemma has authored 48 peer-reviewed articles, with 18 as the first author, published in high-impact journals such as *Nature Medicine, Nature Aging, Molecular Neurodegeneration, and EMBO Molecular Imaging*. Her work has garnered significant recognition, reflected in a substantial increase in citations in recent years (1,558 citations on Scopus and 1,955 on Google Scholar, h-index = 23/24).

Early detection of the pathological components leading to cognitive deterioration



Early detection of the pathological components leading to cognitive deterioration

Anja Schneider
DZNE. University of Bonn

Degrees

2011 Venia legendi, University of Göttingen

2011 Board certification Psychiatry and Psychotherapy

2003 MD thesis, University of Hamburg

Major previous appointments

since 2016 Head, Dept. Neurodegenerative Diseases and Gerontopsychiatry, Univ Clinic Bonn

since 2016 Group Leader, DZNE Bonn (W3, clinical dementia research)

2014-2016 Group Leader, DZNE Göttingen (W2, translational dementia research)

2011-2016 Deputy head, Clinical Dementia Center, University Medical Center Göttingen.

2012-2014 Heisenberg Fellowship (German Research Foundation), Guest Group, MPI Experimental Medicine, Göttingen

Activities in the Scientific Community / Professional Memberships

Since 2017 Research Committee, Medical Faculty, University Bonn, Bonn

Since 2017 Scientific Advisory Board, German PSP Society.

2015-2017 Associated member, European Neuroscience Institute, Göttingen

Honors and Awards

2015 Research Award, German Society for Psychiatry and Psychotherapy

2012 Heisenberg Fellowship, German Research Foundation

2008 Steinberg Krupp Award for Alzheimer research

Early detection of the pathological components leading to cognitive deterioration

Early detection of the pathological components leading to cognitive deterioration

Marc Suárez Calvet
Barcelonaβeta Brain Research Center (BBRC),
Pasqual Maragall Foundation.
Servei de Neurologia, Hospital del Mar, Barcelona

Marc Suárez-Calvet MD PhD is a clinical neurologist with expertise in Alzheimer's disease and other neurodegenerative diseases. He is the Group Leader of the Fluid Biomarkers and Translational Neurology Research Group at Barcelonaβeta Brain Research Center and at the Neurology Department of Hospital del Mar (Barcelona, Spain).

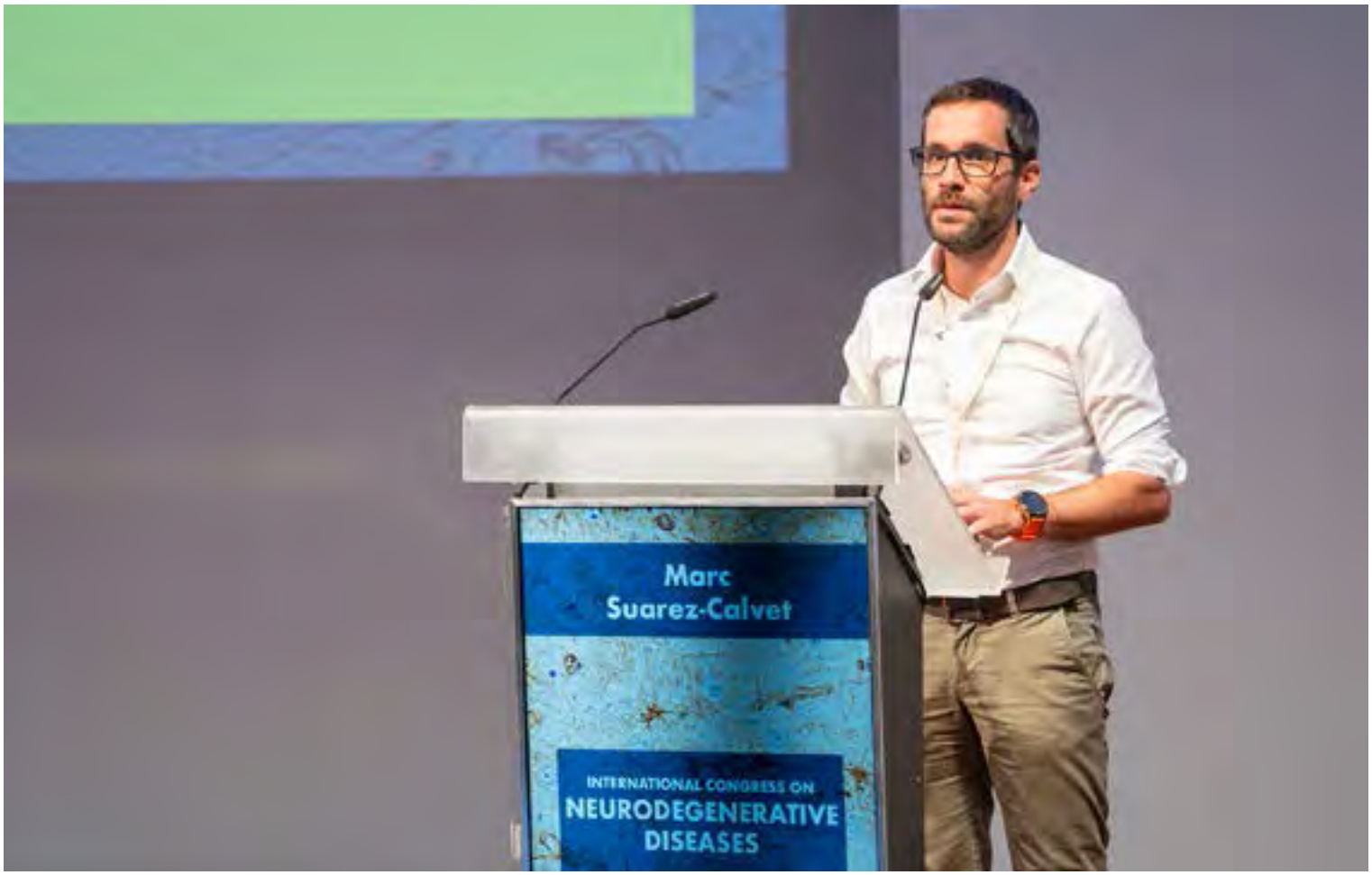
Recipient of the prestigious European Research Council (ERC) Starting Grant, Dr. Suárez-Calvet directs his efforts towards the investigation of blood proteins associated with neurodegeneration and brain aging. His studies on blood biomarkers have been featured in journals such as Nature Medicine, JAMA Neurology, and Neurology.

After earning his medical degree at Universitat Autònoma de Barcelona (2004), he completed his Neurology residency at Hospital de la Santa Creu i Sant Pau (2009), followed by a research fellowship at Harvard University. Between 2012 and 2017, he served as a researcher at Prof Christian Haass Lab at Ludwig-Maximilians Universität and the Deutsches Zentrum für Neurodegenerative Erkrankungen in Munich, Germany. During this period, he led groundbreaking translational projects, uncovering the first post-translational modification specifically associated with FTLD-FUS, namely monomethylated arginine (MMA) FUS, and initiating a biomarker project centered on sTREM2, a microglial biomarker.

In 2017, Dr Suárez-Calvet held an honorary clinical assistant position at the Dementia Research Center in the National Hospital for Neurology and Neurosurgery (UCLH), London, UK, under the guidance of Prof Nick Fox. He completed a fellowship at the University of Gothenburg under the mentorship of Prof. Blennow.

In 2018, he joined the Barcelonaβeta Brain Research Center (BBRC) and combines his research activity with a clinical appointment in Hospital del Mar. Recognizing his outstanding contributions, he was honored with the "Alzheimer Prize 2021" from the Spanish Society for Neurology.

Early detection of the pathological components leading to cognitive deterioration



Personalized Interventions. Secondary prevention

09.30 - 11.00

SCIENTIFIC SESSION V: PHARMACOLOGICAL INTERVENTION

We will discuss pharmacological interventions, specifically tackling some of the main pathological components of dementia

CHAIRPERSONS:

ALBERTO LLEÓ
Unidad de Memoria. Hospital de Sant Pau, Barcelona. Spain.

LUISA ALVES
Universidade NOVA de Lisboa. Portugal.

MALÚ GAMEZ TANSEY
Center for Translational research in Neurodegenerative disease. University of Florida. USA.
Targeting immune dysfunction and chronic inflammation

TIAGO OUTEIRO
Goettingen University. Germany.
From biology to classification: understanding Parkinson's disease

CATHERINE MUMMERY
University College London. UK.
Beyond amyloid: emerging tau therapies in Alzheimer's disease

11.00 - 11.30

COFFEE BREAK & POSTERS

Personalized Interventions. Secondary prevention

SCIENTIFIC SESSION V: PHARMACOLOGICAL INTERVENTION

We will discuss pharmacological interventions, specifically tackling some of the main pathological components of dementia

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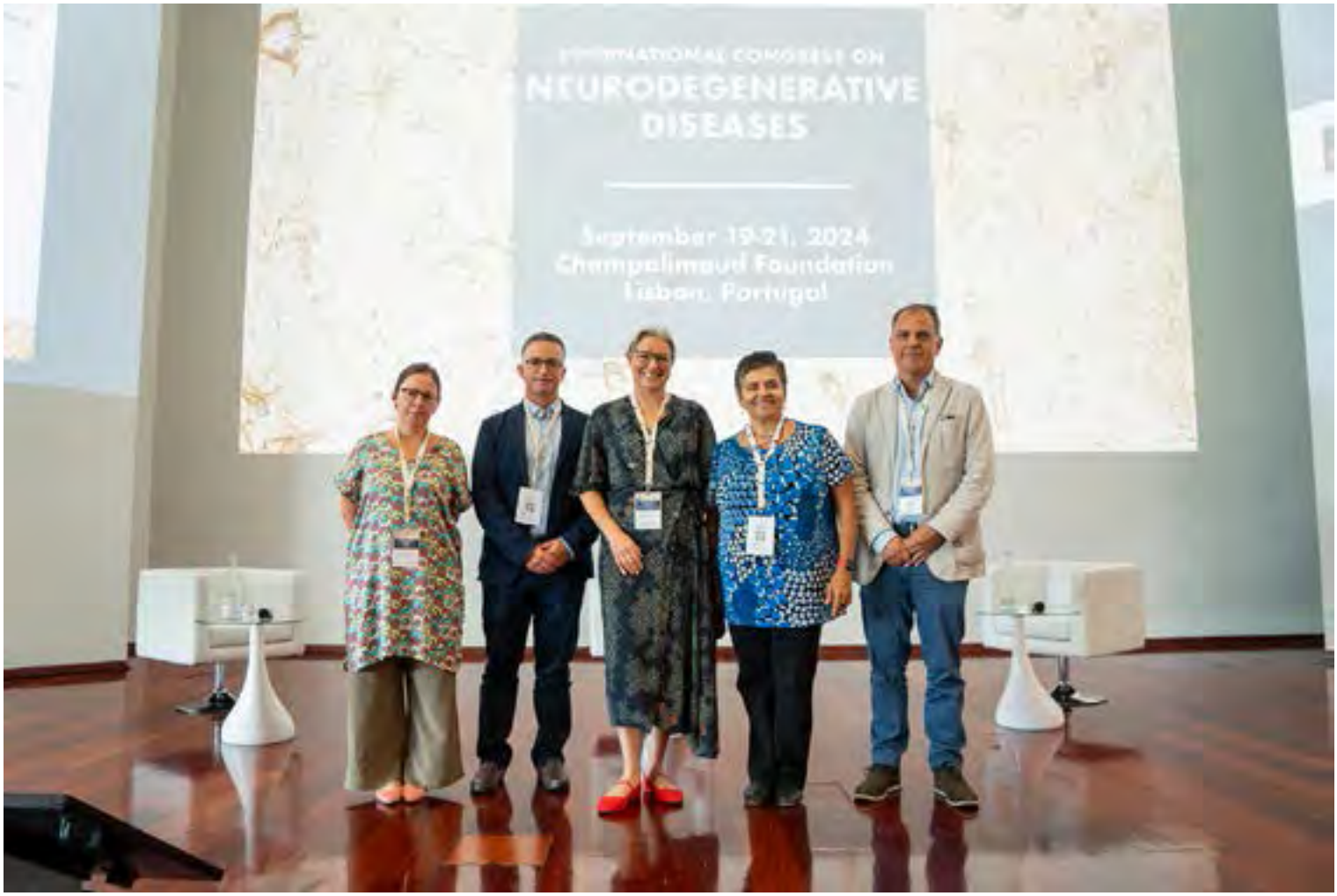
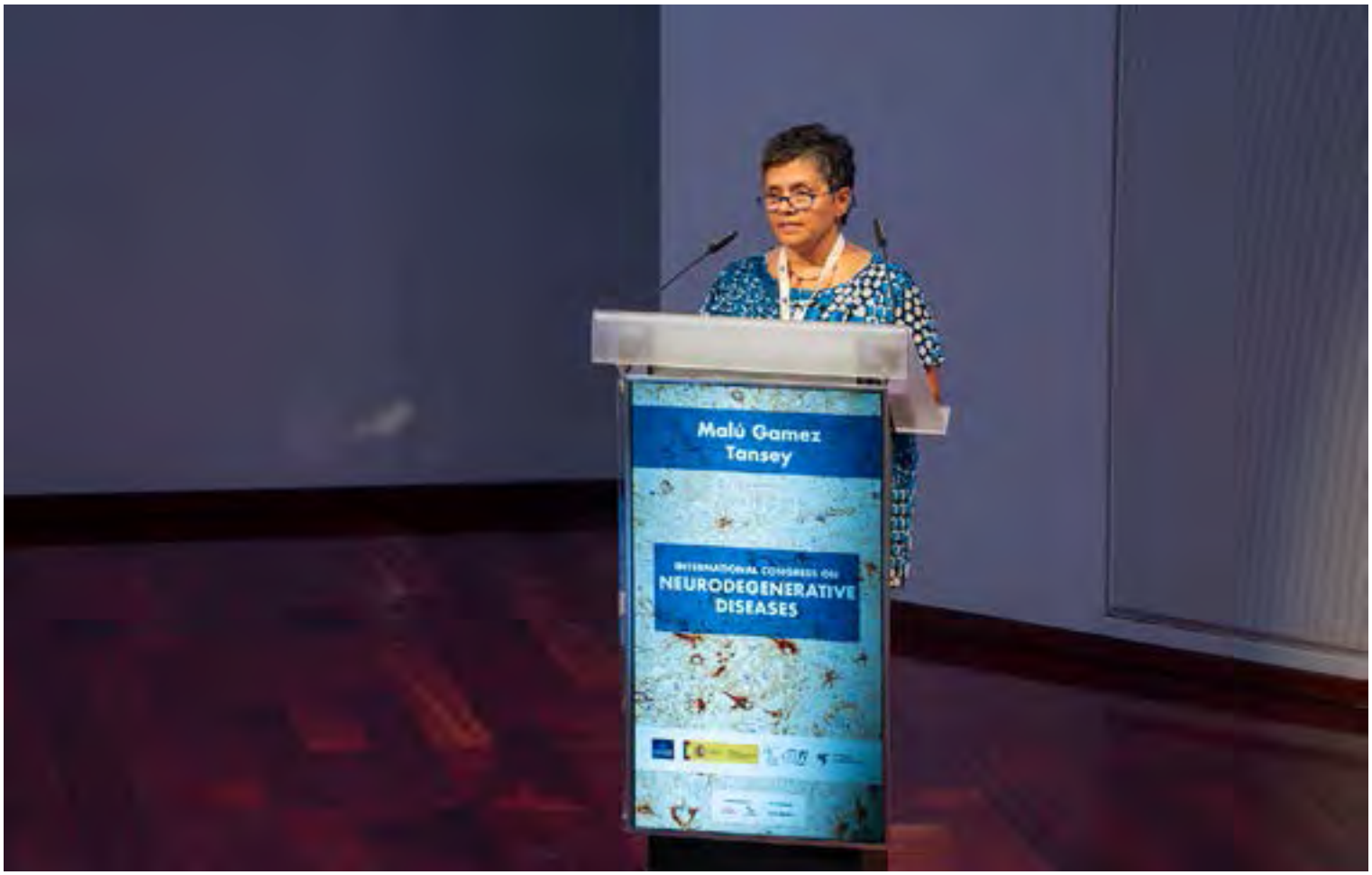


Malú Gámez Tansey
Center for Transaltional research in Neurodegenerative disease. University of Florida

Malú Gámez Tansey, Ph.D. is the Norman and Susan Fixel Chair in Neuroscience and Neurology and former Director of the Center for Translational Research in Neurodegenerative Disease at the University of Florida. Her lab focuses on the role of inflammation and immune system responses in brain health and neurodegenerative disease, with particular focus on central-peripheral neuroimmune crosstalk and the gut-brain axis, with the long-term goal of developing better therapies to prevent and/or delay these diseases.

Dr. Tansey obtained her B.S/M.S in Biological Sciences from Stanford University and her Ph.D. in Cell Regulation from UT Southwestern followed by post-doctoral work in neuroscience at Washington University. As head of Chemical Genetics at Xencor, she co-invented novel soluble TNF inhibitors that have now advanced to clinical trials in Alzheimer's disease. She returned to academia as an Assistant Professor of Physiology at UT Southwestern in 2002 and was recruited to Emory University School of Medicine as a tenured Associate Professor in 2009. After 10 years at Emory and rising to the rank of Full Professor where she earned several mentoring awards from students and faculty for her efforts in championing early-stage investigators, women and other under-represented groups in STEM, she was recruited to the Department of Neuroscience in the College of Medicine at the University of Florida, where she served on the executive committees for the McKnight Brain Institute and the Fixel Institute for Neurological Diseases. She will be moving to the Stark Neuroscience Research Institute at Indiana University in Indianapolis in January of 2025 as the first Director of Neuroimmunology Research and Executive Associate Director of Education at the Stark.

Personalized Interventions. Secondary prevention



Personalized Interventions. Secondary prevention

Tiago F. Outeiro
University Medical Center Gottingen

Tiago Outeiro graduated in Biochemistry at the University of Porto and was an Erasmus student at the University of Leeds in the UK. Tiago then did his PhD thesis at the Whitehead Institute for Biomedical research – MIT and worked as a Research Scientist at FoldRx Pharmaceuticals as a Research Scientist and Consultant.

Tiago served as vice-President, President, and chairman of PAPS during his stay in the US.

Tiago was a Postdoctoral Research Fellow in the Department of Neurology of the Massachusetts General Hospital – Harvard Medical School where he focused on the study of Neurodegenerative disorders such as Parkinson's and Alzheimer's disease.

Tiago is Full Professor and the Director of the Department of Experimental Neurodegeneration at the University Medical Center Gottingen, in Germany. Since November 2017, Tiago is also Professor of Neurodegeneration at Newcastle University, UK.

Personalized Interventions. Secondary prevention

Personalized Interventions. Secondary prevention

Catherine Mummery
University College London

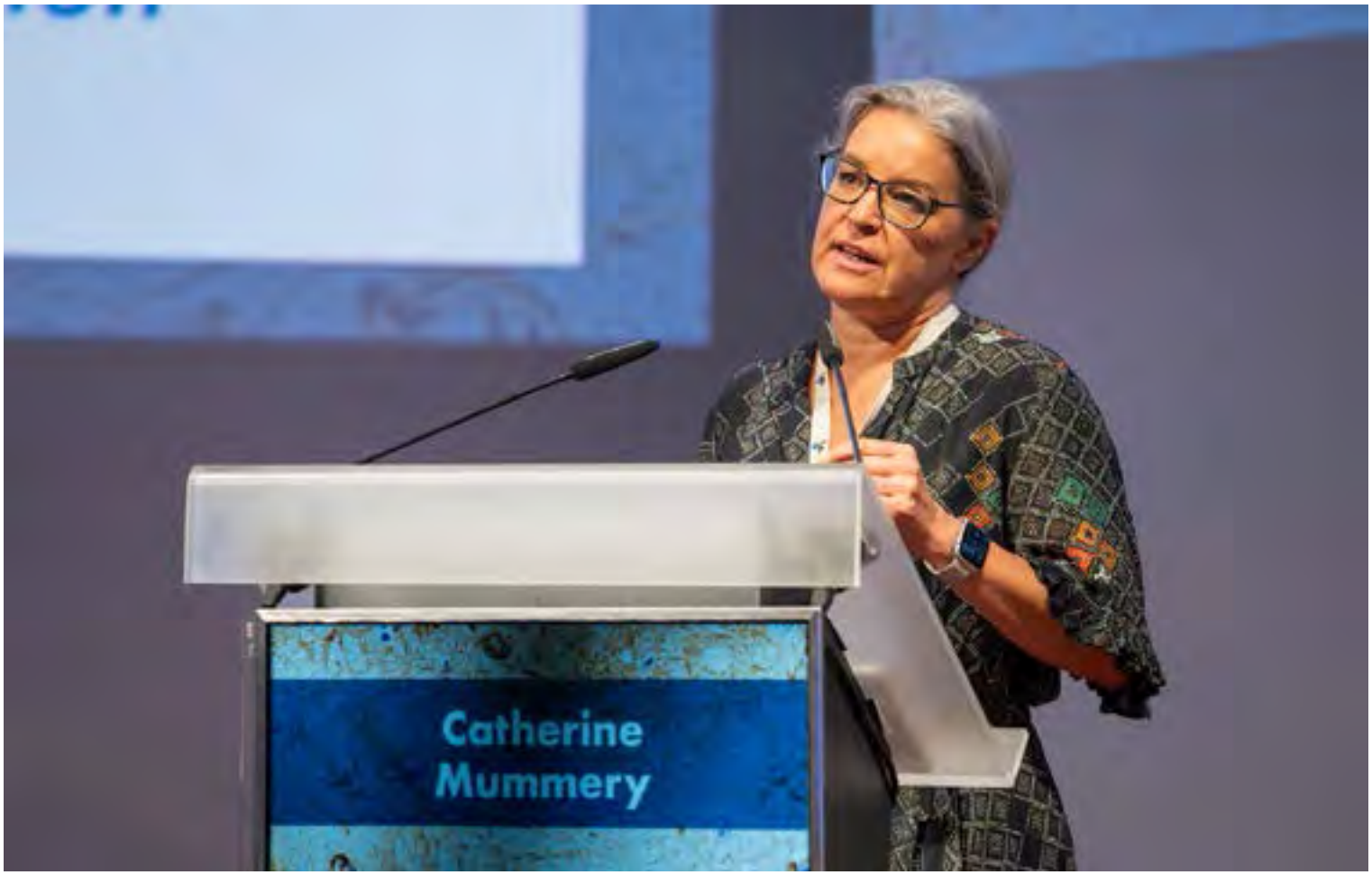
Professor Cath Mummery is a consultant neurologist at the National Hospital for Neurology and Neurosurgery. She is chair of the NIHR Dementia Translational Research Collaboration, building a national unified trials network for early phase clinical trials and working with the Mission to accelerate and enhance dementia translational research in novel treatments.

She is Head of Clinical Trials at the Dementia Research Centre at University College London, and Deputy Director for the Leonard Wolfson Experimental Neurology Centre. She has been chief investigator on over 20 early phase drug trials of potential disease modifying agents in sporadic Alzheimer's disease (AD), and genetic forms of AD and frontotemporal dementia.

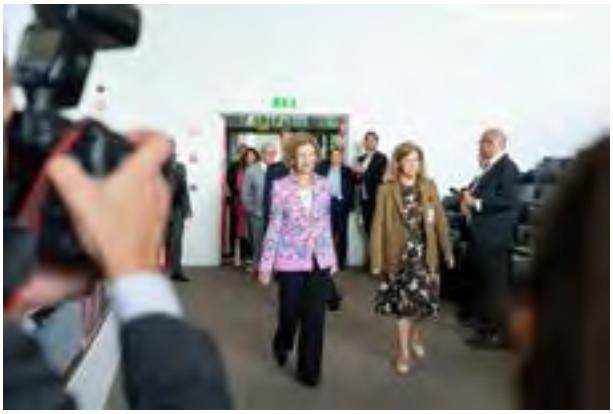
As clinical lead for the UCL Neurogenetic Therapies Programme, she leads a programme of innovative collaboration between industry and academia to accelerate progress in genetic therapies in dementia.

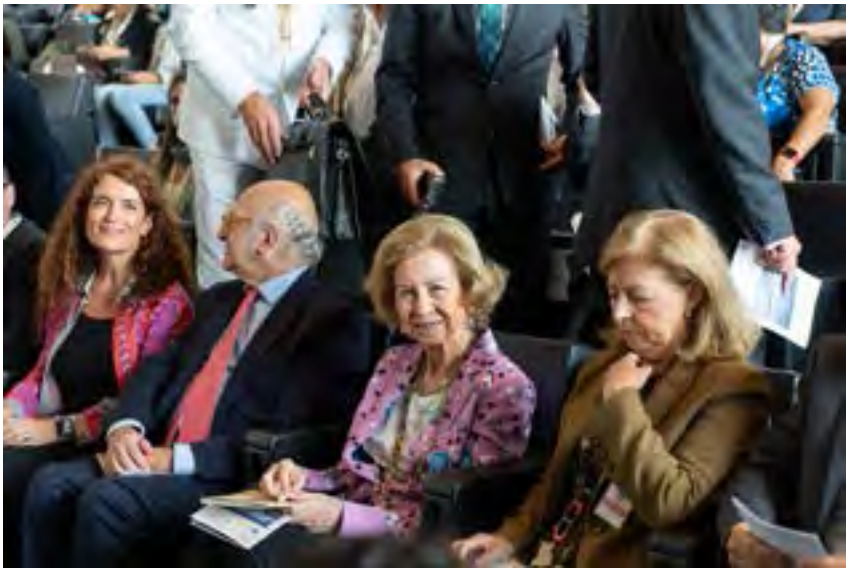
Her driving ambition is to ensure we not only have treatments that can alter the course of neurodegenerative diseases like Alzheimer's, but that we can deliver them promptly, safely and equitably.

Personalized Interventions. Secondary prevention



Personalized Interventions. Secondary prevention





Personalized Interventions. Secondary prevention

11.30 - 13.00

SCIENTIFIC SESSION VI: NON-PHARMACOLOGICAL INTERVENTIONS

Non-pharmacological interventions. Here, we will discuss non-pharmacological interventions and the available evidence of how these address specific pathological components

CHAIRPERSON:

JOAQUIM FERREIRA

CNS – Campus Neurológico. University of Lisbon. Portugal.

PABLO MARTÍNEZ LAGE

Fundación CITA Alzheimer. Donostia. Spain.

Successful adaptation of the FINGER model in Southern Europe: From the GOINZ-ZAINDU pilot study to the CITA GO-ON trial

GIACOMO KOCH

Foundation Santa Lucia, Rome. Italy.

Personalized Neuromodulation of the Default Mode Network for Treatment of Alzheimer's Disease: Foundations and Next Steps

ARIANNE GRAVESTEIJN

Amsterdam UMC, The Netherlands.

Beyond Medication: The Role of Exercise and other non-pharmacological interventions in Managing Anxiety and Depression in Neurodegenerative Disorders

13.00 - 15.00

LUNCH & POSTERS

Personalized Interventions. Secondary prevention

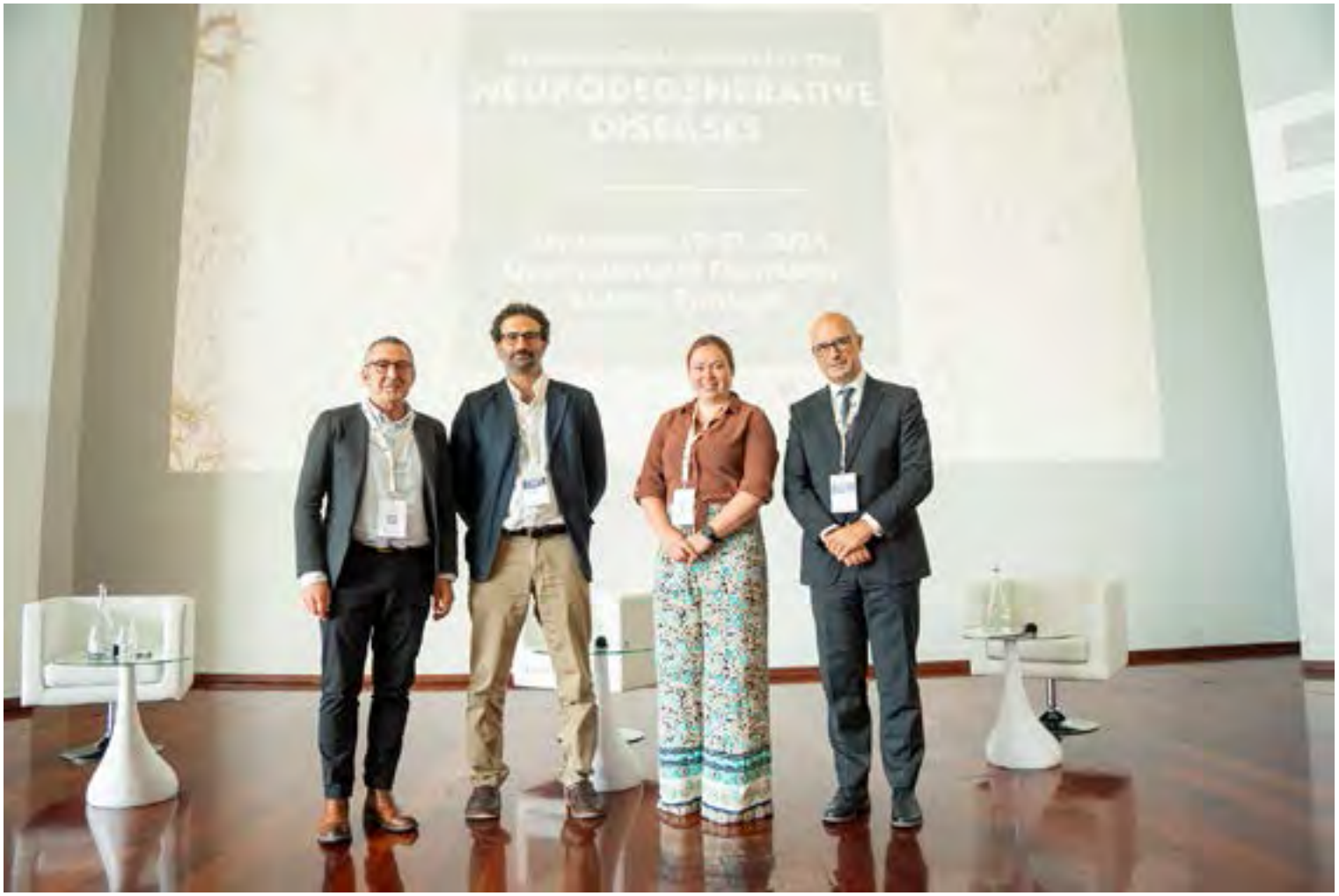
SCIENTIFIC SESSION VI: NON-PHARMACOLOGICAL INTERVENTIONS

Non-pharmacological interventions. Here, we will discuss non-pharmacological interventions and the available evidence of how these address specific pathological components

CHAIRPERSON:

Joaquim Ferreira

CNS – Campus Neurológico. University of Lisbon. Portugal.

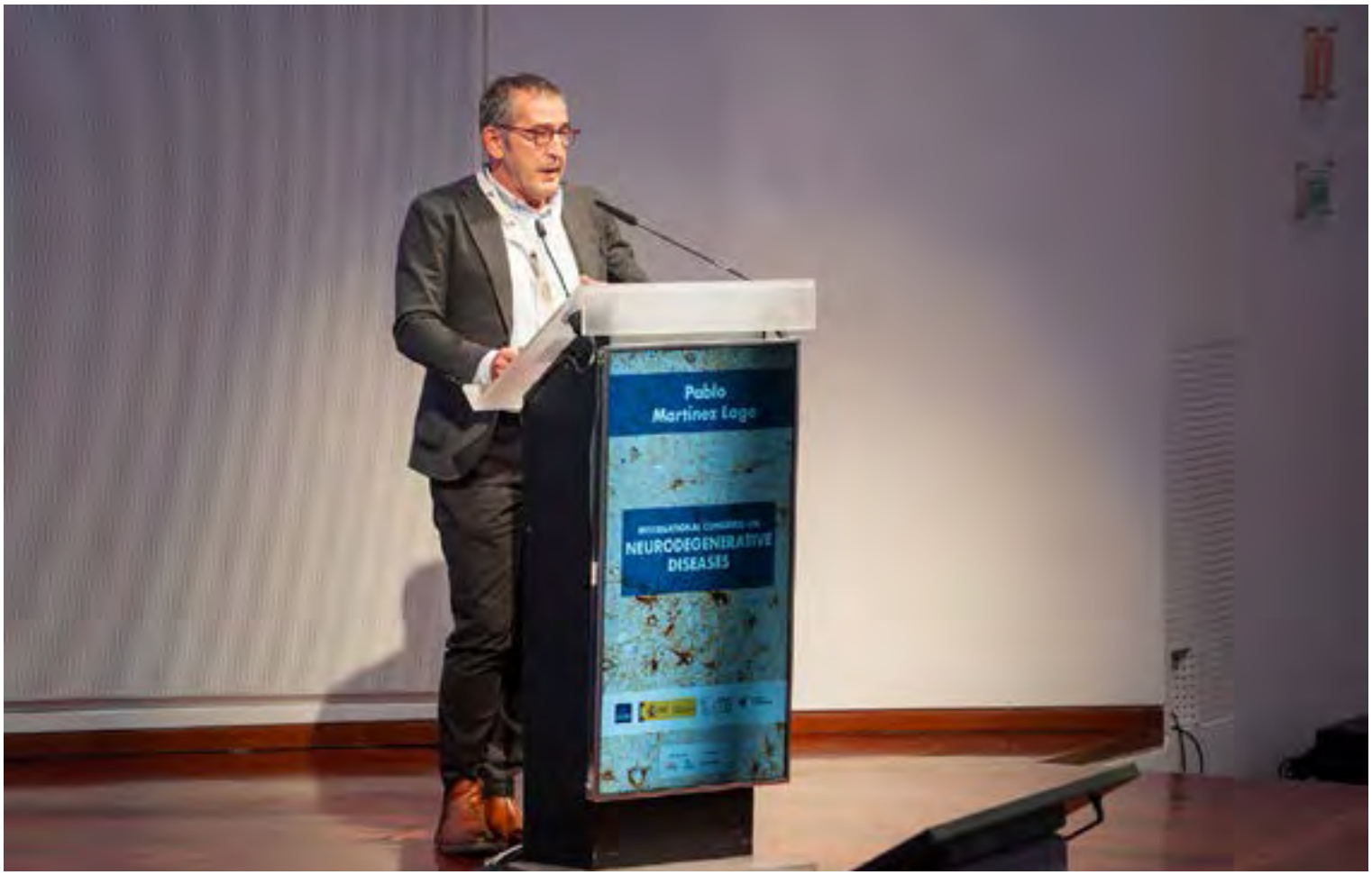


Personalized Interventions. Secondary prevention

Pablo Martínez-Lage[1]
Neurólogo. Director Científico
Fundación CITA-Alzheimer Fundazioa
Donostia-San Sebastián

Graduate in Medicine and Surgery at the University of Navarra and specialist via MIR in Neurology. Since then, I have worked in the field of care, research and teaching in the field of cognitive impairment in various institutions such as the Virgen del Camino Hospital in Pamplona, the Landazabal Psychogeriatric Center in Burlada, the Memory Disorders Unit of the University Clinic of Navarra, Fundación Alzheimer Center Educacional-Institut Català de Neurciències Aplicades in Barcelona and the Center for Research and Advanced Therapies of the CITA-Alzheimer Foundation in San Sebastián. I have specialized in caring for people with cognitive impairment and dementia and research in Alzheimer's disease, vascular cognitive impairment, and other dementias, with a special interest in early detection and diagnosis, treatment and prevention.

Personalized Interventions. Secondary prevention



Personalized Interventions. Secondary prevention

Giacomo Koch
University of Ferrara
Santa Lucia Foundation IRCCS Rome

Professor Giacomo Koch trained in London at the University College of London.

For more than ten years he has been involved in the acute treatment of stroke in the Stroke Unit of the Policlinico Tor Vergata in Rome.

He has directed the noninvasive brain stimulation laboratory at the Fondazione S. Lucia IRCCS in Rome since 2006. He has decades of experience in the field of clinical neurophysiology of the motor system and cognitive functions, with a translational approach to the study and treatment of cerebrovascular diseases, movement disorders and Alzheimer's disease.

He has held the position of Full Professor of Physiology at the University of Ferrara since 2020.

His main expertise is based on the application of non-invasive brain stimulation techniques such as Transcranial Magnetic Stimulation (TMS) and Transcranial Continuous Current Stimulation (TDCS). Used in conjunction with clinical neuroimaging methods such as functional magnetic resonance imaging (fMRI) and electroencephalography (EEG), they allow the identification of the neuro-anatomical basis of neurophysiological modulations of cortical activity induced by noninvasive brain stimulation.

In that field, Professor Giacomo Koch has won numerous international awards, appearing among the top ten top experts in the world by number of publications. He is the author of about 330 international publications with an H-index of 70, is on the list of Top Italian Scientists (TIS), and is a Distinguished Professor in Neurology and Physiology.

The translational approach of his research is evidenced by the design and conduct of clinical trials for which he has been Principal Investigator (PI) in the field of cerebrovascular pathology and neurodegenerative diseases such as Alzheimer's disease

Personalized Interventions. Secondary prevention

Arianne Gravesteijn
Department of Rehabilitation Medicine,
Amsterdam UMC, MS Centrum Amsterdam
Amsterdam Movement Sciences,
Amsterdam Neurosciences

PhD Candidate, Exercise PRO-MS Study and Post-doctoral Researcher, HersenFIT Study at the Department of Rehabilitation Medicine, Amsterdam UMC, MS Centrum Amsterdam. Research Institutes: Amsterdam Movement Sciences, Amsterdam Neurosciences

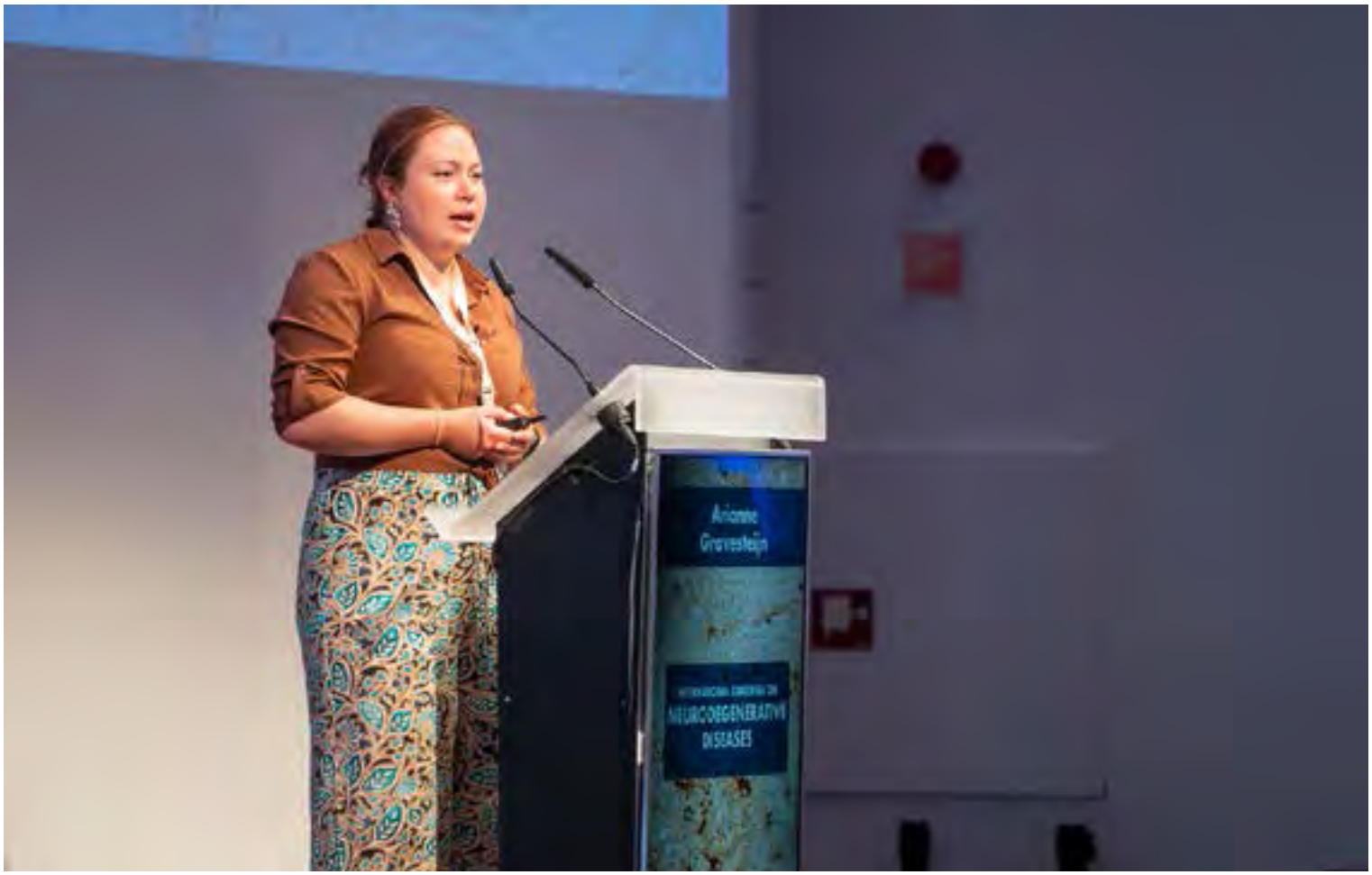
Education Background:

Bachelor's Degree: Physiotherapy

Master's Degree: Human Movement Sciences

Specialization/Focus of research: Exercise Physiology, Neurodegeneration, Multiple Sclerosis.

Personalized Interventions. Secondary prevention



Personalized Interventions. Secondary prevention

LUNCH & POSTERS

Personalized Interventions. Secondary prevention

15.00 - 16.30

SCIENTIFIC SESSION VII: DIGITAL THERAPIES IN ASSESSMENT AND INTERVENTIONS

CHAIRPERSON:

JOHN KRAKAUER
Champalimaud Foundation & Johns Hopkins University.

DANIELA PIMENTA DA SILVA
CNS, Lisboa. Portugal.
The role of exercise in prevention and treatment

ARKO GHOSH
Leiden University. The Netherlands.
Interpreting real world behavior captured on the smartphone

DINA KATABI
Massachusetts Institute of Technology, USA.
AI-Enabled Digital Biomarkers for Neurodegenerative Diseases (On-Line)

16.30 - 18.00

COFFEE BREAK & POSTERS

18.00 - 19.30

LIVE MUSIC & COCKTAIL

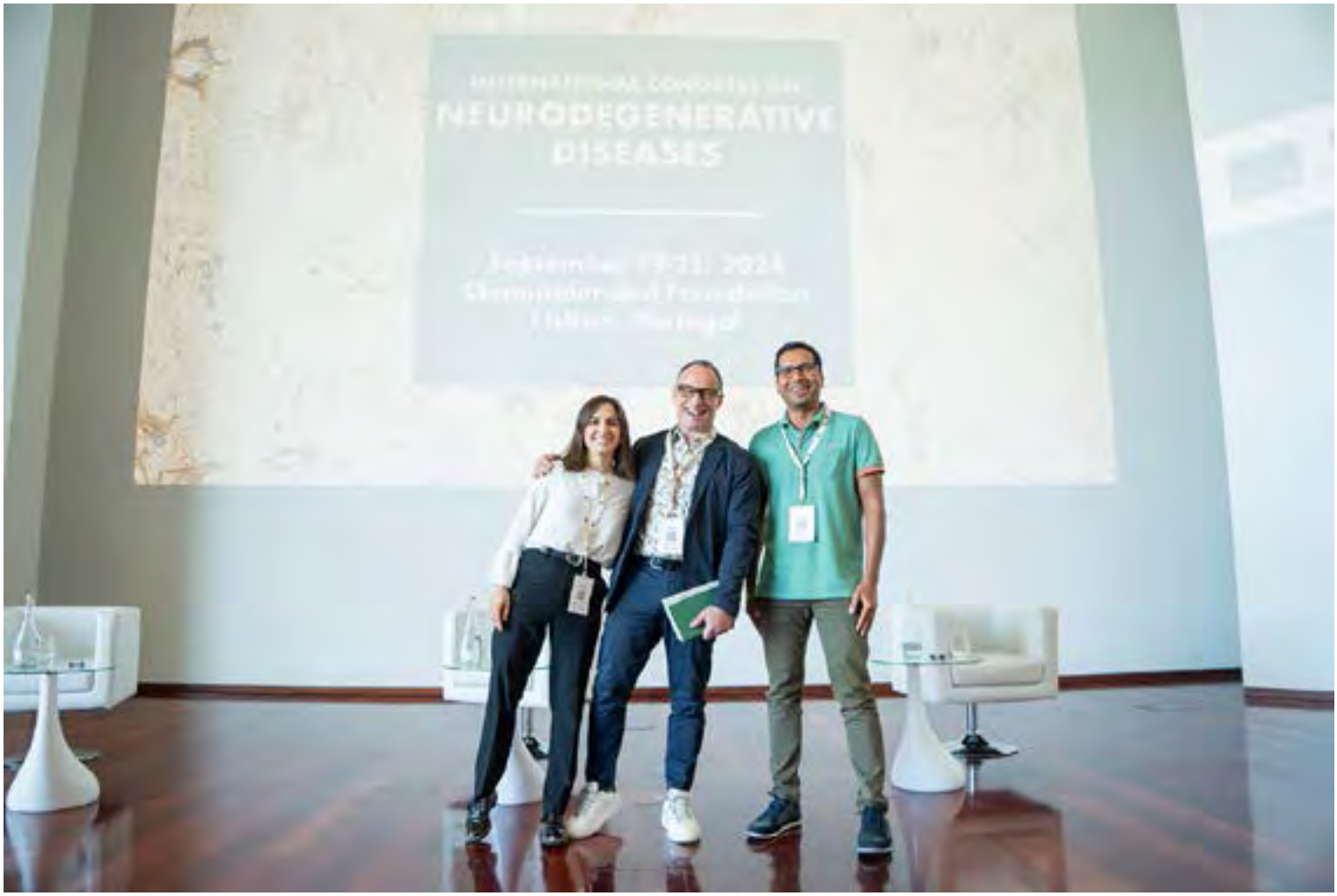
Personalized Interventions. Secondary prevention

SCIENTIFIC SESSION VII: DIGITAL THERAPIES IN ASSESSMENT AND INTERVENTIONS

CHAIRPERSON:

John Krakauer

Champalimaud Foundation & Johns Hopkins University.



Personalized Interventions. Secondary prevention

Daniela Pimenta Silva
CNS Portugal

Daniela Pimenta Silva graduated in Medicine at the University of Coimbra and completed her Neurology residency at Centro Hospitalar Universitário Lisboa Norte, Hospital Santa Maria. During residency, she developed a special interest in movement disorders and pursued a clinical fellowship at Hôpitaux Universitaires Pitié-Salpêtrière in Paris, France, where she had the opportunity to learn and practice with Professor Marie Vidailhet.

Currently, Daniela is a PhD student at the University of Lisbon. Her research is focused on the use of Virtual Reality in the non-pharmacological management of Parkinson's disease, under the supervision of Prof. Dr. Miguel Coelho and Prof. Dr. Joaquim Ferreira.

Personalized Interventions. Secondary prevention

Personalized Interventions. Secondary prevention

Arko Ghosh
Leiden University

Arko is at the Institute of Psychology at Leiden University, The Netherlands, and holds a B.S. in Neuroscience from Trinity College, USA, and a Ph.D. in Neuroscience from ETH Zurich, Switzerland. His research program is to build innovative science to explain complex real world behavioral structures. His focus on the real world has a growing imprint – from capturing how we age to discovering fundamental patterns like multi-day behavioral rhythms. He also plays a foundational role in launching spin-offs that focus on solving real-world problems using the science and technology developed in the laboratory.

BS, Trinity College (CT), USA

PhD, Swiss Federal Technological Institute (ETH Zurich), Switzerland

Post-doctoral researcher at University College London, University of Zurich and ETH Zurich

Spin-offs: QuantActions (co-founder), AXITE (scientific advisor)

Personalized Interventions. Secondary prevention

Dina Katabi
MIT Computer Science & Artificial Intelligence Lab

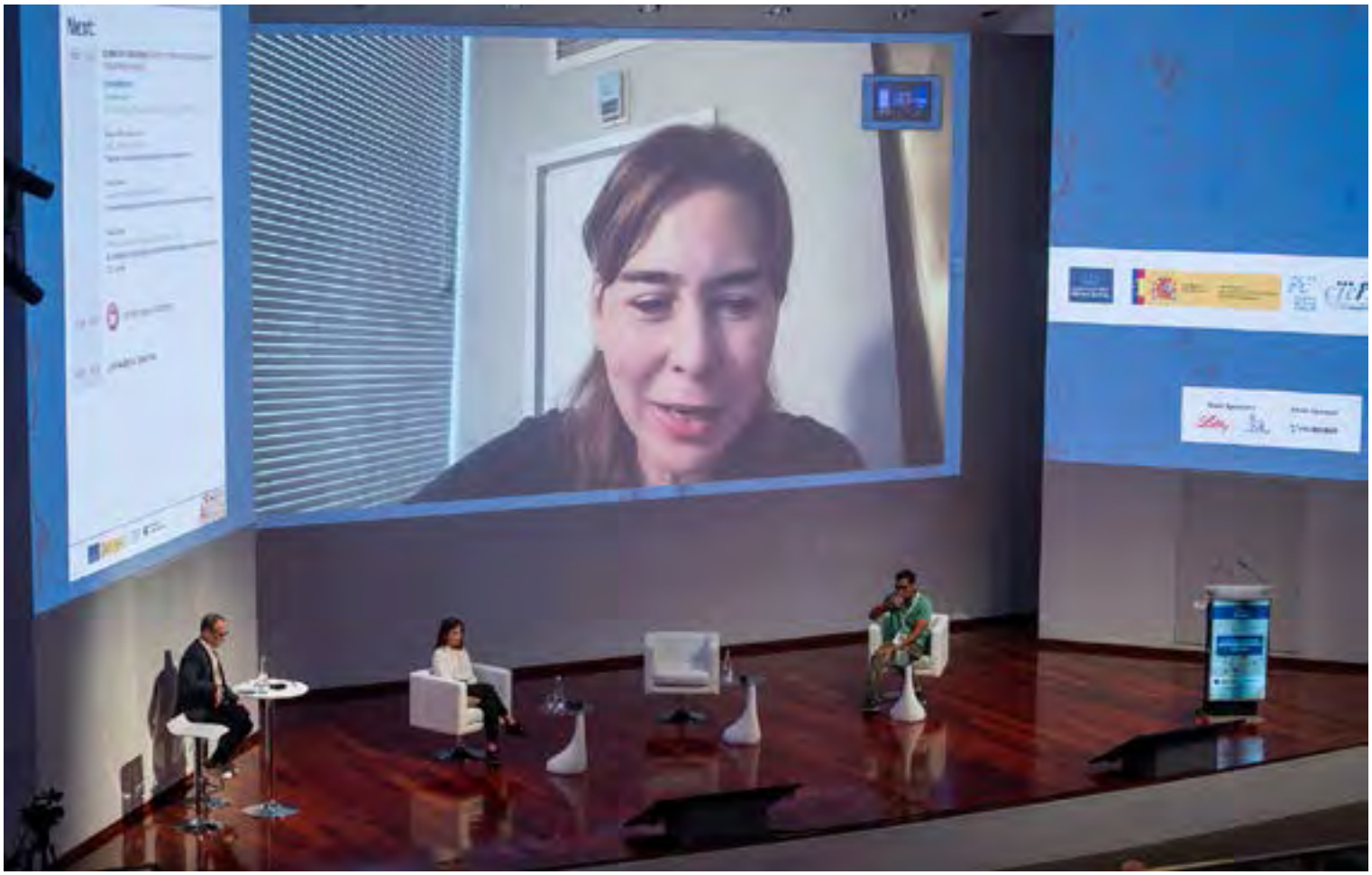
Academic biography

Katabi received a bachelor's degree in electrical engineering from the University of Damascus in 1995, then an M.S in Computer Science and a Ph.D. in Computer Systems Networking and Telecommunications from MIT in 1998 and 2003, respectively. In 2003, Katabi joined MIT, where she holds the title of Professor in the Department of Electrical Engineering and Computer Science. She is the co-director of the MIT Center for Wireless Networks and Mobile Computing and a principal investigator at MIT's Computer Science and Artificial Intelligence Laboratory. Katabi was selected as a MacArthur Fellow in 2013. She was elected to the National Academy of Engineering, American Academy of Arts and Sciences, and National Academy of Sciences in 2017, 2022, and 2023 respectively.

Research and career

Katabi's research focused on signals, machine learning and health. Her work started in networks (especially the congestion control challenge), where she found solutions for a better reliability of networks. Then, with her team, she used machine learning and signals to analyze the human body. Based on how RF signals bounce off our bodies, the researchers could measure human breathing, heart rates, emotion and sleep stages, without having the "patient" wear any sensor. Her most recent research combined medicine with AI, where she developed with her team a system capable of diagnosing Parkinson's Disease.

Personalized Interventions. Secondary prevention



Personalized Interventions. Secondary prevention

LIVE MUSIC & COCKTAIL


















Patient's perspective

10.00 - 10.20 **ARRIVAL AND SEATING OF ALL ATTENDEES**

10.30 - 13.30 **SESSION:** PEOPLE LIVING WITH AD AND PD – THE PATIENT AT THE CENTER OF CARE AND THEIR ENGAGEMENT IN RESEARCH

CHAIRPERSON:

MARCELO MENDONÇA
Champalimaud Foundation, Lisbon. Portugal.

10.30 - 11.30 Presentations:

JOSEFA DOMINGOS
Parkinson's Europe and Young Parkies Portugal

MARIA DO ROSÁRIO ZINCKE
Alzheimer Europe and Associação Alzheimer Portugal

MARILÓ ALMAGRO
Confederación Española de Alzheimer y otras demencias (CEAFA). Spain.

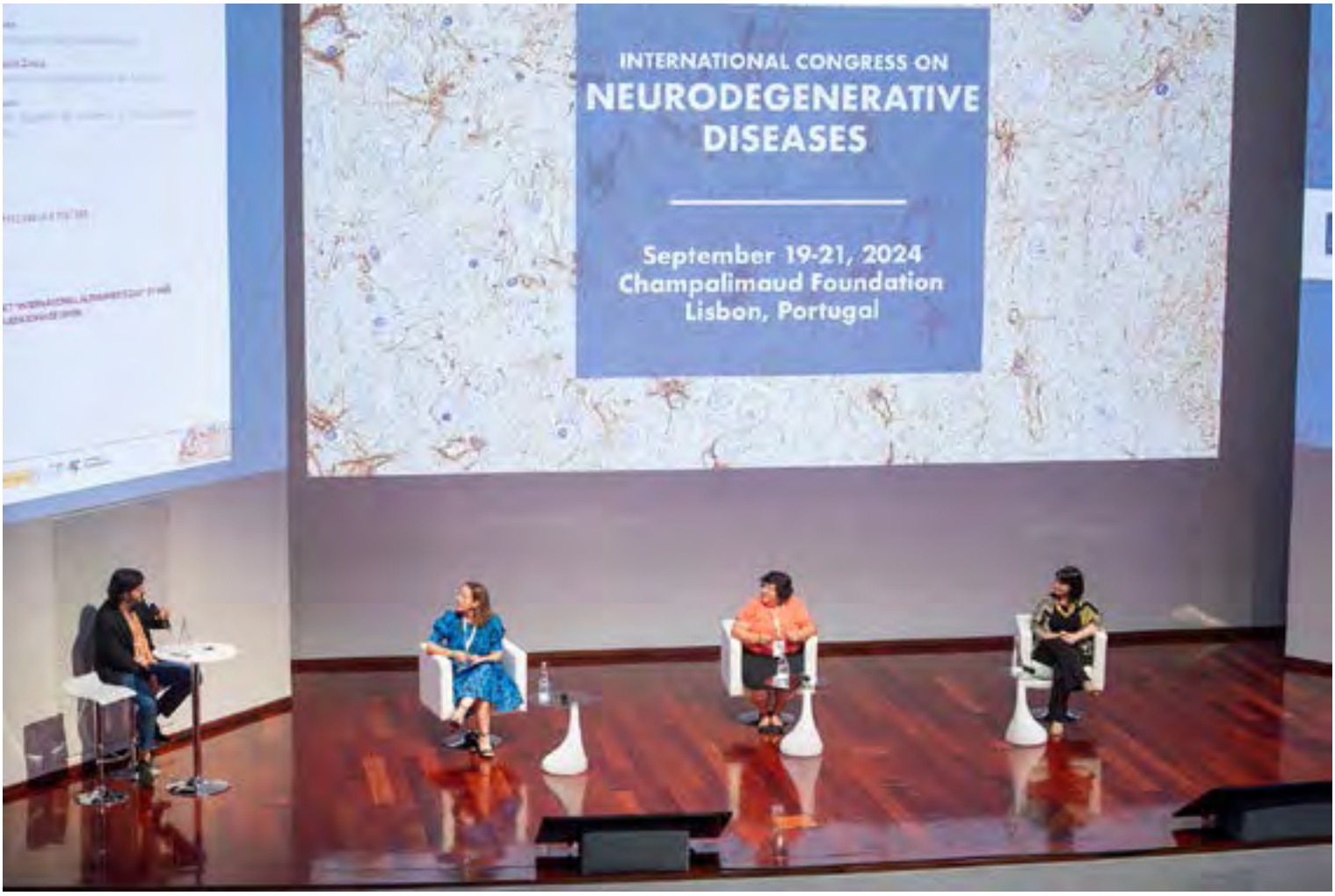
11.30 - 12.00 **COFFEE BREAK & POSTERS**

Patient's perspective

SESSION: PEOPLE LIVING WITH AD AND PD – THE PATIENT AT THE CENTER OF CARE AND THEIR ENGAGEMENT IN RESEARCH

CHAIRPERSON:

MARCELO MENDONÇA
Champalimaud Foundation, Lisbon. Portugal.



Patient's perspective

Josefa Domingos
President of Parkinson's Europe

Dr. Josefa Domingos is the president of Parkinson's Europe, a physiotherapist specializing in Parkinson's disease (PD) with two decades of experience working exclusively with people with PD. Since 2005, she has played a pivotal role in creating and developing specialized health services for people with Parkinson's in Portugal, Sweden, and the US. Currently, Dr. Domingos serves as the National Health Coordinator at the Portuguese Parkinson Patient Association (APDPk) and co-founder of Young Parkies Portugal (YPP). She also contributes to the Davis Phinney Foundation's scientific committee and is a member of the International Movement Disorder Society's Wellness Group. Dr. Domingos is an educator, clinician, and researcher; in 2023, she completed her PhD on the practicalities of implementing community-based exercise in Parkinson's at Radboud University, Nijmegen, The Netherlands, under the supervision of Prof. Dr. Bas Bloem and Prof. Dr. Joaquim Ferreira. Her dedication to advancing Parkinson's research, advocacy, and support extends both nationally and internationally.

Patient's perspective

Patient's perspective

Maria do Rosário Zinke
Alzheimer Europe and Associação Alzheimer Portugal

Maria do Rosário has been practicing law in Portugal since 1987, with a focus on family law and the legal rights of people with diminished capacity. She is also a trainer on these same issues.

She is the Chairperson of Alzheimer Portugal.

On behalf of Alzheimer Portugal she is the General Meeting Chairperson of "Plataforma Saúde em Diálogo", an umbrela organisation of 70 organisations, mostly of patients organisations of people living with chronic diseases.

She is a former member of CEIC – Narional Ethics Committee for Clinical Research.

Maria do Rosário has been a member of the Alzheimer Europe Board since 2008, was the Honorary Treasurer from 2010-2020, after that started to serve as Chairperson.

Patient's perspective

Patient's perspective

Mª Dolores Almagro Cabrera
Presidenta de CEAFA

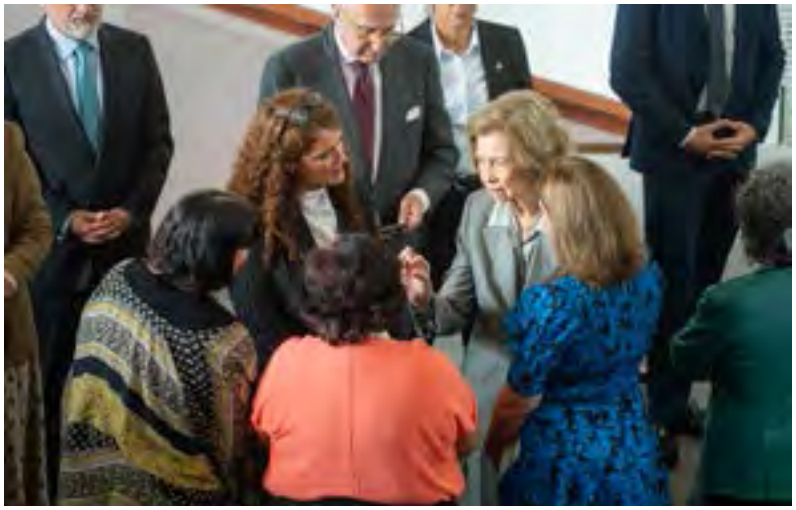
Mariló empezó su labor como voluntaria en el mundo Alzheimer cuando su madre fue diagnosticada de esta enfermedad en Motril. Ante la falta de recursos en esa ciudad, en el año 2010 fundó la Asociación Afacontigo Alzheimer de Motril. Desde entonces entró a formar parte en la Federación Granadina de Alzheimer, de la que es presidenta desde 2014. Actualmente también es Tesorera de la Confederación Andaluza de Alzheimer y Otras Demencias.

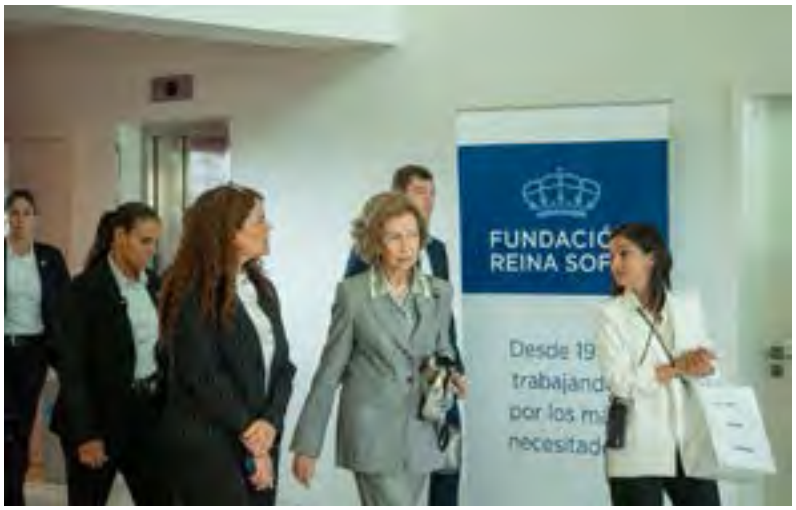
En el año 2016 entró a formar parte de la Junta de Gobierno de CEAFA como vocal, y ya en 2019 ocupó el cargo de vicepresidenta. Desde 2022 ocupa la presidencia de CEAFA.

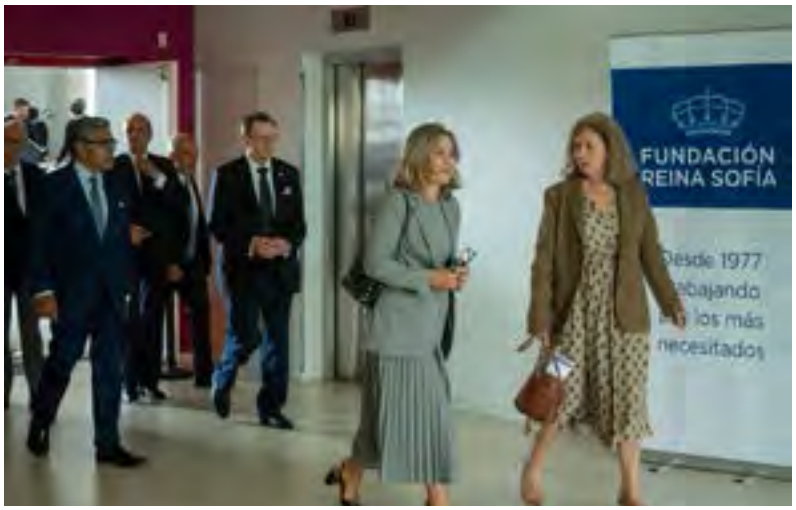
Durante su mandato, la Junta de Gobierno tiene como meta continuar trabajando para poner el Alzheimer en la agenda política, buscando el necesario compromiso social para llevar a cabo acciones que ayuden a cambiar el estigma asociado a la enfermedad.

Patient's perspective

Patient's perspective







Patient's perspective

12.00 - 12.30

OFFICIAL ACT "INTERNATIONAL ALZHEIMER'S DAY" BY HER MAJESTY QUEEN SOFÍA OF SPAIN

12.30 - 13.30

ROUND TABLE

Patient associations:

Josefa Domingo
Parkinson's Europe and Young Parkies Portugal

Maria do Rosário Zincke
Alzheimer Europe and Associação Alzheimer Portugal

Mariló Almagro
Confederación Española de Alzheimer y otras demencias (CEAFA). Spain.

Kina García
Panel de expertos de personas con Alzheimer (PEPA).

Alicia Campos
Federación Española Parkinson.

Neurologists/Researchers:

Isabel Santana
Faculdade de Medicina, Universidade de Coimbra. Portugal.

Alberto Lleó
Unidad de Memoria. Hospital de Sant Pau, Barcelona. Spain.

Pascual Sánchez-Juan
Fundación CIEN, Madrid.Spain.

Joaquim Ferreira
CNS – Campus Neurológico. University of Lisbon. Portugal.

Geneticists:

Agustín Ruiz
Fundació ACE, Barcelona. Spain.

13.30

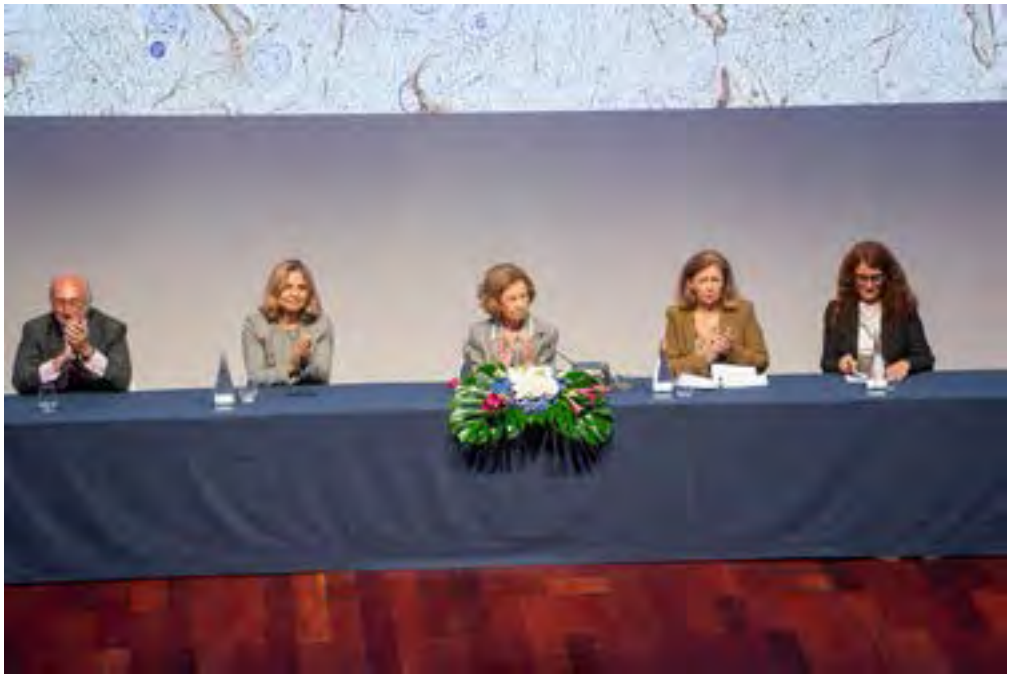
END OF MEETING

Patient's perspective

**OFFICIAL ACT "INTERNATIONAL ALZHEIMER'S DAY"
BY HER MAJESTY QUEEN SOFÍA OF SPAIN**

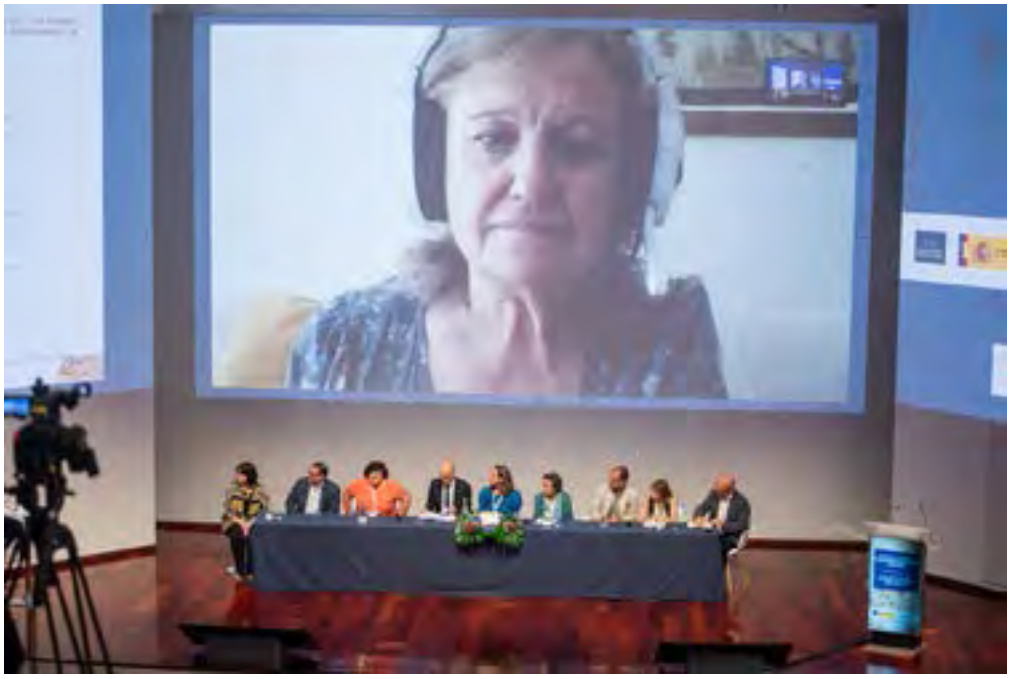
Patient's perspective

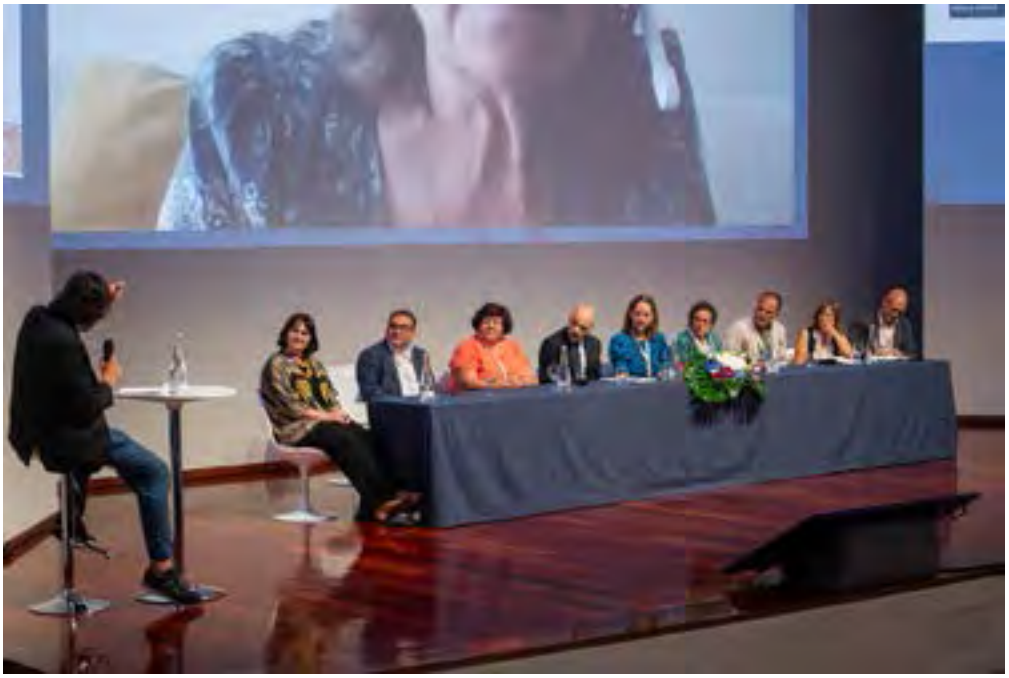
**OFFICIAL ACT "INTERNATIONAL ALZHEIMER'S DAY"
BY HER MAJESTY QUEEN SOFÍA OF SPAIN**



Patient's perspective

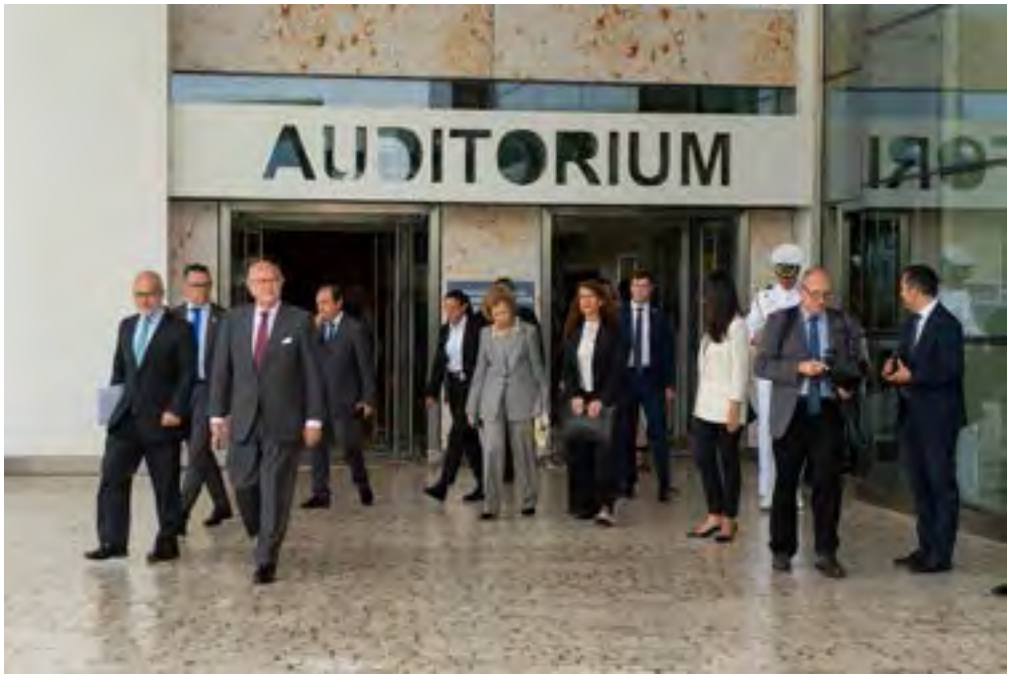
**OFFICIAL ACT "INTERNATIONAL ALZHEIMER'S DAY"
BY HER MAJESTY QUEEN SOFÍA OF SPAIN**

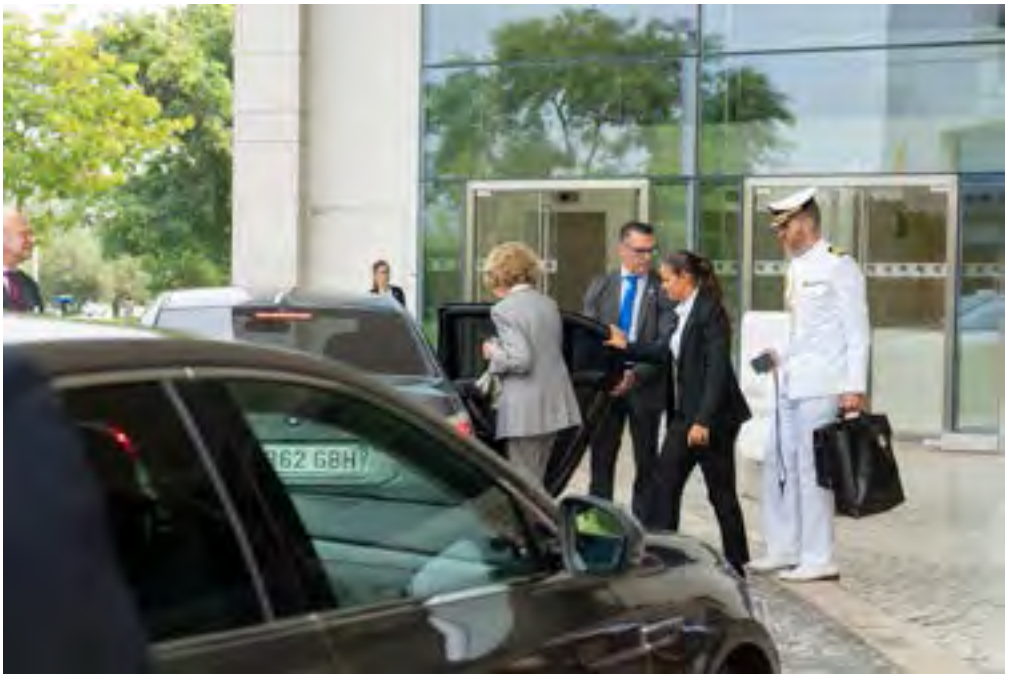




Patient's perspective

END OF MEETING





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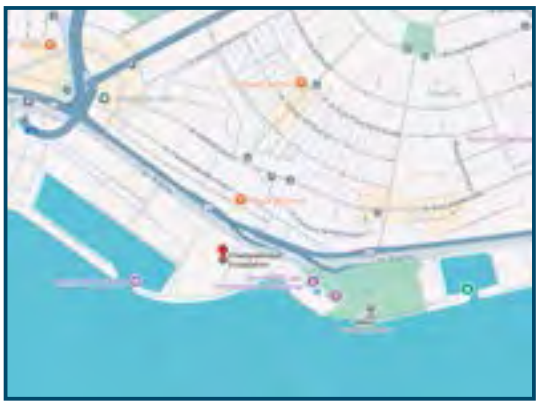
Almudena Flores
Beatriz Salado
Aina Frontera Sáchez
Ana Mulás Sierra
Laura Saiz Aúz
C/ Valderrebollo, 5 - 28031 Madrid.
congreso@fundacioncien.es
Phone: (+34) 91 385 22 00

CONGRESS SECRETARIAT:

OASIS EVENTS & TRAVEL SOLUTIONS
C/ Sor Ángela de la Cruz , 8 - 1º A - 28020 Madrid.
Phone: (+34) 91 555 11 19
www.viajesoasis.com / jsanchez@viajesoasis.com

VENUE:



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Fundação Champalimaud

INTERNATIONAL CONGRESS ON NEURODEGENERATIVE DISEASES

September 19-21, 2024

Champalimaud Foundation
Lisbon, Portugal

Image: Dancing Astrocytes

ABSTRACTS BOOK 2024

INDEX

INDEX

POSTER N.º 1

Introduced by: López Torres, Isabel

Title:

SCAP-AD: RESEARCH PROJECT FOR THE EARLY DETECTION OF ALZHEIMER'S DISEASE

Principal Investigator: Isabel López-Torres

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Abstract:
"Objectives Alzheimer's disease (AD) is the most common neurodegenerative disease (60-80% of dementias) that seriously impacts the lives of many patients and their families. There are no curative treatments for AD, but in recent years new drugs are being developed that could modify its course. To apply these new therapies, it is necessary to make an early and accurate diagnosis of the disease. The SCAP-AD project aims to validate precision medicine tools to identify preclinical Alzheimer's disease. Material and Methods The SCAP-AD is a multicenter project, coordinated by CIEN, involving 13 Spanish research centers. Two large cohorts of subjects over 60 years of age, without a diagnosis of dementia will be recruited: a digital cohort (n=50,000) where the use of digital biomarkers through a web platform will be explored, and a clinical validation cohort (n=1,000) in which clinical, neuroimaging, plasma and cerebrospinal fluid markers studies will be performed. Results The main goal of the project is to improve the prevention and diagnosis of AD by integrating precision medicine tools to create strategies to identify the disease in its early preclinical and clinical stages. Conclusions This project is funded by the Instituto de Salud Carlos III (ISCIII) under the European NextGenEu funds that finance the actions of the Recovery and Resilience Mechanism and has the approval of its Research Ethics Committee. Keywords: Alzheimer's disease, precision medicine, biomarkers, early detection.

POSTER N.º 2

Introduced by: Uceda-Heras, Alicia

Title:

GFAP STAINING ALONG THE MEDIAL TEMPORAL LOBE IN HUMAN POSTMORTEM BRAIN TISSUE OF PATIENTS FROM THE VARS DEMENTIA COHORT

Principal Investigator: Alicia Uceda-Heras

Authors: Alicia Uceda-Heras, Iván Burgueño-García, Laura Saiz-Aúz, Paloma Ruiz-Valderrey, María José López-Martínez, Alberto Rábano

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Abstract:
“GFAP labels activated astrocytes and has been proposed as biomarker of Alzheimer’s disease (AD). GFAP was recently analyzed with findings that demonstrated an association between serum GFAP levels and post-mortem tau pathology. Now, we have studied the histological detection of GFAP in association with neuropathological variables. We analyzed 154 donated brains from the Vallecas Alzheimer’s Reina Sofía (VARS) cohort. We developed immunohistochemistry assays for GFAP in entorhinal cortex (EC) and amygdala (A) and measured the area stained with GFAP antibody through Cell-Profiler program. We observed that the extension of GFAP marker is higher in the superficial layers of EC as compared to the deep layers, therefore, we continued the analysis restricted to superficial layers of EC and the basolateral nuclei of A. We found a correlation ($r=0.173$; $P<0.05$) between GFAP in EC and Braak tau stages, and a correlation ($r=0.189$; $P<0.05$) of GFAP in A with LPC classification. Regarding EC, we observed a trend of increasing GFAP staining with higher stages of Braak tau, NIA B, Thal stage, NIA A, Nia C, HS (stages 0-4) and TDP-43 stage (Josephs et al). Finally, with respect to copathologies, we noted that in both structures, A ($P<0.05$) and EC ($P<0.01$), GFAP staining is higher in presence of 3-4 copathologies with middle/high burden of the pathology as compared with <2 copathologies. These findings altogether support the role of GFAP as AD biomarker, and as a possible marker of copathologies. The prospects for the study are to compare plasma levels of GFAP with histological data.

POSTER N.º 3

Introduced by: Vicario Abejón, Carlos

Title:

HUMAN APOE POLYMORPHISM IS INVOLVED IN THE MORPHOLOGICAL AND FUNCTIONAL CHANGES OF IPSC-DERIVED ASTROCYTES FROM ALZHEIMER'S PATIENTS DURING INFLAMMATION

Principal Investigator: Rebeca Vecino

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Abstract:
"Alzheimer´s disease (AD) is the leading cause of dementia in the aging population, with the ε4 allele of apolipoprotein E (APOE) being the strongest genetic risk factor. Astrocytes mediate key processes during AD progression, such as the clearance of amyloid-beta aggregates and the inflammatory response. However, the impact of different APOE alleles during astrocyte development, maturation, and function remains to be fully understood. Here, we obtained induced pluripotent stem cells (iPSCs) from fibroblasts of AD patients carrying ε3 and ε4 alleles (in homozygosis) and from healthy individuals. We also used gene-edited iPSC lines homozygous for the main APOE variants and an APOE knock-out line. Human astrocytes were generated by establishing a differentiation protocol by adding small molecules and growth factors. Then, the expression of typical markers and APOE was analyzed to confirm its astrocytic phenotype. In addition, astrocytes exhibited calcium wave production and glutamate uptake capacity. They also responded to an inflammatory stimulus or the presence of Aβ by increasing the expression levels and release of proinflammatory cytokines (such as IL-6) and changing their morphology. Our results show that APOE polymorphism affects the basal state of astrocytes and their capacity to react to both stimuli by acquiring different morphologies. Furthermore, the ε4 allele could alter Aβ uptake/degradation capacity by astrocytes and its distribution within the cell. Our findings highlight the relevance of APOE polymorphism in the morphological and functional profile of astrocytes and their potential correlation with the risk of developing AD."

POSTER N.º 4

Introduced by: Trujillo Estrada, Laura

Title:

MITOCHONDRIA ULTRASTRUCTURAL PATHOLOGY OF REACTIVE ASTROCYTES IN ALZHEIMER'S DISEASE

Principal Investigator: Elba Lopez-Oliva

Authors: Lopez-Oliva E1, Trujillo-Estrada L1, Fernandez-Valenzuela JJ1, Sanchez-Mejías E1, Mejias-Ortega M1, Vizuete M2, Vitorica J2 and Gutierrez A1

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Abstract:
"In Alzheimer's disease (AD) astrocytes become reactive participating in the inflammatory response and playing a key role in disease progression. However, there is a limited understanding of the changes in reactive astrocytes that might lead to a dysfunctional state contributing neuronal disturbances in AD. Astrocytes are the prevalent glial cells and have many functions aimed at maintaining brain homeostasis including regulation of brain energy metabolism and maintenance of the blood-brain barrier. AD is characterized by brain hypometabolism and mitochondrial dysfunction, and although most studies have focused on neurons little is known about the dysfunction of astrocytic mitochondria in this disorder. Here, we have performed an ultrastructural analysis of reactive astrocytes, using transmission electron microscopy combined with immunogold labeling and image analysis, in the hippocampus of amyloidogenic (APP/PS1) and tauopathy (P301S) transgenic mice (4, 6 and 12 months). Our results show remarkable morphological alterations in the reactive astrocytes mitochondria that include double membrane rupture, cristae loss and loss of their circularity. Since mitochondrial morphology is directly related to mitochondrial fusion/fission processes, the ultrastructural changes observed in astrocyte mitochondria suggest dynamic abnormalities in these organelles that may lead to deficits in astroglial function compromising their capability to maintain brain homeostasis. A better understanding of cell type-specific mitochondrial dysfunction might hold great potential for the exploration of novel molecular targets for future disease modifying therapies. Supported by PI21-0915(AG),PI21-00914(JV) from Instituto de Salud Carlos III co-financied by FEDER funds from European Union; Universidad de Málaga PPIT.UMA.B1-2021_32(LTE); Junta de Andalucia and CIBERNED (AG and JV).

POSTER N.º 5

Introduced by: Ricciardi Serra, Mario Emiliano

Title:

DETECTION OF CEREBRAL AMYLOID ANGIOPATHY (CAA) IN ALZHEIMER'S DISEASE (AD) USING BLOOD BIOMARKERS.

Principal Investigator: Mario Ricciardi

Authors: Mario Ricciardi | Elizabeth Valeriano-Lorenzo| María Ascensión Zea-Sevilla | Meritxell Valenti | Belén Frades | Alicia Ruiz-Gonzalez | Ana Belén Pastor | Francisco LópezGonzalez | Paloma Ruiz | Laura Saiz | Iván Burgueño-Garcií | María José López-Martinez | Alberto Rábano | Teodoro Del Ser | Pascual Sánchez-Juan

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Abstract:
"Background: The Amyloid-Related Imaging Abnormalities (ARIA) produced by anti-amyloid drugs and the clinical repercussions of the disease raise the need for earlier biomarkers than MRI in CAA. This research aims to evaluate in patients with pathologically confirmed AD the correlation between CAA severity, serum biomarker levels and APOE genotypes. Methods: Cases with a pathological diagnosis of AD according to NIA-AA criteria were selected. In each case, CAA was assessed according to the criteria of Vonsattel et al. (grade 0 to 3), APOE was genotyped, and serum levels of Ab40, Ab42, Tau-total, and p-Tau181 were determined using SIMOA. A descriptive, correlation (Spearman coefficient) and comparison (Mann-Whitney U test) analysis of the data were performed. Using a Kruskal-Wallis analysis, biomarker levels and APOE genotypes were compared among the 3 groups of CAA severity (grade 0-1, grade 2, and grade 3). Results: A total of 104 cases were included: 10 grade 0, 36 grade 1, 46 grade 2, and 12 grade 3 CAA. 97% had a high Braak stage (5 or 6). A higher grade of CAA correlated with lower levels of Ab40 (rs= -0.225, p= 0.02) and p-Tau181 (rs= -0.251, p= 0.01) and was associated with a higher frequency of APOEε4 (U= 865.5, z-score 3.20281, p= 0.00069). Ab40 levels showed an inverse gradient to the degree of CAA severity in the 3 groups (medians: 235 pg/mL, 212 pg/mL and 190 pg/mL), with a significant difference between groups (H= 6.407, p= 0.04). Conclusions: The severity of CAA is associated with decreasing serum levels of Ab40 and increased presence of APOEε4."

POSTER N.º 6

Introduced by: Oliveira, Cátia

Title:

ENCEPHALOPATHY WITH REVERSIBLE MULTIFOCAL CEREBRAL EDEMA: AN ATYPICAL PRESENTATION OF A NEURODEGENERATIVE DISEASE

Principal Investigator: Cátia Oliveira

Authors: Oliveira C. 1, Castro R. 1, Pires A. 1, Fontão L.1

Filiation: 1 Neurology Department, Unidade Local de Saúde Entre Douro e Vouga

Abstract:
“Introduction: Cerebral amyloid angiopathy is an increasingly reported disorder in older people, although its inflammatory subtype (iCAA) is much rarer. It can present in several ways and its diagnosis may be challenging. Case Report: A 64-year-old woman with untreated hypertension and a history of treated low-grade urothelial carcinoma presented with progressive cognitive dysfunction, including language, memory, and visuospatial impairments, over the course of four weeks. No history of seizures or psychiatric symptoms was present. Examination revealed a blood pressure of 170/73 mmHg, attention deficits, and mixed aphasia. Brain imaging showed extensive bilateral cortico-subcortical edema, particularly in the frontal, temporoparietal, and occipital regions, brainstem, left hippocampus, and caudate nuclei, without contrast enhancement or vascular abnormalities. After aggressive blood pressure control and a course of corticosteroids (Methylprednisolone 1g for five days), the patient showed clinical and imaging improvement. Initial CSF analysis revealed hyperproteinorrachia, but all tests for neoplasms, systemic autoimmune disorders, and infections were negative. One year later, the patient continues to have slowly progressive memory and visuospatial dysfunction. Follow-up MRI showed complete resolution of the edema but the appearance of cortical microhemorrhages. The CSF analysis indicated low levels of beta-amyloid (1-42) and elevated phosphorylated tau. Conclusions: This case presents a striking clinical and imaging example of a reversible multifocal vasogenic brain edema. Initially, the differential diagnosis seemed to be primarily between atypical reversible posterior encephalopathy syndrome or iCAA, after exclusion of other neoplastic, autoimmune, and infectious causes. During follow-up, iCAA appears as the most likely diagnosis highlighting the importance of clinical suspicion of this condition.”

POSTER N.º 7

Introduced by: Sanchez-Mejías, Elisabeth

Title:

UNRAVELING THE PLAQUE-ASSOCIATED MYELOID CELL LANDSCAPE IN THE HUMAN ALZHEIMER'S BRAIN

Principal Investigator: Elisabeth Sanchez-Mejias

Authors: Elisabeth Sanchez-Mejias1,3, Marina Mejias-Ortega1,3, Clara Muñoz-Castro2,3, Marisa Vizuete2,3, Javier Vitorica2,3 and Antonia Gutierrez1,3

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Abstract:
Microglia are the resident innate immune myeloid cells of the brain and play a critical role in the pathological process of Alzheimer´s disease (AD) since genetic risk variants for late-onset AD are expressed exclusively or highly in these glial cells. Though the diversity of functional states of microglial cells in AD continuum is still unknown, loss of microglial neuroprotective and phagocytic function indicates the critical involvement of malfunctioning microglia in driving pathological progression and neurodegeneration. Moreover, little is known about the diversity of myeloid cells across different brain regions along the AD continuum. In this work, we have analyzed the responsive myeloid phenotype to amyloid pathology in two AD vulnerable brain regions, frontal cortex and hippocampus. For this purpose, immunolabeling for a wide range of markers combined with image analysis approaches have been carried out in postmortem samples from AD patients with dementia (Braak V-VI) and age-matched asymptomatic cases (Braak II). While the frontal cortex showed strong microglial activation around plaques, the hippocampus of the same individuals showed an exhausted microglial response including degenerative/senescent features. Regional differences were also found in the microglial phagocytic capacity. By counting the number of different microglial subsets, according to the combination of markers expressed, we found that the microglial composition of plaques was highly heterogeneous. Interestingly, even though some Braak II individuals presented high amyloid pathology, only AD patients exhibited infiltration of CD163 monocyte-derived cells that invaded plaque near blood vessels. These results reveal the co-existence of distinct myeloid populations associated with amyloid plaques during disease progression and open the opportunity to design targeted therapies, not only to microglia, but also to peripheral immune cell population to modulate amyloid pathology. Supported by ISCiii grants PI21/00915 (to AG) and PI21/000914 (to JV) co-financed by FEDER funds from European Union; and by CIBERNED

POSTER N.º 8

Introduced by: Sáez-Valero, Javier

Title:

RISK FOR SARS-COV2 BRAIN ENTRY THROUGH ACE2 AND TMPRSS2 IS NOT INCREASED IN ALZHEIMER'S DEMENTIA, BUT IT IS IN DOWN SYNDROME

Principal Investigator: Javier Sáez-Valero

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Abstract:
Many studies suggest an increased vulnerability of Alzheimer's disease (AD) patients and Down syndrome (DS) subjects to COVID-19; however, it is unclear whether it is derived from an increased risk of brain infection. The SARS-CoV-2 coronavirus infects cells through the angiotensin-converting enzyme 2 (ACE2), and the serine protease TMPRSS2 for the priming of viral spike (S) protein. ACE2 is cleaved from the plasma membrane through constitutive and regulated shedding, whereas TMPRSS2 undergoes autoproteolytic cleavage at the ectodomain to acquire proteolytic activity. For both proteins ectodomain fragments and soluble full-length forms co-exist in biological fluids, but different dynamics in species may overlap during disease progression, hindering the interpretation of changes. We have addressed whether ACE2 and TMPRSS2 are altered in AD and DS subjects regarding vulnerability to SARS-CoV-2 infection. ACE2 and TMPRSS2 are present in cerebrospinal fluid (CSF) including fragments and full-length forms. Increases in cleavage of membrane ACE2 and in CSF levels can be interpreted as protective for SARS-CoV-2 infection since soluble ACE2 can also bind the virus. In AD cases ACE2 full-length species decreased in CSF, mirroring the decrease in membrane resident ACE2. However, DS patients presented less proteolytic processing of ACE2. Regarding TMPRSS2, increases in the active fragment could reflect increased vulnerability to infection. In DS subjects, as expected due to the trisomy in the TMPRSS2 gene, full-length and fragments are increased in CSF. In conclusion, DS patients displayed changes in ACE2 and TMPRSS2 that determined increased vulnerability to SARS-CoV-2 brain infection, but this condition is not associated with proneness to develop AD.

POSTER N.º 9

Introduced by: Wolfram, Martin

Title:

CASCADE AUTOHYDROLYSIS OF ALZHEIMER'S AB PEPTIDES

Principal Investigator: Morten Meldal

Authors: "Martin Wolfram Manish K. Tiwari Tue Hassenkam Ming Li Morten J. Bjerrum Morten Meldal "

Filiation: Department of Chemistry, University of Copenhagen, Denmark.

Abstract:
Protein/peptide self-assembly into amyloid structures associates with major neurodegenerative disorders such as Alzheimer's disease (AD). Soluble assemblies (oligomers) of the Aβ peptide and their aggregates are perceived as neurotoxic species in AD. While screening for synthetic cleavage agents that could break down such aberrant assemblies through hydrolysis, we observed that the assemblies of Ab oligopeptides, containing the nucleation sequence Aβ14–24 (H14QKLVFFAEDV24), could act as cleavage agents by themselves. Autohydrolysis showed a common fragment fingerprint among various mutated Aβ14–24 oligopeptides, Aβ12–25-Gly and Aβ1–28, and full-length Aβ1–40/42, under physiologically relevant conditions. Primary endoproteolytic autocleavage at the Gln15–Lys16, Lys16–Leu17 and Phe19–Phe20 positions was followed by subsequent exopeptidase self- processing of the fragments. Control experiments with homologous D-amino acid enantiomers Aβ12–25-Gly and Aβ16–25-Gly showed the same autocleavage pattern under similar reaction conditions. The autohydrolytic cascade reaction (ACR) was resilient to a broad range of conditions (20–37 °C, 10–150 μM peptide concentration at pH 7.0–7.8). Evidently, assemblies of the primary autocleavage fragments acted as structural/compositional templates (autocatalysts) for self-propagating autohydrolytic processing at the Aβ16–21 nucleation site, showing the potential for cross-catalytic seeding of the ACR in larger Aβ isoforms (Aβ1–28 and Aβ1–40/42). This result may shed new light on Aβ behaviour in solution and might be useful in the development of intervention strategies to decompose or inhibit neurotoxic Aβ assemblies in AD.

POSTER N.º 10

Introduced by: Zhang, Linda

Title:

KLOTHO-VS HETEROZYGOSITY AMELIORATES THE EFFECTS OF APOE E4 ON LONGITUDINAL HIPPOCAMPAL ATROPHY

Principal Investigator: Linda Zhang

Authors: Linda Zhang1, Eva Alfayate1, Miguel Calero 2,3, Miguel Medina 2,3, Bryan Strange 1,4, Pascual Sanchez-Juan 1, and Michel J. Grothe 1

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Abstract:
“Background: KLOTHO-VS heterozygosity (KL-VShet+) has been posited to be a protective factor against age-related disease and cognitive decline, having been associated with increased cortical volumes and brain connectivity, as well as improved cognition in healthy elderly individuals. Conversely, the APOE-ε4 allele is a primary risk factor for the development of Alzheimer’s disease (AD), with ε4 carriers more likely to have greater β-amyloid burden, earlier age of AD onset, and accelerated rates of cognitive decline. Relatively few studies have investigated the interaction between these two genetic factors, with those that have presenting conflicting findings. Methods: 720 cognitively normal elderly participants with 3T T1-weighted MRI, APOE and KL-VS genotyping were selected for cross-sectional analyses from the Vallecas Project cohort, a single-centre 12-year longitudinal study with annual follow-ups. Of these participants, 443 had at least two visits and were included in longitudinal analyses (average follow-up 4.7 years). Linear regression and linear mixed-effects models were conducted to study cross-sectional and longitudinal interactions between KL-VS heterozygosity and APOE-ε4 on FreeSurfer-derived hippocampal volumes, neuropsychological test scores, and plasma AD biomarkers (available for a subset, n=166). Results: A significant KL-VShet+ by APOE-ε4 interaction was observed in longitudinal right hippocampal volumes, where KL-VShet+ ε4-carriers had similar rates of hippocampal atrophy compared to ε4-noncarriers, and slower rates compared to KL-VShet- ε4-carriers (β=52.94, SE=25.03, p=0.03). No significant interactions were found for any other variables. Conclusion: Our study provides evidence to suggest that KL-VS heterozygosity attenuates the detrimental effects of APOE-ε4 on brain structure in healthy elderly adults. “

POSTER N.º 11

Introduced by: Fernández Acosta, Francisco José

Title:

IMPACT OF APOE POLYMORPHISM AND G206D-PSEN1 MUTATION ON HIPPOCAMPAL NEURONS FROM ALZHEIMER´S DISEASE PATIENTS

Principal Investigator: Rebeca Vecino

Authors: R. Vecino1,2, E. Díaz-Guerra1,2, FJ. Fernández Acosta1,2, E. Arribas-González1,2, S. Alberquilla1,2, L. Vicario del Río1, M. Sánchez Calvo1, A. Orellana2,3, L. Boveda1, E. P. Moreno-Jiménez1,2, E. Soriano2,4, JM. García Verdugo2,5, A. Ruiz2,3, R. Moratalla1,2, C. Vicario1,2.

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Abstract:
Alzheimer´s disease (AD) is characterized by progressive neurodegeneration of the main brain areas involved in memory function, namely the entorhinal cortex and hippocampus. The human APOE polymorphism, more specifically the presence of the ε4 allele (coding the APOE4 protein isoform), represents a genetic form of late-onset AD, while mutations in the PSEN1 gene are responsible for many cases of early-onset familial AD. There is increasing evidence of APOE4 being involved in numerous aspects of AD pathogenesis, but the impact of APOE alleles on human neuronal maturation, function and degeneration remains to be fully elucidated. Furthermore, the effect of G206D-PSEN1 mutation on human neurons has been little explored. Using induced pluripotent stem cells (iPSCs) derived from fibroblasts of AD patients carrying the ε3 and ε4 alleles (in homozygosis) or having the G206D-PSEN1 mutation, and from healthy patients, hippocampal neurons were obtained by adding small molecules and growth factors. iPSC-derived neurons expressed hippocampal markers and showed a functional profile, illustrated by glutamate release, electrical activity and synapse formation visualized by electron microscopy and synaptic bouton analysis. In addition, the role of APOE4 in neurodegeneration was confirmed by determining amyloid-beta 42/40, total Tau and phosphorylated Tau in the culture medium, as well as by the presence of an increased number of extracellular amyloid-beta-like plaques and intracellular p-Tau181 aggregates. Finally, APOE polymorphism also affected neuronal morphology and the number of synaptic boutons. Overall, our results point to specific actions of APOE polymorphism and G206D-PSEN1 mutation affecting neuronal maturation, dysfunction and neurodegeneration in AD.

POSTER N.º 12

Introduced by: Burgueño García, Iván

Title:

HIPPOCAMPAL SCLEROSIS IN ALZHEIMER'S DISEASE: DIFFERENTIAL FEATURES IN PATIENTS WITH EARLY ONSET.

Principal Investigator: Iván Burgueño García

Authors: Burgueño-García I1,Saiz-Aúz L1, Ruiz P1, Uceda-Heras A1, López-Martínez MJ1, Rodrigo Lara H2, Rábano A1.

Filiation: 1 Reina Sofia Alzheimer Centre, CIEN Foundation, ISCIII, Spain. 2 Hospital Clínico Universitario Virgen de la Arrixaca, Murcia, Spain.

Abstract:
"INTRODUCTION Hippocampal sclerosis of aging (HS) is defined by severe neuronal loss in the hippocampal cortex, especially in the subiculum-CA1 sectors. HS is primarily related to advanced age, is highly associated with limbic-predominant age-related TDP-43 encephalopathy (LATE) and combines frequently with other highly prevalent pathologies such as Alzheimer's disease (AD) or Lewy body dementia (LBD). In order to address the double association of HS to age and main neurodegenerative causes of dementia here we analyse HS and LATE in two cohorts of late (LOAD) and early onset AD (EOAD) patients, respectively. MATERIALS AND METHODS A total of 106 donated brains from the Vallecas Alzheimer's Reina Sofía (VARS) cohort were included in the LOAD group, while 56 additional brains donated to the CIEN Tissue Bank (CIEN-TB) or the Murcia Region Brain Bank form the EOAD series. The basic data set of the CIEN-TB, including full neuropathological classification of patients, was included in the analysis. For the evaluation of HS, a scale (0-4) recently proposed by our group was used, distinguishing between early (0-2) and late (3-4) HS. RESULTS Whereas LOAD brains showed a higher prevalence of HS (any stage), the proportion of advanced HS was higher in the EOAD group ($p<0.05$). Correlation (CC) between HS stage and both survival time and age at death ($p<0.001$) was highest for EOAD. Similarly, the EOAD group showed the highest CC between HS and LATE stages. CONCLUSIONS EOAD may be associated with a specific clinico-pathological profile of HS that deserves further research in larger cohorts.

POSTER N.º 13

Introduced by: Madrid Lafarga, Nerea

Title:

CHARACTERIZATION OF HIPPOCAMPAL MICROINFARCTS IN POST-MORTEM TISSUE: RESULTS FROM TWO BRAIN BANK COHORTS

Principal Investigator: Nerea Madrid

Authors: "Nerea Madrid Iván Burgueño Alicia Uceda Paloma Ruiz Laura Saiz María José López Martínez Alberto Rábano"

Filiation: Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain

Abstract:
"In aged and advanced dementia patients the combination of highly prevalent pathological conditions (e.g., Alzheimer's, Lewy body and cerebrovascular diseases, as well as limbic-predominant age-related TDP-43 encephalopathy) is most common, with specific lesions in the hippocampal formation. Accordingly, neuropathological phenotyping requires a full characterization of all pathologies involved. Cerebrovascular pathology is particularly variable and difficult to classify, and here we present our experience in the identification and description of microinfarcts in the hippocampus. The full neuropathological and clinical dataset of a large series of brains (n=414) from two brain banks (BT-CIEN and BCRM) were included in the study. Patients were either participants in the VARS dementia cohort (n=166), or external donors (n=248). A full neuropathological work-up was done in the left hemibrain, which included the examination of histological sections from the head and body of the hippocampus. Microinfarcts (MxI) were identified, counted, measured and classified morphologically, and its location within the hippocampal architecture was registered. 9.2% of brains showed hippocampal microinfarcts, and no difference was observed between the cohorts. As expected, cerebrovascular pathology scores were higher in MxI(+), that belonged predominantly to the vascular and mixed dementia diagnostic groups. Most MxI(+) cases were TDP-43(-). A morphological classification of hippocampal microinfarcts is proposed. MxI were most frequent in the CA1 and CA2 sectors of the hippocampal body. A systematic approach to the identification of MxI in the hippocampus, as the one here proposed, will help in the assessment of the contribution of cerebrovascular pathology to cognitive decline in dementia patients."

POSTER N.º 14

Introduced by: López González, Francisco Javier

Title:

A PRELIMINARY ANALYSIS ON PRECUNEUS SHAPE CHANGES IN ALZHEIMER'S DISEASE

Principal Investigator: Emiliano Bruner

Authors: Emiliano Bruner1,2, Rafael Gallareto 3, Francisco J. López-González 2, Linda Zhang2, Michel J. Grothe 2, Pascual Sánchez-Juan 2

Filiation: 1 Museo Nacional de Ciencias Naturales, CSIC, Madrid, Spain; 2 Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain; 3 Universidad de Burgos, Burgos, Spain

Abstract:
"The precuneus suffers structural, functional, and metabolic impairments during the prodromal and early stages of Alzheimer's Disease (AD). However, most of the information on the corresponding anatomical changes comes from imaging methods that do not preserve the original morphology and homology of the involved cerebral regions. In this preliminary study, we consider whether some of these changes can be recognized in terms of gross geometry and spatial arrangement. We compare a sample of non-AD aging individuals with a sample of patients diagnosed with Alzheimer's Disease. Geometric morphometrics was used to analyse shape differences between groups, through a landmark-based model including the precuneus boundaries and other neighboring references. After registration, differences between the two groups in the dorsal region, in terms of gross atrophy, are not patent. Instead, in AD patients there is a significant reduction of the ventral region, namely those parts contiguous with the retrosplenial cortex. These results are interpreted in terms of brain topology and morphological burden of these areas. Keywords: brain shape; geometric morphometrics; parietal lobe; atrophy; retrosplenial cortex."

POSTER N.º 15

Introduced by: Carmo e Pinto, Inês

Title:

A DECADE OF PART: FINDINGS FROM A TERTIARY CENTRE.

Principal Investigator: Inês Carmo e Pinto

Authors: Carmo e Pinto I, Fernandes M, Alves L.

Filiation: Neurology Department, Hospital Egas Moniz, Western Lisbon Local Health Unit, Lisbon, Portugal; NOVA Medical School, NOVA University Lisbon, Lisbon, Portugal.

Abstract:

“INTRODUCTION: In 2014, the term “”primary age-related tauopathy”” (PART) was introduced to describe a subset of patients clinically diagnosed with Alzheimer’s Disease (AD) who exhibit no or minimal amyloid plaques upon post-mortem examination. While PART and AD share considerable overlap, PART typically affects older individuals and is associated with milder cognitive decline. In the absence of clinical diagnostic criteria, a biomarker profile of A-/T+ and mesial temporal lobe atrophy may be considered non-specific biological and radiological markers for PART, potentially enabling its ante-mortem identification. METHDOLOGY: This unicentric, observational, retrospective study analysed consecutive patients presenting with predominant amnestic cognitive complaints and a CSF biomarker profile of A-/T+/N+ between 2014 and August 2024, as recorded in our SNAP database. Exclusion criteria included transitional amyloid and tau cutoff values, as well as clinical syndromes better accounted for by different diagnoses. RESULTS: We identified five cases compatible with PART. Clinical presentation ranged from subjective memory complaints to amnestic mild cognitive impairment, with a mean age at presentation of 77 years. None of the patients progressed to dementia. Radiological findings varied, showing patterns of mesial temporal lobe atrophy and generalized cortical atrophy. All patients had a CSF biomarker profile of A-/T+/N+. In none of these patients was the diagnosis of PART considered. Pathological confirmatory examination was not performed. CONCLUSION: Despite its introduction to the scientific community a decade ago, PART remains underrecognized by clinicians. However, the increasing use of ante-mortem biomarkers holds promise for improving diagnostic accuracy, drawing attention to this neurodegenerative disorder.”

POSTER N.º 16

Introduced by: Valeriano Lorenzo, Elizabeth Lucía

Title:

LEVELS OF P-TAU217 IN BLOOD AND ITS CAPACITY TO PREDICT CHANGE PATTERNS OF COGNITIVE TRAJECTORIES IN ELDERLY

Principal Investigator: Elizabeth Valeriano Lorenzo

Authors: Elizabeth Valeriano-Lorenzo1,2, David García3, Sonia Wagner 1, Alicia Ruiz 1, Ana Belén Pastor 1, Belén Frades 1, Meritxell Valentí 1, Mario Riccardi 1, Ma Ascencion Zea1, Marta Antón1, Teodoro del Ser1, Pascual Sánchez-Juan1.

Filiation: 1Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain. 2Universidad Autónoma de Madrid, Madrid, Spain. 3Universidad Complutense de Madrid, Madrid, Spain.

Abstract:
“Aim Longitudinal trajectories of cognitive performance in older adults are non-homogeneous across time. For this reason, one approach to capturing that heterogeneity is the estimation of patterns of change or latent classes (LC) that explain the formation of groups of subjects with stable or changing cognitive trajectories. We conducted this study to analyse the effect of baseline plasma p-tau217 levels on cognitive change patterns in the elderly. Methods 913 participants, cognitively healthy at baseline, from the Vallecas Project, were selected (583 women, 64%) with a mean baseline age of 73.8±3.8 years and an average follow-up of 10.2±0.6 years. Patterns of change based on the non-linear trajectories of 5 cognitive domains were analysed using Growth Mixed Models (GMM) and adjusting for educational level, baseline age, sex and number of drugs in the medication list. Three LCs explaining individual cognitive change were obtained, and then the distribution of baseline plasma p-tau217 levels and other relevant clinical variables were examined according to the previously established LCs. Results The group with a stable pattern of their cognitive trajectory shows significantly lower concentrations of p-tau217 than the groups with slight cognitive decline or markedly declining, in domains such as delayed verbal memory (P<.001), semantic fluency (P<.001), and processing speed (p < 0.001). In addition, a lower number of drugs in the medication list consumed, a greater speed and motor control ability, lower depression indicators and other clinical variables characterise the group with stable patterns in their cognition. Conclusions Significantly lower concentrations of plasma p-tau217 are associated with a stable longitudinal change pattern and therefore a lower probability of accumulation of AD pathology. Keywords: blood biomarkers, p-tau217, cognitive change pattern, growth mixture model.”

POSTER N.º 17

Introduced by: Marta Aramburu-Núñez

Title:

NOVEL P-TAU MONOCLONAL ANTIBODY-BASED IN-VITRO AND PRECLINICAL THERAGNOSTIC STUDIES FOR TAUOPATHIES

Principal Investigator: Marta Aramburu Núñez

Authors: Marta Aramburu-Núñez1,2, Lara García-Varela2,3,4, Antía Custodia1,2, Noemí Gómez-Lado2,3,4, Mónica Castro-Mosquera1, Mariña Rodríguez-Arrizabalaga1, Manuel Debasa-Mouce1, Juan Manuel Pías-Peleteiro1,2, José Manuel Aldrey1,2, Daniel Romaus-Sanjurjo1,2, Alberto Ouro1,2, Pablo Aguiar2,3,4, Tomás Sobrino1,2.

Filiation: 1NeuroAging Laboratory Group, Clinical Neurosciences Research Laboratory, Health Research Institute of Santiago de Compostela (IDIS), Santiago de Compostela, Spain. 2Centro de Investigación Biomédica en Red en Enfermedades Neurodegenerativas (CIBERNED), Instituto de Salud Carlos III, Madrid, Spain. 3Molecular Imaging Group, Health Research Institute of Santiago de Compostela (IDIS), Santiago de Compostela, Spain. 4Molecular Imaging Biomarkers and Pharmacokinetic Modelling, Center for Research in Molecular Medicine and Chronic Diseases (CiMUS), University of Santiago de Compostela, Spain.

Abstract:
Tauopathies are the primary cause of older people losing their sense of autonomy, which suggests a steady reduction in cognitive function. Early diagnosis of tauopathies is not associated with a successful therapy. Using a new phosphorylated Tau (p-Tau) monoclonal antibody (mAb), which is responsible for tau self-aggregation and control, we present an in-vitro and theragnostic method. In order to identify p-Tau in 89 cerebrospinal fluid (CSF) samples from individuals with moderate cognitive impairment(MCI) and Alzheimer's disease(AD), a diagnostic Sandwich ELISA kit was created. The levels of the tau isoforms exhibited the same behaviour with a rise in CSF as the disease develops, and the proof-of-concept study produced distinct signals beyond the limit of detection while retaining a low intra-assay coefficient of variation. Additionally, we evaluated the impact of p-Tau mAb on behaviour and brain functions in transgenic mice models of tauopathy delivered intraventricularly. According to the findings, p-Tau mAb therapy for four weeks decreased p-Tau levels in the cortex and hippocampus and enhanced motor outcome by postponing hindlimb clasping and latency to fall in the rotarod test. Moreover,p-Tau mAb was radiolabelled with 89Zr and the results in transgenic mice showed that the mAb-89Zr were stable in circulation up to 10 days, but the amount reaching the brain was <0.2%. These findings demonstrated a new theragnostic p-Tau mAb that identifies an early p-Tau biomarker in AD patients.Therefore, it is essential to enhance the quantity of mAb that enters the brain and multicenter clinical studies are required to verify these encouraging results.

POSTER N.º 18

Introduced by: Moreno Gonzalez, Ines

Title:

LATE-LIFE DEPRESSION EXACERBATES COGNITIVE IMPAIRMENT AND TAU-ASSOCIATED PATHOLOGY IN P301S MICE

Principal Investigator: Ines Moreno-Gonzalez

Authors: Vegas-Gomez L1, Arredondo-Alcala MA1, Gutierrez A1,2, Moreno-Gonzalez I1.

Filiation: 1 Departamento Biologia Celular, Genetica y Fisiologia, Instituto de Investigacion Biomedica de Malaga-IBIMA, Facultad de Ciencias, Universidad de Malaga. 2 Centro de Investigacion Biomedica en Red Sobre Enfermedades Neurodegenerativas (CIBERNED)

Abstract:
Recent studies suggest that depression may be a crucial risk factor for the development of cognitive impairment and Alzheimer's disease (AD). There is a strong association between late-life depression and AD, with the onset of AD being accelerated in patients with mild cognitive impairment (MCI) who have a history of depression. Women appear to be particularly vulnerable to this condition. In addition, individuals with MCI who present depressive symptoms have an elevated burden of amyloid-beta (Aβ), the main toxic protein associated with Alzheimer's pathology, and an increased risk of developing AD compared to non-depressed MCI patients. While some transgenic models of AD exhibit depression-like symptoms in advanced stages, the induction of Alzheimer's pathology due to a depressive process has not been thoroughly studied under experimental conditions that emulate late-life depression as a risk factor for AD. The objective of this study is to determine whether depression is a cause, rather than a consequence, of AD development by inducing unpredictable mild chronic stress (CUMS) in tau transgenic P301S mice. Our results indicate that CUMS induction in transgenic animals leads to phenotypic changes related to a depressive state. Behavioral and histological studies suggest that depression-like induction can worse AD pathology. The findings from this project could provide evidence of depression as a risk factor for AD, elucidate its mechanisms of action, identify new early biomarkers, and contribute to the discovery of new therapies for AD.

POSTER N.º 19

Introduced by: López Martínez, María José

Title:

COPATHOLOGY IN PROGRESSIVE SUPRANUCLEAR PALSY: A SPECTRUM OF PATTERNS AND POTENTIAL SYNERGIES

Principal Investigator: María José López Martínez

Authors: María José López Martínez (1), Héctor Rodrigo Lara (2), Laura Sáiz Auz (1), Paloma Ruiz Valderrey (1), Iván Burgueño García (1), Alicia Uceda Heras (1), Alberto Rábano Gutiérrez (1)

Filiation: "1. Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain. 2. Hospital Clínico Universitario Virgen de la Arrixaca, Murcia, Spain."

Abstract:
"Introduction: Progressive supranuclear palsy (PSP) is a sporadic 4-repeat tauopathy with cortical and subcortical involvement. The regional distribution of neuronal loss and tau pathology, and the coexistence of PSP with other neurodegenerative diseases are sources of phenotypical variability. Objectives: Our aim is to analyze the frequency and severity of copathologies in patients with neuropathological diagnosis of PSP. Materials and Methods We analyze a case-series including all PSP cases with neuropathological evaluation in the Neurological Tissue Bank of the CIEN Foundation in Madrid, Spain. We assessed copathologies according to standardized diagnostic criteria and current staging schemes. Results: From 37 patients included, 65% were male. Mean age at death was 75 years (IQR 70-83). Antemortem clinical diagnosis of PSP was made in 54% of cases. Isolated PSP pathology was observed in 24% of brains. The remaining cases exhibited a broad spectrum of combined pathologies: argyrophilic grain disease in 46% of patients, Alzheimer's disease neuropathologic change in 38% of patients and Lewy body pathology in 8% of cases. Corticobasal degeneration overlapped with PSP in 3 cases. One case showed limbic-predominant age-related TDP-43 encephalopathy (LATE) with hippocampal sclerosis, and 2 cases had hippocampal sclerosis with Pick-like hippocampal spherical inclusions in the absence of TDP-43 immunoreactivity. One patient had a genetically confirmed diagnosis of Huntington's disease, which was found to be combined with PSP in the neuropathologic workup. Conclusions: Mixed pathologies are widely prevalent in PSP, brain autopsies remain useful for a better understanding of potential disease synergies and its further impact on targeted therapies.

POSTER N.º 20

Introduced by: Sevilla, Mª Ascension

Title:

FRONTOTEMPORAL DEMENTIA (FTD) - MADRID CONSORTIUM. CREATION OF AN FTD STUDY COHORT FOR THE VALIDATION OF CUTTING EDGE BIOMARKERS

Principal Investigator: Mª Ascension Zea Sevilla

Authors: Mª Ascensión Zea Sevilla1, Frontotemporal Dementia Madrid Consortium2,María Belen Frades Payo1,Elizabeth Valeriano-Lorenzo1, Francisco J López1 Mª,Meritxell Valenti1, Mario Ricciardi,. Isabel López1 Alicia Ruiz1, Sonia Wagner1. Ana Belén Pastor1, Minerva Martinez1, Teodoro del Ser1 Alberto Rábano 1, MªJose López Martínez 1, Alicia Uceda 1, Paloma Ruiz1, Laura Saiz 1, Nekane Moreno . 1, Marta Antón Michel Grothe1 Pascual Sánchez-Juan1

Filiation: 1. Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain. 2. Memory Clinics of the Autonomous Community of Madrid (CAM)

Abstract:
“Background FTD is the third cause of neurodegenerative dementia in our environment. The diagnosis is complex and usually uncertain without neuropathological study. Therefore, the development of any diagnostic technique or biomarker would be of paramount importance for clinical practice. The most common “misfolded” proteins in FTD are Tau and TDP-43. Identifying specific pathogenic species of tau and TDP-43 correlated with the disease would represent a straightforward diagnosis approach. Material and methods The FTD-Madrid Consortium is a multicenter project, coordinated by CIEN, to recruit a cohort of FTD patients with behavioral variant or primary progressive aphasia. A clinical assessment, plasma biomarkers, cerebrospinal fluid analyses, and neuroimaging will be performed on every participant. All the Memory Clinics of the Autonomous Community of Madrid (CAM) participate in this project. The RT-QuIC (Real-Time Quaking-Induced Conversion), a seeding assay technique, will be evaluated to detect pathogenic isoforms of 3R and 4R tau, and with SIMOA technology we will quantify TDP-43 in extracellular vesicles. Also, we will test the diagnostic utility of a set of plasma/CSF biomarkers determined by the SIMOA technique (TDP43, p-Tau 181, t-Tau, ABeta 40, ABeta42, NfL and GFAP) and its combination with RT-QuIC. In a neuropathological cohort from the CIEN Biobank we will also study the same biomarkers trying to validate them. The project is funded by Carlos III Health Institute (PI23/01314. AES-ISCIII). Current Situation Patient recruitment has just started. Conclusions We are committed to creating a deeply phenotyped cohort of FTD patients, aiming to validate new biomarkers, improve the diagnosis of FTD by integrating precision medicine tools, and create a patient identification platform for future clinical trials.

POSTER N.º 21

Introduced by: Mirfakhar, Farzane

Title:

CHARACTERIZATION OF LYSOSOMAL DYSFUNCTION IN STEM CELL MODELS OF FRONTOTEMPORAL DEMENTIA USING SUPER RESOLUTION MICROSCOPY

Principal Investigator: Farzaneh S. Mirfakhar

Authors: Farzaneh S. Mirfakhar[1], Jacob A. Marsh[1], Miguel Minaya[1], David Perlmutter[2], Celeste M. Karch[13]

Filiation: [1]Department of Psychiatry, Washington University in St. Louis School of Medicine, St. Louis, Missouri, USA. [2]Department of Pediatrics, Washington University in St. Louis School of Medicine, St. Louis, Missouri, USA. [3]The Charles F. and Joanne Knight Alzheimer Disease Research Center, Washington University in St. Louis School of Medicine, St. Louis, Missouri, USA.

Abstract:

“Objectives Impaired proteostasis has been implicated in frontotemporal dementia with tau inclusions (FTD-tau). This impairment may lead to the accumulation of the tau protein. Here, we sought to determine whether MAPT mutations that cause FTD-tau impact tau degradation by the lysosome. Methods Human induced pluripotent stem cells (iPSC) expressing MAPT p.R406W and isogenic controls with NGN2 stably expressed within the AAV safe harbor locus. Neurons were cultured for 14 days and evaluated by super resolution microscopy. Results MAPT p.R406W neurons displayed morphological and functional deficits in the lysosomes, including elevated size and density of LAMP1-positive vesicles. These phenotypes were reversed upon correction of the mutant allele with CRISPR/Cas9. Given these lysosomal defects, we sought to investigate the impact on lysosomal-mediated tau degradation using super resolution microscopy. MAPT p.R406W neurons exhibited fewer empty lysosomes compared to isogenic controls, suggesting the degradative capacity is reduced in these vesicles. MAPT p.R406W neurons exhibited a significant increase in tau-retention in the membrane and lumen of LAMP1-positive vesicles compared to isogenic controls. Interestingly, tau phosphorylated at pThr231 was significantly enriched in the membrane of LAMP1-positive vesicles compared to isogenic controls, suggesting defects in the transport of pTau-231 into the lysosome. Time-lapse live imaging data revealed that lysosomes in MAPT p.R406W neurons travel shorter distances and at slower speed while FRAP analysis illustrated similar microtubule stability in MAPT p.R406W and isogenic control neurons. Conclusions Together, our findings suggest that MAPT p.R406W may be sufficient to cause impaired lysosomal function leading to disrupted tau degradation by lysosomes, which may contribute to the development of tau pathology.

POSTER N.º 22

Introduced by: Naranjo, José Ramón

Title:

DREAM LIGANDS ACTIVATE UPR-DEPENDENT NEUROPROTECTION IN A TDP-43 MOUSE MODEL OF FRONTOTEMPORAL LOBAR DEGENERATION

Principal Investigator: Gonzalo-Gobernado, Rafael

Authors: Rafael Gonzalo Gobernado1,2, Paz González 1, Xose M. Dopazo 1,2, Damián Tandalla2, Britt Mellström1,2 and José R. Naranjo1,2.

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Abstract:
“DREAM ligands activate UPR-dependent neuroprotection in a TDP-43 mouse model of Frontotemporal Lobar Degeneration Rafael Gonzalo Gobernado1,2, Paz González 1, Xose M. Dopazo 1,2, Damián Tandalla2, Britt Mellström1,2 and José R. Naranjo1,2. 1 Centro de Investigación Biomédica en Red de Enfermedades Neurodegenerativas (CIBERNED), Madrid, Spain. (rd.gonzalo@cnb.csic.es, naranjo@cnb.csic.es) 2 Centro Nacional de Biotecnología, Consejo Superior de Investigaciones Científicas (CSIC), Madrid, Spain. (rd.gonzalo@cnb.csic.es, naranjo@cnb.csic.es) Funding: CIBERNED (PI2022/02), Asahi Kasei Pharma and Fundación Luzón Frontotemporal dementia is a neurodegenerative syndrome that encompasses a heterogeneous group of diseases, all of which have in common the degeneration of the frontal and temporal lobes (Frontotemporal Lobar Degeneration, FTLD). Patients exhibit personality changes, social behaviour and language problems, amnesia and movement disorders. Around 45% of FTLD patients present neuronal cytoplasmic inclusions of TDP-43, a phenomenon considered as a key pathological feature. The aggregation of TDP-43 and ubiquitinated proteins alters proteostasis and triggers the unfolded protein response (UPR). Our group identified the interaction of ATF6, a UPR key regulator, with the neuronal calcium sensor DREAM, as a potential therapeutic target in neurodegeneration, and demonstrated that the modulation of this interaction using DREAM binding molecules exerted neuroprotection in models of neurodegenerative diseases. The aim of this work was to study the potential neuroprotective effect of repaglinide, a known DREAM binding molecule, in the hTDP-43/CamKII□ mouse model of FTLD Repaglinide treatment increased lifespan, improved social deficits (3-chamber test) and cognitive impairment (Novel Object Recognition, marble burying and Y-maze tests) of TDP-43 mice. Furthermore, TDP-43 animals treated with repaglinide showed reduced neuronal loss and microgliosis and an increased ATF6 processing in the frontal cortex. These findings suggest that the modulation of the DREAM-ATF6 interaction by repaglinide improves FLTD-related symptoms and neuronal loss, potentially through the regulation of ATF6 signaling. Further research will be needed to fully elucidate the mechanisms of action by which repaglinide ameliorates FTLD progression in this model of TDP-43-mediated pathology.

POSTER N.º 23

Introduced by: Martínez Castillo, Minerva

Title:

PLASMA EXTRACELLULAR VESICLES AS A POTENTIAL BIOMARKER IN AMYOTROPHIC LATERAL SCLEROSIS

Principal Investigator: Minerva Martinez-Castillo

Authors: Minerva Martinez-Castillo1, Sonia Wagner-Reguero1, Alicia González Ruiz 1, Iván Burgueño-García 1, Paloma Ruiz-Valderrey 1, Laura Saiz-Aúz 1, Pamela Martino-Adami 2, Selçuk Özdemir3, Alberto García-Redondo4, Alberto Rábano 1, Alfredo Ramirez 2,3,5,6,7, Anja Schneider 3, Pascual Sánchez-Juan 1

Filiation: 1Alzheimer's Centre Reina Sofia-CIEN Foundation-ISCIII. Madrid, Spain. 2Division of Neurogenetics and Molecular Psychiatry, Department of Psychiatry and Psychotherapy, Faculty of Medicine and University Hospital Cologne, University of Cologne. Cologne, Germany. 3German Center for Neurodegenerative Diseases (DZNE). Bonn, Germany. 4ALS Research Laboratory Unit, Department of Neurology, Hospital Universitario 12 de Octubre. Madrid, Spain. 5Department of Neurodegenerative Diseases and Geriatric Psychiatry, University Hospital Bonn, Medical Faculty. Bonn, Germany. 6Department of Psychiatry and Glenn Biggs Institute for Alzheimer's and Neurodegenerative Diseases. San Antonio, United States of America. 7Cluster of Excellence Cellular Stress Responses in Aging associated Diseases (CECAD), University of Cologne. Cologne, Germany.

Abstract:
Protein TDP-43 accumulates in more than 95% of the brains of ALS patients, which normally occurs within the nucleus of brain cells but is localised in atypical regions in these patients. Detection of TDP-43 in the cerebrospinal fluid or blood of ALS patients is a potential diagnostic biomarker of great interest. However, determinations of this free protein have not been successful in diagnosing neurodegenerative diseases. The CIEN Foundation, in collaboration with the research groups of Prof. Dr. Anja Schneider (DZNE, Bonn, Germany) and Prof. Dr. Alfredo Ramirez (Uniklinik Köln, Germany), has just started a new project to study TDP-43 not in free form but within extracellular vesicles (EVs) circulating in the blood. Isolation and characterisation of EVs are performed in plasma samples, following serial centrifugation steps and size exclusion chromatography to purify small (≈80-150 nm) and medium (≈100-400 nm) EVs. Concentration of TDP-43 and other potential biomarkers in the vesicular interior are measured using the ultrasensitive Simoa. The determination of vesicle number and size is carried out by Nanoparticle Tracking Analysis (NTA). In addition, it is intended to further characterise neuropathological samples from ALS patients by quantification of histological lesions. In this way, neuropathological findings could be correlated with these biomarkers, and proteomic analyses both in their free and intravesicular form could be carried out. In conclusion, the aim is to discover peripheral biomarkers that will allow us to make an early and accurate diagnosis of ALS, as well as to estimate disease progression and eventual responses to future treatments.

POSTER N.º 24

Introduced by: Conte, Carmela

Title:

TOLL-LIKE RECEPTOR 4 UPREGULATION IN THE SUBSTANTIA NIGRA PARS COMPACTA AND MEDIAL TEMPORAL GYRUS FROM PARKINSON'S DISEASE PATIENTS: A POTENTIAL MECHANISM DRIVING INFLAMMATORY RESPONSE.

Principal Investigator: Carmela Conte

Authors: Carmela Conte1*, Angela Ingrassia2, John Brevè2, John J. Bol2, Evelien Timmermans-Huisman2, Anne-Marie van Dam2, Tommaso Beccari1 and Wilma D. J. van de Berg2.

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Abstract:
"Introduction Neuroinflammation and immune dysfunction play a critical role in the pathophysiology of Parkinson's disease (PD) and correlates with the accumulation and aggregation of alpha-synuclein (αSyn). Toll-like receptors (TLRs) are a group of innate immune receptors widely distributed in the CNS able to sense several exogenous and endogenous stimuli which trigger inflammatory responses and cause the accumulation of αSyn. On the other hand, αSyn can bind the TLRs and evoke an inflammatory response in attempt to clear itself and restore brain homeostasis. Therefore, the precise role of TLRs is debated as helpful and harmful effects can occur. In the present study, we investigated the expression levels of TLR4 and αSyn in substantia nigra (SN) and medial temporal gyrus (GTM) of patients with PD. Moreover, we analysed the posphpS129 αSyn (pS129 αSyn) levels considered the most common used marker of αSyn pathology, and the possible co-localization with TLR4. Moreover, we examined Iba1 expression as a marker of microglia activation, and its co-localization with TLR4. Materials and methods Post-mortem human brain tissue was obtained from the Netherlands Brain Bank and the Department of Anatomy and Neurosciences of VU University Medical Center (VUmc, Amsterdam, The Netherlands). A total of 60 samples (15 cases and 15 control donors per GTM and SN) were analysed by qPCR. A total of 25 samples (6 PD/PDD donors and 6 controls for GTM; 6 PD/PDD donors and 7 controlled cases for SN) were analysed by immunofluorescence and confocal microscopy for TLR4, pS129-αSyn, and Iba1. The presence of somatic pS129-αSyn immunoreactivity was used to distinguish between cells with and without cytopathology. Results In the present study, we observed that the levels of TLR4 were increased in the SN and GTM of patients with PD, while αSyn was downregulated, probably because of the significant depletion of dopaminergic neurons. In PD patients, we also found co-localization between TLR4 and pS129-αSyn and between TLR4 and glial Iba-1 in SN Lewy bodies and pyramidal neurons within GTM compared with the same regions of the control donors. Conclusions Our findings provide evidence that TLR4 is up-regulated in PD patients. Moreover, the co-localizations between TLR4 and pSer129-αSyn and Iba1 suggest a physical interaction that may evoke the activation of inflammatory response.

POSTER N.º 25

Introduced by: Rivas Santisteban, Rafael

Title:

CANNABINOIDS AND ANGIOTENSIN II MODULATE CALCIUM HANDLING IN STRIATAL NEURONS

Principal Investigator: Rafael Rivas-Santisteban

Authors: Rafael Rivas-Santisteban1,2, Ana Muñoz2,3, Jaume Lillo2,4, Iu Raïch2,4, Ana I. Rodríguez-Pérez2,3, Gemma Navarro2,4*, José L. Labandeira-García2,3*, Rafael Franco2,6,7

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Abstract:

"Parkinson's disease (PD) is a neurodegenerative condition characterized by the degeneration of dopaminergic neurons within the substantia nigra, which results in motor dysfunction. Current therapeutic approaches primarily aim to manage symptoms, highlighting the urgent need for interventions that address the root cause of neuronal death. Emerging evidence in the scientific literature suggests that the endocannabinoid system, particularly through the activation of cannabinoid receptor 1 (CB1R)—the most prevalent G protein-coupled receptor (GPCR) in central nervous system (CNS) neurons—holds promise for neuroprotection in PD. This study examines the interaction between the CB1R and the angiotensin II type 1 receptor (AT1R), the latter being involved in maintaining neuronal calcium homeostasis, a crucial factor in neurodegeneration and cell death. We explored the functional dynamics of the AT1-CB1 receptor complex (AT1CB1Hets) concerning signaling pathways and its potential role in PD pathophysiology. Notably, our findings revealed that CB1R-selective agonist (ACEA) and antagonist (rimonabant) modulated calcium signaling induced by AT1R activation. The direct association between these receptors to form AT1-CB1 heteromers (AT1CB1Hets) was confirmed through bioluminescence resonance energy transfer (BRET2) assays. Additionally, our results indicate that cannabinoids decrease AT1R-mediated signaling in striatal neurons. In situ proximity ligation assays (PLA) further validated the formation of AT1CB1Hets in neurons, with a higher prevalence of these complexes near the neuronal soma (NeuN positive) as compared to more distant regions such as dendrites (MAP2 positive). Furthermore, AT1CB1Hets expression was evaluated in the striatal neurons of rats subjected to a 6-hydroxydopamine (6-OHDA) model of PD. A reduction in AT1CB1Hets expression was observed in neurons from lesioned animals relative to non-lesioned controls. Interestingly, AT1CB1Het expression fluctuated depending on the lesion status and the effects of L-DOPA treatment, such as the development of dyskinesias versus the absence of involuntary movements. In animals that exhibited L-DOPA-induced dyskinesias, a partial restoration of AT1CB1Het expression was noted. These findings suggest that AT1CB1Hets may play a compensatory role in modulating the susceptibility to L-DOPA-induced dyskinesias in Parkinson's disease.

POSTER N.º 26

Introduced by: Monteiro, Sara

Title:

CEREBRAL BLOOD FLOW DYNAMICS IN A PARKINSONIAN MOUSE LINE

Principal Investigator: Sara Pires Monteiro

Authors: “Sara Pires Monteiro Ruxanda Lungu Patricia Figueiredo Noam Shemesh”

Abstract:
Parkinson’s disease (PD) is a prevalent neurodegenerative disorder typically manifesting α-synuclein (α-syn) deposition, loss of dopaminergic neurons, brain atrophy, severe motor symptoms and cognitive decline. Interestingly, the vascular system may be also implicated in the disease, with patients also reported to exhibit reduced venous outflow and lower perfusion compared to healthy subjects. Here, we harness a mouse model of PD exhibiting extensive human α-syn deposition to investigate cerebral blood flow properties in PD. We use a novel setup enabling high resolution Pseudo-Continuous Arterial Spin Labelling, a non-invasive technique for perfusion mapping in-vivo without injection of contrast agents. Adult C57BL/g mice (~20 weeks old, weights 25–30g) (N=3), the transgenic αSYN mouse model (C57BL/6-DBA/2 Thy1- αSYN) (N=3) and their wildtype littermates (healthy controls, N=3) 36-42 weeks of age and weighing 42±15g, were housed in 12h/12h light/dark cycles with ad-libitum access to food and water. Animals were sedated using 1.5-2.5% isoflurane. Respiratory rate was kept at 60-90bpm. An unbalanced pCASL sequence was used as described in Hirschler et al. (2018). The mice were positioned on top of a custom-built ramp to control carotid positioning for increased labelling efficiency. The labelling plane was positioned at the mouse neck (~8mm below the isocenter), labelling duration (LD)=3s, post-labelling delay (PLD)=300ms. A single-shot EPI was implemented: FOV=12x12mm2, slice thickness=0.5mm, spatial resolution=100x100m2, TR/TE=4000/25ms, 30 repetitions, Tacq=4min. For cerebral blood flow (CBF) quantification, the T1 map was obtained from an inversion recovery sequence. A pCASL encoded FLASH was employed to estimate the inversion efficiency (IE) 3mm above the labelling plane (PLD=0ms, LD=200ms). CBF maps (ml/100g/min) was calculated pixel-by-pixel to obtain high resolution CBF maps. T-tests were used to compare the average whole-brain CBF values across 3 different groups. In the CBF maps, clear differences in perfusion brain-wide can be observed with pronounced increased perfusion in the PD and their WT littermates when compared to the C57BL/g are obvious, mostly in cortical and thalamic regions. Distributions across animals of the whole-brain average CBF further show that the PD model shows significantly increased perfusion compared to the C57BL/g mouse line, but not compared to its wildtype littermates, which also exhibit higher CBF than the C57BL/g (paired t tests, $p<0.05$). Our findings suggest that the PD mouse line and their WT littermates have altered perfusion properties across their entire brains compared to control C57bl/6 mice. Thus, local effects of α-syn deposition may not fully explain the altered vascular properties. The much higher values compared with the standard C57BL/g mouse line likely reflect either an underlying genetic difference between the strains causing higher perfusion in the PD line, or otherwise reflect other auxiliary factors (e.g. how isoflurane affects perfusion between the lines). Future experiments in awake animals and physiological measurements of e.g. heart-rate, blood pressure, and vascular density could further narrow down the sources of these differences. Nevertheless, our findings highlight the importance of accounting for these potential sources of variability in future work with these lines

POSTER N.º 27

Introduced by: Ruiz, Alicia

Title:

POST-MORTEM CSF FOR DETECTING A- SYNUCLEIN SEEDING.

Principal Investigator: González Ruiz. A

Authors: González Ruiz. A1, Wagner Reguero. S 1, Pastor. AB1, Ruiz Calvo. A1, Martínez. M1, Moreno Manzano. N1, Burgueño García. I 1, Saiz Aúz 1 . L, Rábano Gutiérrez. A1, Schmitz. M2, Canaslan. S 2, Zerr. I 2, Sánchez Juan. P1 .

Filiation: 1. Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain. 2.Department of Neurology, National Reference Center for TSE, The German Center for Neurodegenerative Diseases (DZNE), Georg-August-University, University Medicine Göttingen, Göttingen, Germany.

Abstract:

"Synucleinopathies such as Parkinson's disease (PD), dementia with Lewy bodies (DLB), and multiple system atrophy (MSA) are characterized by the accumulation of misfolded α-synuclein aggregates in the central nervous system. Seed amplification assay (SAA), such as real time quaking-induced conversion (RT-QUIC), have emerged as highly sensitive and specific method for detecting this misfolded α – synuclein in biological samples. The main fluid used is cerebrospinal fluid (CSF). There are a few publications demonstrating the usefulness of postmortem CSF for RT-QUIC. Our aim is to validate post-mortem CSF S to detect the α-synuclein seeding by RT-QUIC. Ten postmortem CSF samples were collected from VARS project participants, classified according to their neuropathological diagnoses. •

Synucleinopathy group: Four participants neuropathologically diagnosed as DLB and one as PD. •

ALS group: Five participants neuropathologically diagnosed as amyotrophic lateral sclerosis (ALS). This assay was performed at the Universitätsmedizin laboratory in Göttingen, Germany. Each sample was run in triplicate by the FluoStar Omega Series over 24 hours. In the synucleinopathy group, three participants diagnosed as DLB were considered positives, their triplicates got amplification (3/3) and crossed Threshold (Fmean1: 11,051 RFU; Fmean2= 8,472 RFU and Fmean3: 16,629 RFU). On the other hand, within the ALS group, the five participants didn't get amplification and were considered as negative. These results support the utility of postmortem CSF as samples for α-synuclein SAA. The future goal will be to study a larger sample, and correlate α-synuclein seeding with neuropathological variables to validate its clinical use.

POSTER N.º 28

Introduced by: Martínez Castillo, Minerva

Title:

ASSOCIATION BETWEEN PLASMA DOPA DECARBOXYLASE AND NEUROPATHOLOGY IN DEMENTIAS: INSIGHTS FROM THE VARS COHORT

Principal Investigator: Minerva Martinez-Castillo

Authors: Minerva Martinez-Castillo1, Sonia Wagner-Reguero1, Iván Burgueño-García 1, Paloma Ruiz-Valderrey 1, Mario Ricciardi 1, Laura Saiz-Aúz 1, Pamela Martino-Adami 2, Alfredo Ramirez 2,3,4,5,6, Alberto Rábano 1, Pascual Sánchez-Juan 1

Filiation: 1Alzheimer's Centre Reina Sofia-CIEN Foundation-ISCIII, Madrid, Spain. 2Division of Neurogenetics and Molecular Psychiatry, Department of Psychiatry and Psychotherapy, Faculty of Medicine and University Hospital Cologne, University of Cologne. Cologne, Germany. 3Department of Neurodegenerative Diseases and Geriatric Psychiatry, University Hospital Bonn, Medical Faculty. Bonn, Germany. 4German Center for Neurodegenerative Diseases (DZNE). Bonn, Germany. 5Department of Psychiatry and Glenn Biggs Institute for Alzheimer's and Neurodegenerative Diseases. San Antonio, United States of America. 6Cluster of Excellence Cellular Stress Responses in Aging associated Diseases (CECAD), University of Cologne. Cologne, Germany.

Abstract:
DOPA decarboxylase (DDC) has been proposed as a novel cerebrospinal fluid (CSF) biomarker with increased concentrations in Lewy body disorders. However, findings on DDC levels in plasma are inconsistent between studies. Patients from Vallecas Alzheimer Reina Sofía (VARS) cohort were all demented, diagnoses were confirmed by autopsy (Alzheimer's disease [AD], N=103; vascular dementia [VD], N=14; dementia with Lewy bodies [DLB], N=12), and were classified into groups considering the depigmentation of substantia nigra (SN; 0=normal, 1=neuronal loss and gliosis, 2=massive neuronal loss and severe gliosis) followed by the quantification of neurons in this region. Plasma ante-mortem DDC levels were quantified as part of the Olink Explore 3072. DDC levels and number of neurons were compared between groups using generalized linear models, adjusted for age and sex. Correlations between plasma DDC and neuropathology were performed by Spearman's rank correlation. Plasma DDC levels were not significantly different between diagnostic groups. However, DLB subjects had lower number of neurons in SN, being this difference significant compared to AD and VD. In addition, plasma DDC levels were not significantly different between different stages of SN depigmentation. Negative association was observed between the number of neurons in SN and the main neuropathological variables for DLB (Braak stage for a-synuclein and Lewy Pathology Consensus Criteria [LPC]) but no correlation of these with plasma DDC. In contrast to CSF, plasma DDC may have limited use as a diagnostic biomarker. Even so, it is needed to consider and further study the effect of medication uses when analyzing plasma DDC.

POSTER N.º 29

Introduced by: Lungu, Ruxanda

Title:

MULTIMODAL EVALUATION OF SENSORY DEFICITS IN PARKINSON'S DISEASE: INSIGHTS FROM FMRI, C-FOS, AND CBF STUDIES

Principal Investigator: Ruxanda Lungu

Authors: "Ruxanda Lungu Francisca F. Fernandes Sara Monteiro Tiago F. Outeiro Noam Shemesh"

Abstract:
Parkinson's disease (PD) is primarily known for its severe motor symptoms and cognitive decline, but the involvement of the brain's sensory systems, including olfactory and visual deficits, is less well understood and often overlooked. In this study, we report abnormalities in BOLD-fMRI responses along the olfactory and visual pathways in an α-synuclein mouse model of PD. We validate these findings by assessing neuronal origins through C-FOS protein expression levels and ASL measurements. Our fMRI results demonstrated decreased activity in most sensory areas, which was corroborated by reduced C-FOS staining, confirming a neural basis for the observed abnormalities. Additionally, ASL measurements ruled out any vascular differences that could confound the fMRI signals, solidifying the sensory deficits as a result of PD pathology.

POSTER N.º 30

Introduced by: Saiz Aúz, Laura

Title:

GENOMICS AND DIGITAL NEUROPATHOLOGICAL PHENOTYPING OF IBERIAN BRAINS (GADIR): A CHALLENGE FOR SPANISH AND PORTUGUESE BRAIN BANKS

Principal Investigator: Laura Saiz Aúz

Authors: Laura Saiz1, Victoria Fernández2, María José López1, Alberto Rábano1, on behalf of the Neurological Tissue Banks Working Group (ISCIII Platform for Biomodels and Biobanks).

Filiation: 1 Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain. 2 Fundacio ACE. Institut Català de Neurociènces Aplicades.

Abstract:
“GADIR is an ambitious endeavour aimed at the genomic characterization of thousands of donated brains in Spain and Portugal with a full and updated neuropathological assessment. Additionally, the project will focus on a subset of early (EOAD) and late onset (LOAD) Alzheimer’s disease brains, with deep neuropathological phenotyping and digital characterization of key histological lesions. GADIR is a 3-year project starting in autumn 2024 and based on the collaboration between CIEN and ACE Foundations. Here we present the main organizational and logistic aspects of GADIR. Over 3500 brains from 16 brain banks (BB) in Spain and Portugal with diverse neuropathological diagnoses will be studied. We estimate that around 50% of them will bear a main diagnosis of Alzheimer’s disease. Aim 1 establishes the coordination with BB for collection and harmonization of samples and associated metadata (1.1), and for revision and updating of neuropathological classification of brains (1.2). Aim 2 will be centred on genotyping of samples at CEGEN (2.1.), and calculation of polygenic risk scores for various pathologies (2.2). Aim 3 will focus on GWAs of neuropathological traits (3.1), in-silico functional exploration (3.2), and replication of findings in other datasets (3.3). Aim 4 will be directed to digitalization and AI/ML analysis of selected histological sections of 350 EOAD and LOAD brains (4.1 and 4.2), and to genome/morphology analysis for selected pathological traits (4.3). Logistics of samples and associated data and fine tuning between the coordination centres and BB will be crucial for the success of this ambitious and unique project.

POSTER N.º 31

Introduced by: Bonilla Escribano, Pablo

Title:

PREDICTING MILD COGNITIVE IMPAIRMENT IN HEALTHY INDIVIDUALS UP TO 9 YEARS BEFORE ITS ONSET: A MULTISOURCE APPROACH

Principal Investigator: Pablo Bonilla Escribano

Authors: Pablo Bonilla-Escribano1, Linda Zhang1, Teodoro Del Ser 1, Pascual Sánchez-Juan1, Jussi Tohka 2 and Bryan Strange 1,3

Filiation: "1. Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain. 2University of Eastern Finland, A.I. Virtanen Institute for Molecular Sciences, Kuopio, Finland. 3Laboratory for Clinical Neuroscience, CTB, Universidad Politécnica de Madrid, Spain"

Abstract:

"Age-associated cognitive decline is an open research question, due to the lack of accurate markers to determine which cognitively healthy individuals will undergo a neurodegenerative process at the earliest pre-clinical stages. Here, we study how different feature sources (or modalities) can predict which individuals will develop mild cognitive impairment (MCI). Data of 934 participants from the Vallecas project, cognitively normal at baseline, were used: 113 developed MCI during the 9 follow-up visits (converters), and 821 were taken as controls. Thus, this analysis simultaneously predicts MCI conversion in the next year, in two years, …, up to 9 years. An initial set of 742 features from 9 modalities, all measured at the first visit, was considered. A logistic regression regularized with an elastic net was used to parsimoniously select groups of features. The analysis was repeated for each feature source combination independently. Repeated nested cross-validation was used for model assessment and hyperparameter tuning via Bayesian optimization, thereby addressing the risk of data leakage and overfitting. The dataset was split 7 times into 25 folds across the two levels. The best performing models achieve an AUC of ~0.75. Cognitive examination and the brain MRI analysis with the SPM software provided the best performance on their own. The worst performing sources were typical parameters included in visual neuroradiological reporting, and the neurological examination. Overall, memory performance, genetic factors (like APOE and polygenic risk scores), and patters of atrophy in the entorhinal cortex, amygdala and hippocampus were the most important predictors."

POSTER N.º 32

Introduced by: Osta Pinzolas, Rosario

Title:

CLINICAL PARAMETERS ARE STRONG PROGNOSTIC FACTORS OF PROGRESSION TO DEMENTIA IN AN ELDERLY COHORT OF MCI PATIENTS.

Principal Investigator: Nora Molina Torres

Authors: Nora Molina Torres (1,2,3) | Carlos Platero Dueñas (4)| María Abadía Morales (5) | Laura Moreno Martínez (1,3) | Pol Andrés Benito (6)| Mónica Povedano Panades (6) | Oscar Pérez Berasategui (2) | Pilar Mesa Lampré (2) | Ana Cristina Calvo Royo (1,3) | Concepción de la Cámara Izquierdo (3,7) | Rosario Osta Pinzolas (1,3)

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Abstract:

“Introduction: Mild Cognitive Impairment (MCI) is a state between dementia and healthy ageing. This study describes the clinical features of a cohort of elderly patients and the role of p-tau 181 during disease progression. The prognostic role of cognitive, functional and frailty scales was also considered. Methods: This study was approved by the regional ethics comitee (CEICA, PI-19-455). This is a longitudinal prospective nested case-control study. Each patient had a first interview, and then two yearly interviews to monitor clinical progression. Patients performed neuropsychological and functional tests (MMSE, clock test, verbal fluidity, Barthel’s Index, Lawton’s Index, EURO-D). Patients over 70 years old were included if they matched the MCI International Working group criteria. P-tau 181, NFL, GFAP, abeta-40, abeta-42 and total-tau were measured in plasma samples from each patient (SIMOA by Quanterix). A Disease Progression Model (DPM) was designed to predict survival time. Results: 59 patients were included. Median age was 82,5+/-5,1; 68% of the sample were women. The patients were classified in a progression group (pMCI, 27 patients, 46%), and a cognitive stable group (sMCI, 32 patients, 54%). Depression was around 37% in both groups, and frailty was 50% (sMCI) vs 77%(pMCI). 57% patients had over 20 ng/ml of p-tau181. Of all clinical and molecular measures, only MMSE had prognostic value of progression from MCI to dementia. Conclusions: In a cohort of elderly MCI patients, MMSE has a strong prognostic value. P-tau 181 supported the etiological diagnosis of MCI, but it didn’t influence prognosis. “

POSTER N.º 33

Introduced by: Frades Payo, María Belén

Title:

CLINICAL FEATURES OF MIXED NEURODEGENERATIVE PATHOLOGIES IN AGING BRAINS

Principal Investigator: M. Belén Frades-Payo

Authors: M. Belén Frades-Payo1 ; E. Lucía Valeriano-Lorenzo1 ; Alberto Rábano 1,2 ; M. José López Martínez 1,2 ; Alicia Ruiz 1 ; Sonia Wagner 1 ; Nekane Moreno 1 ; Mario Ricciardi 1 ; M. Ascensión Zea1 ; Merixell Valentí1 ; Pascual Sánchez-Juan1 and Teodoro del Ser 1 .

Filiation: 1. Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain; 2., Clinical platform and Biobank, Madrid, Spain.

Abstract:

“ Objective: Analyze the impact of the association of multiple brain pathologies on cognitive function Methodology and Sample: Clinical and pathological data of 164 subjects (78% female; mean age at exitus: 87.3±6.7) with dementia and postmortem neuropathological study. The presence of Alzheimer disease lesions (AD), Lewy bodies (LB), TDP-43 deposits (LATE), small vessel lesions (SVL), argyrophilic grains (AG) and age-related tau astrogliopathy (ARTAG) was recorded, and quantified. The number of pathologies was correlated with cognitive performance (Severe MMSE, Semantic fluency), behavioral disturbances (NPI), functional (Barthel Index), and motor status (Tinetti) at baseline and ante-mortem, adjusting for age at the clinical onset of cognitive decline and sex. Findings: The higher number of pathologies is significantly associated with older age, longer clinical history and more brain atrophy. The number of pathologies is negatively and significantly correlated with cognitive performance functional and motor status both at baseline and at pre-morten assessments; however, it is not correlated with the changes during the observation period. Conclusions: The mixed pathology is very frequent and is associated with more extended age and duration of disease, with worse cognitive, functional and motor status at baseline and ante-mortem time, and with more brain atrophy. However, it does not determine a different clinical progression.

POSTER N.º 34

Introduced by: Castro Labrador, Sandra

Title:

THE EFFECT OF ALZHEIMER'S DISEASE CO-PATHOLOGY ON COGNITIVE PHENOTYPE AND FDG-PET PATTERNS IN PARKINSON'S DISEASE WITH COGNITIVE IMPAIRMENT

Principal Investigator: Sandra Castro-Labrador

Authors: Sandra Castro-Labrador1,2, Jesús Silva-Rodríguez1,2,3, Miguel Ángel Labrador-Espinosa2,3,,4, Laura Muñoz-Delgado2,3, Pablo Franco-Rosado2,3, Ana María Castellano-Guerrero2, Daniel Macías-García2,3, Silvia Jesús2,3, Astrid Adarmes-Gómez2,3, Elena Ojeda-Lepe2, Fátima Carrillo2,3, Juan Francisco Martín-Rodríguez2,3, San Eufrasio M. 2, Cristina Pérez-Calvo2, Nicholas J. Ashton4,5,6, Henrik Zetterberg5,6, Florinda Roldan Lora7, David García-Solís8, Pablo Mir2,3,9, Michel J. Grothe1,2,3

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Abstract:

"Objective: To explore how Alzheimer's disease (AD) co-pathology affects the pattern of cortical neurodegeneration in patients with Parkinson's disease (PD) and cognitive impairment (CI). We used plasma ptau217 to study the effect of AD co-pathology on APOE4 genotype, cognitive profile and cortical hypometabolism on FDG-PET in a well-characterized cohort of PD and CI patients. Methods: Eighty-eight PD patients were classified into PD-CI (N=50; 24 PD-MCI, 26 PDD) and PD with normal cognition (PD-CN; N=38) using neuropsychological testing with the PD-Cognitive Rating Scale. All underwent blood sampling and FDG-PET scanning. Plasma ptau217 levels were measured using the ALZpath ptau217 Simoa immunoassay, with a threshold of 0.4 pg/mL for ptau217 positivity. APOE4 alleles were genotyped and coded as a binary variable. FDG-PET data was processed using SPM12 and brain-wide hypometabolism patterns (vs PD-CN) were assessed across 52 atlas-defined brain regions. Results: Fourteen PD-CI (28%) and 5 PD-CN (13%) patients were classified as ptau217(+). PD-CN-ptau217(+) were excluded from further analysis. PD-CI-ptau217(+) patients showed a higher prevalence of APOE4 carriers (50% vs 16%, p=0.04) and more impaired memory scores (p=0.03). When compared to PD-CN, both PD-CI-ptau217(-) and PD-CI-ptau217(+) showed significant hypometabolism in posterior-occipital, temporal, and frontal areas (p<0.05, FDR-corrected), but hypometabolism in PD-CI-ptau217(+) was considerably more extensive, particularly in temporo-parietal areas. Conclusions: AD co-pathology results in a more memory-predominant cognitive profile and AD-like neurodegeneration phenotype in PD-CI. Novel plasma biomarkers may significantly facilitate clinical detection of AD co-pathology, which may have important implications for personalized diagnosis, prognosis, and treatment of PD patients.

POSTER N.º 35

Introduced by: Iláco, Maria Carolina

Title:

ACCURATE SEGMENTATION OF BRAIN REGIONS OF INTEREST IN [18F]FDG PET IMAGES TO IMPROVE QUANTITATIVE ASSESSMENT AND DIAGNOSIS OF NEURODEGENERATIVE DISEASES USING ARTIFICIAL INTELLIGENCE

Principal Investigator: Maria Carolina Iláco

Authors: Maria C. Iláco (1,2), Francisco Oliveira (1), Cláudia Constantino (1), José M. Fonseca (2), Durval C. Costa (1)

Filiation: 1 Nuclear Medicine - Radiopharmacology, Champalimaud Foundation, Lisbon, Portugal. 2 NOVA School of Science and Technology, NOVA University of Lisbon, Caparica, Portugal.

Abstract:

“Aim: Precise quantification of grey matter (GM) uptake of [18F]FDG helps in the differential diagnosis of neurodegenerative diseases. This study assesses the feasibility of using [18F]FDG PET images for automatically segmenting brain regions of interest (ROI) and its impact on [18F]FDG uptake quantification. Methods: The dataset comprises 264 subjects (cognitively normal, mild cognitive impairment, and Alzheimer’s disease) from the Alzheimer’s Disease Neuroimaging Initiative database. Each subject had both [18F]FDG PET and MRI scans. Two [18F]FDG PET segmentation methods were compared on 23 brain ROI: artificial intelligence-based (FDG-AI-based), and FDG-Atlas-based (standard approach). MRI-based segmentation was considered the gold standard. Segmentation performance was evaluated using a measure of overlap between segmentations – the Dice similarity coefficient (DSC). The agreement and/or correlation of the uptake quantification based on the different segmentation methods was assessed using the intraclass correlation coefficient (ICC) and Pearson correlation coefficient (r). Results: For the FDG-AI-based segmentation, the median DSC was 0.80, consistent across all subject groups and ROI ($0.71 \leq DSC \leq 0.93$). Agreement on the [18F]FDG GM uptake quantification using the FDG-AI-Based and MRI-based segmentation methods was excellent ($0.89 \leq ICC \leq 1.00$, $0.90 \leq r \leq 1.00$), with mean absolute deviation of 2% (maximum 16%). Regarding the FDG-Atlas-based method, the quantification results were significantly inferiors ($0.46 < r < 0.96$) and less consistent among subjects’ groups. Conclusion: The FDG-AI-based segmentation method is reliable, originating more accurate quantifications than the standard FDG-Atlas-based quantification approach. Thus, it may improve diagnosis accuracy in clinical routine. However, further work is required to validate this method in other neurodegenerative diseases and with larger datasets.

POSTER N.º 36

Introduced by: Sánchez Martín, Cristina

Title:

SUBJECT-LEVEL DETECTION OF FOCAL NEURODEGENERATION USING SPATIOTEMPORAL CONNECTOMICS: TOWARDS ATROPHY CHARACTERIZATION IN PRECLINICAL ALZHEIMER'S DISEASE

Principal Investigator: Cristina Sánchez

Authors: Cristina Sánchez[1], Ibai Diez, PhD[2], Elisenda Bueichekú, PhD[3], Chan-Mi Kim, PhD[2], Michel J. Grothe,PhD[1], Pascual Sanchez-Juan, PhD, MD[1] and Jorge Sepulcre, MD, PhD[3],

Filiation: [1]Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Madrid, Spain. [2]Gordon Center for Medical Imaging, Massachusetts General Hospital, Boston, MA, USA. [3]Yale University, New Haven, CT, USA.

Abstract:
"Background: Brain atrophy is a normal part of healthy aging, but it is aggravated by several neurodegenerative diseases. Previous studies have described heterogeneity in individual neurodegeneration patterns, but the underlying brain mechanisms are currently not fully understood. From a graph theory-based framework, the estimation of subject-specific focal or multifocal brain atrophy in healthy aging and in the preclinical stage of different neurodegenerative diseases, such as Alzheimer's disease, will help to better understand individual atrophy networks and likely improve prediction of phenotypic heterogeneity in disease trajectories. Method: The study included 78 older cognitively normal participants from the Vallecas project, who underwent longitudinal T1 MRI scanning with 8 follow-up timepoints. Voxel-wise gray matter volumes were obtained, and the topology of atrophy of each subject was defined. After that, we selected gray matter values with 1% annual decrease as accelerated atrophy measure. A graph theory approach based on the structural similarity of all pairs of voxels with accelerated atrophy was applied to identify uni- or multifocal atrophy patterns. Results: We identified individualized atrophy phenotypes based on the convergent or divergent behavior of voxels with accelerated atrophy, which in turn is characterized by different graph morphologies. Conclusions: We present a novel analytical tool for characterizing individualized atrophy phenotypes in healthy subjects based on graph theory and structural similarity analyses. This method may help to describe the first structural events in preclinical AD and other neurodegenerative diseases and, therefore, could be crucial for predicting differences in disease phenotype and progression in single subjects.

POSTER N.º 37

Introduced by: Uceda-Heras, Alicia

Title:

SYSTEMATIC VARIATION OF MYELIN ACROSS AREAS OF THE HUMAN TEMPORAL CORTEX OBSERVED ON MRI

Principal Investigator: Alicia Uceda-Heras

Authors: Alicia Uceda-Heras, Francisco J López-González, Linda Zhang, Jesús Silva, Alberto Rábano

Filiation: Reina Sofia Alzheimer Center,CIEN Foundation, BT-CIEN, ISCIII, Madrid, Spain

Abstract:
“The expression of synaptic plasticity markers varies systematically across cortical areas in primates, being lower in limbic areas, of poor laminar elaboration, and higher in eulaminate areas, with six-well developed layers. Selective vulnerability of temporal mesocortical areas has been observed in Alzheimer’s disease (AD), suggesting that simpler laminar architecture may be associated with the expression of factors that render mesocortical neurons more vulnerable than in eulaminate areas. Here, we present a simple manual method to quantify the intracortical content of myelin (well-known inhibitor of synaptic plasticity) along the cortex of the temporal lobe. To this end, we used T1-MRI coronal slices of 16 individuals from the Fundación CIEN Brain Bank, which were normalized with MatLab-SPM function and loaded in Image J. Intracortical myelin content was quantified by a ROI for each cortical type at 4 coronal levels of the temporal lobe, obtaining the mean-grey value of myelin. Our data show increased myelin density in eulaminate areas compared to limbic areas. The content of myelin also increased across eulaminate areas of progressively better laminar elaboration. These findings suggest that limbic areas of the human temporal cortex are more plastic than eulaminate temporal areas. Therefore, higher synaptic plasticity of limbic temporal areas may be related to the selective vulnerability to AD. Prospects for this project include the validation of these results within an extensive sample and comparing this method with automated methods, like the Lesion Segmentation Toolbox (LST)-AI-deep-learning-ensemble. Moreover, these results will be compared with histological post-mortem analysis of intracortical myelin. “

POSTER N.º 38

Introduced by: López González, Francisco Javier

Title:

CHOLINERGIC WHITE MATTER PATHWAYS IN ALZHEIMER'S DISEASE, DEMENTIA WITH LEWY BODIES, AND OTHER NEURODEGENERATIVE DISEASES: A POST-MORTEM MRI STUDY

Principal Investigator: Francisco J. López-González

Authors: Francisco J. López-González1, Milan Nemy2, Cene Jerele2,3, Alberto Rábano1, María José López Martínez1, Michel J. Grothe1, Pascual Sánchez-Juan1 and Daniel Ferreira2

Filiation: 1Reina Sofia Alzheimer Center, CIEN Foundation, ISCIII, Madrid, Spain 2Division of Clinical Geriatrics, Center for Alzheimer Research, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet, Stockholm, Sweden 3University of Ljubljana, Faculty of Medicine, Ljubljana, Slovenia

Abstract:
"Background: We propose an imaging-pathologic validation study aimed at investigating cholinergic WM pathways using post-mortem MRI of autopsy-confirmed AD, Lewy body dementia (LBD), mixed pathology (AD+LBD), other neurodegenerative diseases across the frontotemporal lobar degeneration (FTLD) spectrum (OD) and cognitively unimpaired donors (CU). Method: We included 55 brain donors (21 AD, 14 AD+LBD, 8 LBD, 7 OD and 5 CU). All donors underwent post-mortem MRI in situ and a neuropathological examination. Mean diffusivity (MD) maps were estimated using the FSL software for each donor and for two cholinergic WM pathways of interest: cingulum and external capsule. Moreover, regional cholinergic WM signal abnormalities were visually scored on FLAIR images using the Cholinergic Pathways HyperIntensities Scale (CHIPS). Differences in MD, CHIPS and in age-adjusted values/scores (after excluding the CU group) between groups were analysed using the Mann-Whitney U-test. Result: AD donors were older than LBD ($p=0.01$) and than OD ($p=0.02$); and CU donors are significatively younger than all other groups ($p<0.02$). AD and AD+LBD showed higher MD values in cholinergic WM pathways when compared with OD (Cohen's $d\geq0.5$, $p=0.05$) and LBD (Cohen's $d\geq1.3$, $p<0.01$). Qualitatively similar findings were obtained after adjusting for age but at lower effect size and statistical significance Conclusion: We confirmed the degeneration of cholinergic WM pathways in neuropathologically confirmed dementia groups. This degeneration is more severe in the AD groups than in the LBD group, possibly due to differences in the degree of disease/dementia severity. Additionally, DTI-based indices of cholinergic pathway integrity strongly correlate with CHIPS-based visual assessment.

POSTER N.º 39

Introduced by: Sacchini, Simona

Title:

NEURODEGENERATIVE DISEASES: WHAT CAN BE LEARNED FROM TOOTHED WHALES?

Principal Investigator: Simona Sacchini

Authors: Simona Sacchini

Filiation: Universidad de Las Palmas de Gran Canaria (ULPGC). Las Palmas de Gran Canaria, Spain

Abstract:
“Different studies have demonstrated neurodegeneration in animals, as marine mammals. The suborders Mysticeti (baleen whales) and Odontoceti (toothed whales) make up the entire order Cetacea. As “”sentinels”” of the marine environment, toothed whales top-predators can serve as useful models of diseases for their human counterpart. Recent studies have revealed that some marine mammals, as toothed whales, exhibit neuropathological traits that recapitulate an Alzheimer’s-like pathology and might help in improving our comprehension of the neurodegenerative disorders (Sacchini et al. 2020; Vacher et al. 2023; Garamszegi et al. 2024). On the other hand, “”selective neuronal vulnerability”” describes the characteristic of neurodegenerative diseases where the pathology is limited to certain neurons. Conversely, neuromelanin has unique characteristics in humans and primates that are not seen in other animals. Neuromelanin was firstly observed in several species of the family Delphinidae (Sacchini et al. 2018) and transmission electron microscopy revealed the existence of melanin granules linked to lipid droplets and membranes, that closely resembled human neuromelanin, in two toothed whales (Sacchini et al. 2022a). Finally α-synuclein, ubiquitin, and laforin have been checked in toothed whales (Sacchini et al. 2022b), and α-synuclein/ubiquitin immunopositive round bodies were found in the neuropil of the mesencephalon. The advantages of transgenic rats are indisputable, but alternative natural, non-transgenic models may yield more pertinent data on the physiopathology of neurodegenerative diseases.
“

POSTER N.º 40

Introduced by: Ettcheto, Miren

Title:

B-CARYOPHYLLENE ENHANCES COGNITION AND REDUCES ALZHEIMER'S PATHOLOGY IN APPSWE/PS1DE9 MICE

Principal Investigator: Constanza Catalina Paz Rocha Jaures

Authors: Jaures CCPR1,2, Fernandes MJS1, Mourão RHV6, Guzman L2,3,4, Carrasco M2,3,4, Camins A2,3,4,5, Ettcheto M2,3,4,5

Filiation: 1. Department of Neurology and Neurosurgery, Discipline of Neuroscience, Federal University of São Paulo (UNIFESP), São Paulo, Brazil. 2. Department of Pharmacology, Toxicology, and Therapeutic Chemistry, Faculty of Pharmacy and Food Sciences, University of Barcelona (UB), Barcelona, Spain. 3. Network Center for Biomedical Research in Neurodegenerative Diseases (CIBERNED), Madrid, Spain. 4. Institute of Neuroscience, University of Barcelona, Barcelona, Spain. 5. Pere Virgili Health Institute (IISPV), Reus, Spain. 6. Laboratory of Experimental Biology and Bioprospecting - LabBBEx, Federal University of Western Pará (UFOPA), Santarém-Pará, Brazil.

Abstract:
"β-caryophyllene (BCP) is a terpene found in various plants and tree resin, known for its role in regulating inflammation and oxidative stress. As a phytocannabinoid, it is a potential drug for preventing neuroinflammatory and neurodegenerative diseases like Alzheimer's Disease (AD). AD is the most common form of dementia, characterized by beta-amyloid plaques, glial activation, neuronal death, and loss of synaptic function, leading to cognitive decline and memory loss. Therefore, this study aimed to assess whether BCP can inhibit key pathological mechanisms in a familial AD mouse model. For that, five-month-old female APPswe/PS1dE9 (APP) and wild type C57BL6/J (WT) mice were treated with BCP (48 mg/kg) or vehicle (VEH) intraperitoneally three times/week for four weeks. Cognitive function was evaluated using the Morris water maze (MWM) and novel object recognition test (NORT), while anxiety behavior was assessed with the open field (OF) test. Additionally, Aβ42 levels and dendritic spine density were measured. The results showed a significant improvement in learning and memory in APP mice treated with BCP compared to APP VEH. This cognitive improvement was associated with reduced dendritic spine loss in the hippocampus and decreased Aβ42 levels in the cortex. Furthermore, BCP treatment significantly reduced anxiety-like behavior in APP mice. In conclusion, BCP treatment enhanced cognition, reduced anxiety-like behavior, preserved dendritic spine density, and affected Aβ42 levels, demonstrating its potential neuroprotective benefits in AD. These findings suggest that BCP could help mitigate the progression of AD.

POSTER N.º 41

Introduced by: Sánchez, Juan

Title:

EXTRACELLULAR SPACE REMODELING CONTRIBUTIONS TO ADULT BRAIN REGENERATION.

Principal Investigator: Juan Andrés Sánchez

Authors: Sánchez JA1, Santos M1, Simões A1, Alves C1, Encinas JM2, Rhiner C1

Filiation: 1 Stem cell and Regeneration Laboratory, Champalimaud Foundation, Lisbon, Portugal. 2 Laboratory of Neural Stem Cells and Neurogenesis, Achucarro Basque Center for Neuroscience, Bizkaia, Spain.

Abstract:
The regenerative potential of the adult brain lies within the neural stem cells (NSCs), a group of dormant cells capable of generating new neurons and glial cells. The NSCs are embedded in dedicated environments that are composed of heterogeneous cells that cohesively remodel the extracellular space to promote the NSCs activation when it is needed. Poor activity in these niches has been associated with neurodegenerative disorders, including Parkinson and Alzheimer. However, how these specialized regions coordinate the emergence of new cells remain elusive and are the main focus of my research. Previously our group conducted a transcriptomic analysis to identify genes coding for extracellular proteins differentially expressed in the Drosophila brain upon injury. Interestingly we found that the gene CG14309, predicted to be a mammalian Heparanase orthologue, is activated at early time points and is necessary for proliferation upon acute injury in the Drosophila optic lobe. Whether CG14309 functions as a Heparane Sulfated Proteoglycan cleaving protein is still unknow and we hypothesize that it is important for extracellular matrix remodeling and promoting the activation of the quiescent NSCs. Simultaneously, we are generating a cell-cell interaction tracing system to identify and genetically modify the NSCs local environment. Overall, we are working to provide a detailed knowledge about cell interactions within the NSC niches by elucidating molecular and cellular mechanisms and ultimately a comprehensive framework for harnessing the latent regenerative capacity of the brain.

POSTER N.º 42

Introduced by: Serrano-Marín, Joan

Title:

NORMALISATION OF HUMAN TEAR METABOLOMICS DATA ALLOWING INTER-INDIVIDUAL COMPARISONS OF PATIENTS WITH NEURODEGENERATION

Principal Investigator: David Bernal-Casas

Authors: Joan Serrano-Marín1, Silvia Marín2,3,4, Alberto Iglesias1#, Jaume Lillo1,5*, Claudia Garrigós1, Toni Capó1, Irene Reyes-Resina5,6, Hanan Awad7, Marta Cascante2,3,4, Juan Sánchez-Navés8, Rafael Franco2,5,9, David Bernal-Casas10

Filiation: 1 Molecular Neurobiology Laboratory, Department of Biochemistry and Molecular Biomedicine, Universitat de Barcelona, Barcelona, Spain, 2 Department of Biochemistry and Molecular Biomedicine, Faculty of Biology, Universitat de Barcelona (UB), Barcelona, Spain, 3 Institute of Biomedicine of University of Barcelona (IBUB), University of Barcelona (UB), Barcelona, Spain, 4 CIBEREHD, Network Center for Hepatic and Digestive Diseases, Spanish National Health Institute Carlos III (ISCIII), Madrid, Spain. 5 CiberNed, Network Center for Neurodegenerative Diseases, Spanish National Health Institute Carlos III (ISCIII), Madrid, Spain. 6. Department of Biochemistry and Physiology, Faculty of Pharmacy and Food Sciences, Universitat de Barcelona (UB), Barcelona, Spain, 7. Department of Optometry, College of Applied Medical Sciences, Qassim University, Almulida, Qassim, Saudi Arabia. 8 Department of Ophtalmology, Oftalmedic and I.P.O. Institute of Ophthalmology, Palma de Mallorca, Spain, 9 School of Chemistry, Universitat de Barcelona, Barcelona, Spain.

Abstract:
To allow meaningful inter-individual comparisons, this study introduces a new method for analyzing human tear data, known for its high variability in composition. Metabolomic data were used to predict the concentrations of metabolites based on the concentration of a single concomitant metabolite, the individual's age, sex, and fasting time. Central to this approach is the concept of precision medicine, acknowledging that each patient will have a unique tear composition. By combining our method with Linear Discriminant Analysis (LDA), we were able to accurately determine the sex of individuals based on just one metabolomic parameter in tear. Similarly, this development could lead to diagnosis or detection of trends in patients with neurodegenerative diseases using a parameter consisting of the concentration of a predetermined metabolite. This advancement demonstrates the potential for using human tears for diagnostic purposes, as our method allows for the use of tear composition to identify significant differences, including those related to sex. Our findings indicate that this approach could support the use of tear analysis in medical diagnostics, facilitating the identification of various physiological and pathological conditions through tear composition.

POSTER N.º 43

Introduced by: Nascimento, Marta

Title:

ALCOHOL RELATED DEMENTIA OR ALCOHOL RELATED BRAIN DAMAGE?

Principal Investigator: Marta Nascimento

Authors: “Marta Nascimento Liliana Pereira”

Filiation: Neurology department of Unidade de Saúde Local de Almada Seixal

Abstract:
“INTRODUCTION: Alcohol is a major preventable burden for global health. The association between alcohol use disorders and cognitive impairment has long been recognized in epidemiological studies. However, the role of alcohol as an etiological mechanism for dementia itself remains controversial. In fact, alcohol related brain damage is an umbrella for other common causes of cognitive impairment related to alcohol use such as: traumatic head injury, vascular risk factors, hepatic encephalopathy, nutritional deficiencies and psychiatric disorders. METHODS: Nonsystematic review of literature RESULTS: The amount and duration of alcohol consumption enough to cause alcohol related dementia (ARD) is unclear. Some authors, however, stress the drinking pattern, pointing features as binge drinking duration and withdrawal periods. Two lines of research seem to explain ARD: “Glutamatergic excitability” and “Thiamine deficiency”. Both could explain the potential for neuroinflammation and oxidative stress caused by alcohol. In order to stimulate research and to minimize the subjective clinical judgment, some authors proposed a set of criteria for “Probable” and “Possible” diagnosis of ARD. Characteristics of ARD include - presence of other alcohol-related organ damage, reversibility or stability of cognitive deficits and neuroimaging features, symptoms of cerebellar dysfunction and sensory neuropathy. The following do not favor ARD diagnosis: prominent language impairment, focal neurological signs, neuroimaging evidence of cerebrovascular disease or focal brain pathology. Diagnosis cannot be made before 60 days of alcohol abstinence. CONCLUSIONS: In the rising scene of dementia prevalence, knowledge of alcohol contribution for brain lesion may stimulate the development of more efficacious therapeutic and prevention strategies.

POSTER N.º 44

Introduced by: Franco, Rafael

Title:

VITACTIONS. VITAMINS FOR THE BRAIN

Principal Investigator: Rafael Franco

Authors: Rafael Franco

Filiation: 1. CiberNed. Centro de Investigación en Red, Enfermedades Neurodegenerativas. Instituto de Salud Carlos III. Madrid. Spain. 2. Depto. Bioquimica y Biomedicina Molecular. Universitat de Barcelona. Barcelona. Spain

Abstract:
“A novel concept has been recently put forward in the mind/body interface (https://doi.org/10.37349/ent.2024.00074). The new concept has led to a new word: vitaction. Vitactions offer benefits to the brain and mind comparable to the advantages vitamins provide for the body’s overall health. The field of vitactions is as it was the vitamin field one century ago, i.e., without tools to make a complete classification. I propose to classify vitactions into five categories according to the behaviours necessary to maintain balanced brain functionality. A deficit of vitactions would contribute to the enormous prevalence in developed countries of diseases ranging from type 2 diabetes to neuropsychiatric diseases. The concept should help to identify which vitactions are deficient and to outline how they can be progressively implemented to improve the quality of life. The parallelism vitactions/vitamins also extends to overdosing; both hypervitaminosis and hypervitactinosis may be detrimental. This perspective article argues that vitactions should be considered at the practical and the scientific research levels, and that a balanced vitamin and vitaction supply is essential for a better life. In addition, reasons for proposing a synonym, “vitactin”, are given. The article: Vitactions: vitamins for the brain is available at https://www.explorationpub.com/uploads/Article/A100484/100484.pdf

FUNDACIÓN REINA SOFÍA

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INTERNATIONAL CONGRESS ON NEURODEGENERATIVE DISEASES

September 19-21, 2024

Champalimaud Foundation
Lisbon, Portugal

Image: Dancing Astrocytes

DISSEMINATION

PRESS RELEASES

NOTA DE PRENSA

España y Portugal se vuelven a unir para organizar una cumbre internacional sobre Alzheimer en Lisboa

- *Su Majestad la Reina Doña Sofía presidirá el acto oficial de celebración del Día Mundial del Alzheimer el 21 de septiembre, en el marco del Congreso Internacional sobre Enfermedades Neurodegenerativas (International Congress On Neurodegenerative Diseases Lisbon 2024).*

- *El Congreso se celebra del 19 al 21 de septiembre en la capital portuguesa, en el prestigioso Centre for the Unknown de la Fundación Champalimaud, y asistirán investigadores de referencia mundial en enfermedades neurodegenerativas.*

- *Cath Mummery, experta en ensayos clínicos para el Alzheimer de University College London; David Wolk, líder en investigación sobre cognición y envejecimiento y director del Penn Alzheimer's Disease Research Center de la Universidad de Pensilvania; y el Dr. Ed Lein, pionero en atlas cerebrales y genómica del Allen Institute for Brain Science de la Universidad de Washington, son algunos de los participantes del Congreso.*

Madrid, 20 septiembre de 2024. **Su Majestad la Reina Doña Sofía** presidirá el acto oficial de celebración del Día Mundial del Alzheimer, que se conmemora cada 21 de septiembre, acompañada por **Leonor Beleza,** presidenta de la Fundación Champalimaud de Lisboa y exministra de Salud de Portugal, en el marco del **Congreso Internacional sobre Enfermedades Neurodegenerativas,** que se celebra en la capital lusa.

El Congreso, un esfuerzo conjunto de España y Portugal, es organizado por la **Fundación Reina Sofía**, **CIEN** (Centro de Investigación en Enfermedades Neurológicas) y la **Fundación Champalimaud**, y reúne a investigadores de referencia mundial en una plataforma única para compartir los últimos avances en la investigación y tratamiento de patologías neurodegenerativas. La colaboración de ambos países se reafirma en un momento singular y esperanzador, marcado por la **medicina de precisión**, los avances en **la detección precoz con biomarcadores**, la **experimentación con inteligencia artificial para el diagnóstico** o el **horizonte de nuevas terapias farmacológicas**.

Expertos de renombre mundial, investigadores y profesionales de la salud se dan cita en un evento científico que busca no solo promover el intercambio de conocimientos y experiencias, sino también impulsar la investigación y sensibilizar a la sociedad, ámbitos en los que la Fundación Reina Sofía y S.M. la Reina Doña Sofía trabajan desde hace décadas.

Investigadores de primera línea internacional

El Congreso Internacional sobre Enfermedades Neurodegenerativas, consolidado como un evento clave para los profesionales de la investigación en este campo, cuenta con un programa estructurado en siete sesiones científicas y los aportes de más de treinta investigadores y representantes de asociaciones de varios países, en un esfuerzo por integrar la investigación y los cuidados asistenciales.

Entre los ponentes más destacados del congreso se encuentra **Cath Mummery**, neuróloga del University College London y directora del Dementia Research Centre de la institución británica, donde lidera innovadores ensayos clínicos para tratamientos que podrían modificar el curso de enfermedades neurodegenerativas como el Alzheimer; **David Wolk,** director del Penn Alzheimer's Disease Research Center de la Universidad de Pensilvana, reconocido por su trabajo pionero en la investigación sobre cognición y envejecimiento, o **Ed Lein**, investigador senior en el Allen Institute for Brain Science de la Universidad de Washington, quien aportará su experiencia en la creación de atlas cerebrales y en el uso de genómica para comprender mejor estas patologías.

El **programa científico** abarca aspectos de **detección temprana, variabilidad genética, neuroimagen, biomarcadores, medicina personalizada o terapias no farmacológicas**, y fue diseñado por expertos de la Fundación Champalimaud, como **John Krakauer**, director del Programa de Neurociencia Humana; **Joe Paton**, director de Champalimaud Research, y Marcelo Mendonça, neurólogo e investigador, así como por **Pascual Sánchez Juan**, director científico de CIEN.

Varios expertos de renombre de España y Portugal presentarán conferencias magistrales y participarán en debates, incluyendo a **Joaquim Ferreira**, director del Laboratorio de Farmacología Clínica y Terapéutica de la Universidad de Lisboa; **Alberto Lleó**, jefe de Neurología del Hospital de la Santa Creu i Sant Pau y director de su Unidad de Memoria; y **Agustín Ruiz**, director científico del ACE Alzheimer Center Barcelona.

El objetivo de la cumbre es la puesta en común de los avances que se están produciendo en dos áreas bien diferenciadas pero complementarias: el de **la investigación científica** y el de la **intervención terapéutica en el ámbito sociosanitario**. Cada uno de los campos cuenta con un programa de conferencias y ponencias, dentro de un **modelo traslacional** en el que la investigación y los cuidados se conciben de manera integrada.

Acerca de la Fundación Reina Sofía y del Centro Alzheimer Fundación Reina Sofía

Constituida en mayo de 1977 por S.M. la Reina Doña Sofía, la Fundación Reina Sofía es una entidad mixta de carácter benéfico y cultural, sin ánimo de lucro y de naturaleza permanente. En estos 47 años, ha gestionado y promovido más de doscientos cincuenta proyectos con decenas de entidades sociales, de contenido educativo, sanitario, medioambiental, así como de ayuda social y humanitaria, de los que se han beneficiado niños, mayores, inmigrantes, discapacitados, población desfavorecida y afectados por catástrofes naturales.

La investigación en enfermedades neurodegenerativas constituye desde 2002 una de las prioridades de trabajo de la Fundación Reina Sofía, del Centro Alzheimer Fundación Reina Sofía constituye uno de sus hitos centrales. El Centro constituye un complejo asistencial en el que se aborda la enfermedad de Alzheimer desde tres ángulos: investigación, formación y servicio asistencial al enfermo.

Su Unidad de investigación está cedida a CIEN para la realización de proyectos de investigación en enfermedades neurodegenerativas, proyectos que cuentan con la permanente colaboración y financiación de la Fundación Reina Sofía y el apoyo e implicación personales de S.M. la Reina Doña Sofía.

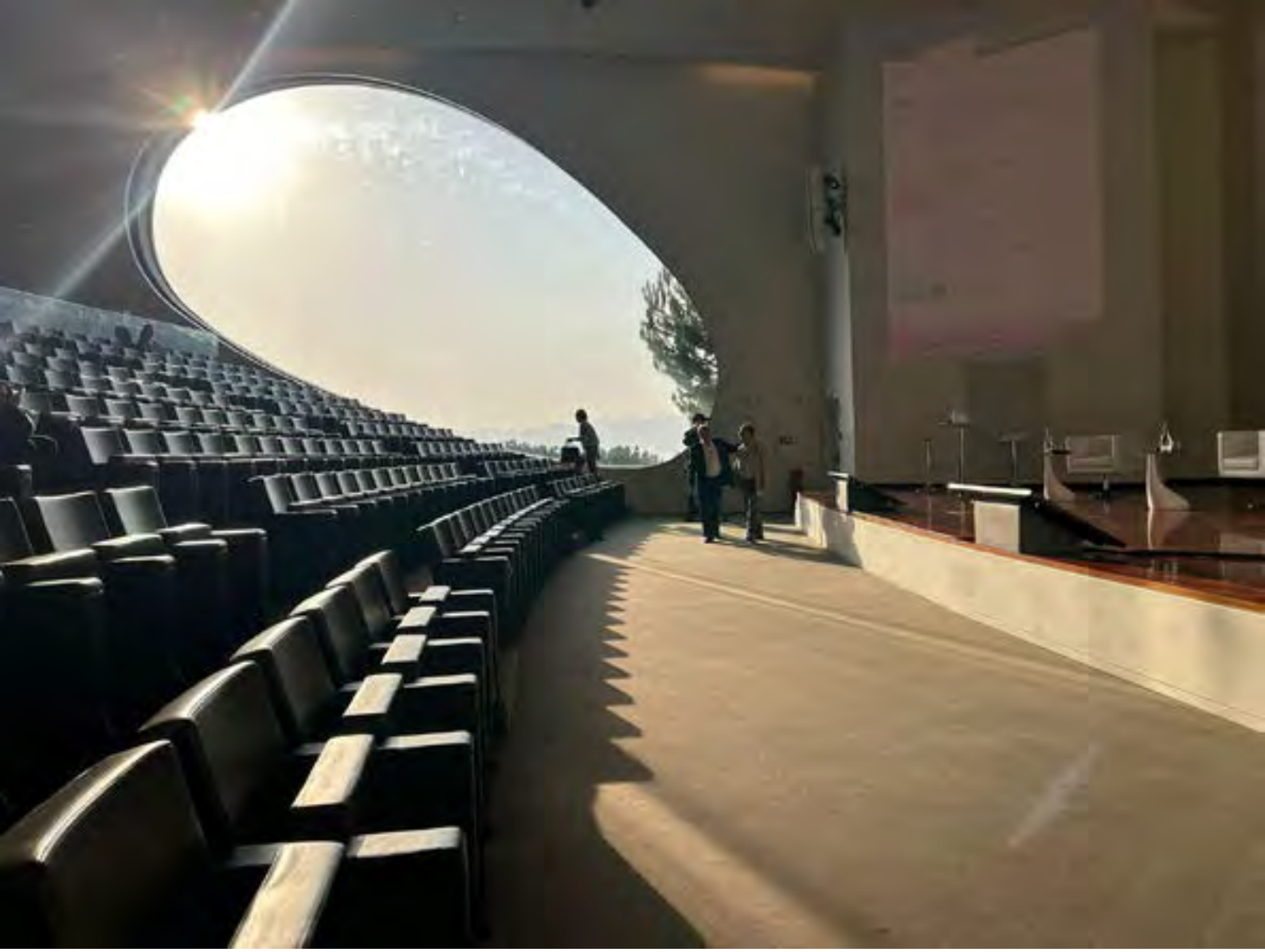
Sobre CIEN (Centro de Investigación de Enfermedades Neurológicas)

CIEN es el Centro de Investigación en Enfermedades Neurodegenerativas dependiente del Ministerio de Ciencia, Innovación y Universidades a través del Instituto de Salud Carlos III. Desde su sede en el Centro Alzheimer Fundación Reina Sofía CIEN apoya, promueve y coordina la investigación sobre enfermedades neurodegenerativas, especialmente Alzheimer pero también otras demencias y ELA (esclerosis lateral amiotrófica).

CIEN cuenta con un banco de tejidos único en España, por su organización y recursos, y es un referente en la difusión y sensibilización social sobre las enfermedades neurodegenerativas.

Sobre la Fundación Champalimaud

La Fundación Champalimaud, con sede en Lisboa, se dedica a la investigación de vanguardia en neurociencia y cáncer, combinando programas de investigación con servicios clínicos de excelencia. Su enfoque se basa en una metodología traslacional que conecta la investigación básica con la actividad clínica, buscando avances científicos significativos para mejorar la salud y el bienestar de las personas.



NOTA DE PRENSA

España y Portugal consolidan su colaboración en la lucha contra el Alzheimer en el Día Mundial de la enfermedad

- *Su Majestad la Reina Doña Sofía ha presidido el acto oficial de celebración del Día Mundial del Alzheimer, en el marco del Congreso Internacional sobre Enfermedades Neurodegenerativas Lisboa 2024.*
- *La iniciativa conjunta de España y Portugal ha estado marcada por la detección precoz con biomarcadores y neuroimagen, las nuevas terapias farmacológicas, los avances en la medicina de precisión o la experimentación con inteligencia artificial para el diagnóstico.*

Madrid, 21 septiembre de 2024. **Su Majestad la Reina Doña Sofía** ha presidido hoy en Lisboa el acto oficial de celebración del **Día Mundial del Alzheimer,** en el marco del Congreso Internacional sobre Enfermedades Neurodegenerativas que ha reunido en la capital portuguesa a destacados científicos, profesionales de la salud y asociaciones de pacientes.

El evento, celebrado en la **Fundación Champalimaud**, ha contado con la participación de **Ana Paula Martins**, ministra de Salud de Portugal; **Eva Ortega**, Secretaria General de Investigación; **Juan Fernández Trigo**, embajador de España en Portugal; y **Leonor Beleza**, presidenta de la Fundación Champalimaud.

El congreso ha reafirmado **la colaboración de los dos países** en la investigación y el tratamiento de patologías neurodegenerativas como el Alzheimer, una enfermedad que tiene mayor incidencia en sociedades envejecidas como las de España y Portugal, además de crecientes costes sociales y económicos.

Organizado por la **Fundación Reina Sofía**, **CIEN** (Centro de Investigación en Enfermedades Neurológicas, dependiente del Instituto de Salud Carlos III) y la **Fundación Champalimaud**, el congreso reunió a investigadores de todo el mundo, profesionales de la salud y asociaciones de pacientes como **CEAFA** (Confederación Española de Alzheimer y otras demencias), **Alzheimer Europe** o la **Associação Alzheimer Portugal**. Una combinación que responde a los objetivos de consolidar el diagnóstico precoz, los cuidados y el desarrollo de terapias farmacológicas y no farmacológicas, y sensibilizar a la sociedad.

Nuevas terapias y avances científicos, punto de inflexión en la lucha contra el Alzheimer

El congreso marca un momento esperanzador en el estudio y tratamiento del Alzheimer, con el **horizonte concreto de nuevas terapias farmacológicas** aprobadas por primera vez en décadas, así como importantes avances en la **medicina de precisión**, la **detección precoz con biomarcadores y neuroimagen** o la **experimentación con inteligencia artificial para el diagnóstico**.

Eva Ortega, Secretaria General de Investigación, subrayó en su intervención el trabajo de CIEN, de la colaboración internacional y redes de trabajo conjuntas de investigadores e investigadoras en las que España ocupa un lugar destacado, y "la importancia central de la educación y la concienciación social para la detección precoz de las enfermedades neurodegenerativas".

Marcelo Mendonça, neurólogo y investigador de la Fundación Champalimaud, destacó que «este congreso fue esencial para debatir las diversas cuestiones que rodean a las enfermedades neurodegenerativas como el Parkinson y el Alzheimer, centrándose en la importancia de un diagnóstico preciso y en las diversas terapias, farmacológicas o no, que pueden aplicarse para combatir estas enfermedades».

John Krakauer, director del programa de neurociencias humanas de la Fundación Champalimaud, afirmó que «en esta reunión con diversos socios internacionales, abordamos la importancia de debatir cuestiones centrales en materia de salud para buscar soluciones innovadoras en el ámbito de las enfermedades neurodegenerativas como el Parkinson y el Alzheimer».

Pascual Sánchez Juan, director científico de CIEN, incidió en que "Con técnicas cada vez más avanzadas y enfoques combinados ya es posible detectar el Alzheimer de manera temprana, especialmente con biomarcadores en sangre. Si a esto le sumamos las nuevas terapias farmacológicas y los avances en medicina de precisión y en genética, podemos afirmar que estamos en un momento de cambio que no veíamos hace años."

Asimismo, **Mª Ángeles Pérez**, gerente de CIEN, destacó que "Si bien es positivo que la esperanza de vida en países como España o Portugal supere los 80 años y estamos viendo avances importantes en la investigación en Alzheimer, ello nos obliga a redoblar esfuerzos en investigación y concienciación social, a continuar apoyando la investigación con medios y recursos, tareas que no serían posibles sin el apoyo de la Fundación Reina Sofía y el compromiso personal de Su Majestad la Reina Doña Sofía."

Con la implicación activa de instituciones científicas, gobiernos y organizaciones de pacientes, el congreso ha servido como una plataforma para **reforzar la cooperación internacional y desarrollar nuevas estrategias** de abordaje del Alzheimer y otras enfermedades neurodegenerativas en el contexto del fenómeno global, y particularmente europeo, del envejecimiento poblacional.

Acerca de la Fundación Reina Sofía y del Centro Alzheimer Fundación Reina Sofía

Constituida en mayo de 1977 por S.M. la Reina Doña Sofía, la Fundación Reina Sofía es una entidad mixta de carácter benéfico y cultural, sin ánimo de lucro y de naturaleza permanente. En estos 47 años, ha gestionado y promovido más de doscientos cincuenta proyectos con decenas de entidades sociales, de contenido educativo, sanitario, medioambiental, así como de ayuda social y humanitaria, de los que se han beneficiado niños, mayores, inmigrantes, discapacitados, población desfavorecida y afectados por catástrofes naturales.

La investigación en enfermedades neurodegenerativas constituye desde 2002 una de las prioridades de trabajo de la Fundación Reina Sofía, de la el Centro Alzheimer Fundación Reina Sofía constituye uno de sus hitos centrales. El Centro constituye un complejo asistencial en el que se aborda la enfermedad de Alzheimer desde tres ángulos: investigación, formación y servicio asistencial al enfermo.

Su Unidad de investigación está cedida a CIEN para la realización de proyectos de investigación en enfermedades neurodegenerativas, proyectos que cuentan con la permanente colaboración y financiación de la Fundación Reina Sofía y el apoyo e implicación personales de S.M. la Reina Doña Sofía.

Sobre CIEN (Centro de Investigación de Enfermedades Neurológicas)

CIEN es el Centro de Investigación en Enfermedades Neurodegenerativas dependiente del Ministerio de Ciencia, Innovación y Universidades a través del Instituto de Salud Carlos III. Desde su sede en el Centro Alzheimer Fundación Reina Sofía CIEN apoya, promueve y coordina la investigación sobre enfermedades neurodegenerativas, especialmente Alzheimer pero también otras demencias y ELA (esclerosis lateral amiotrófica).

CIEN cuenta con un banco de tejidos único en España, por su organización y recursos, y es un referente en la difusión y sensibilización social sobre las enfermedades neurodegenerativas.

Sobre la Fundación Champalimaud

La Fundación Champalimaud, con sede en Lisboa, se dedica a la investigación de vanguardia en neurociencia y cáncer, combinando programas de investigación con servicios clínicos de excelencia. Su enfoque se basa en una metodología traslacional que conecta la investigación básica con la actividad clínica, buscando avances científicos significativos para mejorar la salud y el bienestar de las personas.

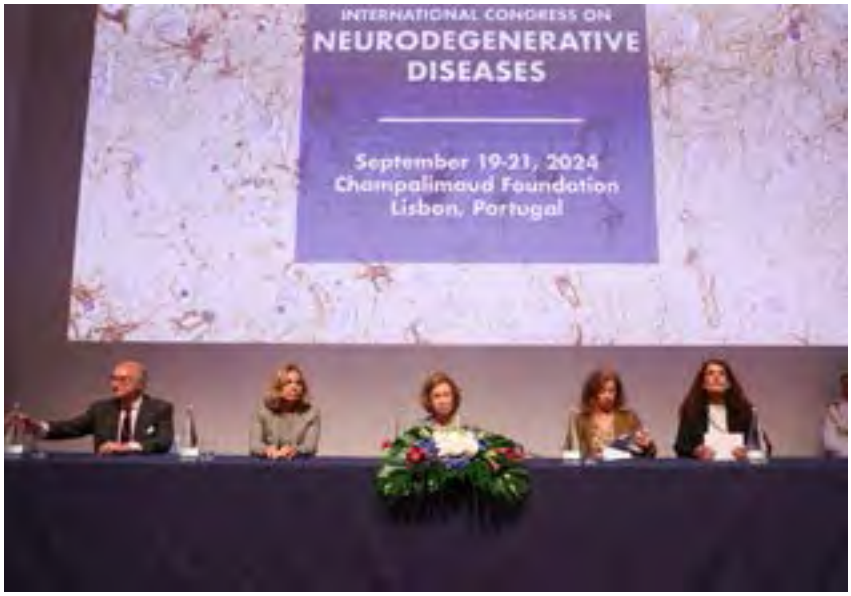


Foto 1 - De izquierda a derecha: Juan Fernández Trigo, embajador de España en Portugal; Ana Paula Martins, ministra de Salud de Portugal; S.M. la Reina Doña Sofía; Leonor Beleza, presidenta de la Fundaçao Champalimaud; Eva Ortega, Secretaria General de Investigación.

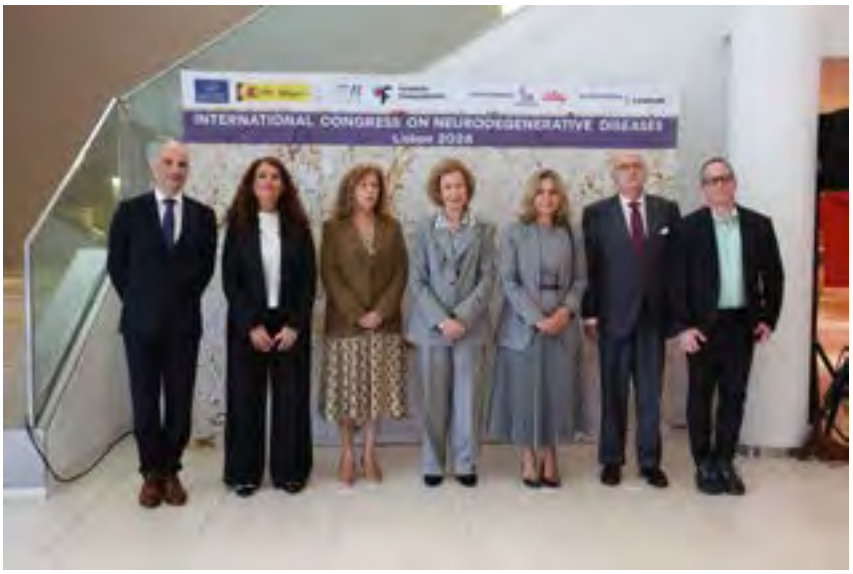


Foto 2 - Foto de familia. De izquiera a derecha: Pascual Sánchez Juan, director científico de CIEN; Eva Ortega, Secretaria General de Investigación; Leonor Beleza, presidenta de la Fundaçao Champalimaud; S.M. la Reina Doña Sofía; Ana Paula Martins, ministra de Salud de Portugal; Juan Fernández Trigo, embajador de España en Portugal; John Krakauer, director del programa de Neurociencia Humana de la Fundaçao Champalimaud.

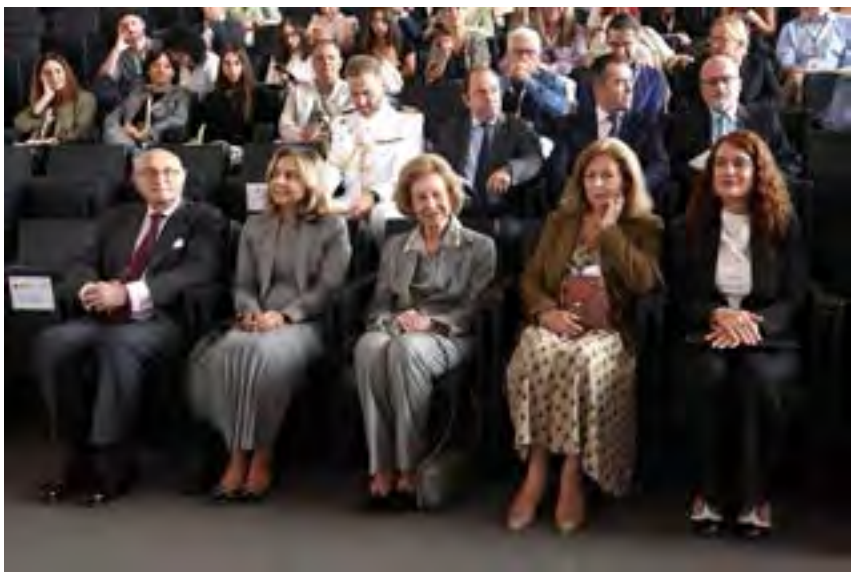


Foto 3 - Mesa presidencial del acto Día Mundial del Alzheimer

PRESS CLIPPING

International Congress on Neurodegenerative Diseases Lisbon 2024

Clipping de prensa

Septiembre de 2024

SUMARIO

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20/09/2024

Informativos Telecinco, Especial Alzheimer (20/09/24)

Informativos Telecinco, Especial Alzheimer (20/09/24)

20/09/2024 22:36h.

Informativos Telecinco, Especial Alzheimer, de la mano de Carlos Franganillo Informativos Telecinco

Informativos Telecinco ha emitido un especial en la edición noche de este viernes sobre el alzhéimer; este sábado se celebra el Día Mundial

Carlos Franganillo ha visitado el Centro Alzheimer Fundación Reina Sofía (CAFRS) para entrevistar al doctor Pascual Sánchez-Juan

Montse y el miedo del Alzhéimer: "Temo mucho el día que no me conozca, el día que piense que soy una extraña"

Día Mundial del Alzheimer

Este sábado, 21 de septiembre, se celebra el Día Mundial del Alzheimer . Con motivo de esta fecha, Informativos Telecinco ha emitido un especial en la edición noche de este viernes, en el que se puede ver cómo se aborda esta enfermedad incurable que afecta a 800.000 españoles y qué esperanzas hay. El alzhéimer es cada vez más visible según se extiende la esperanza de vida.

Visita al Centro Alzheimer Fundación Reina Sofía

Carlos Franganillo se ha desplazado hasta un sitio único, una residencia de enfermos de alzhéimer, pero también un centro puntero de investigación, donde se hace un estudio de la enfermedad neurodegenerativa siguiendo, día a día, la evolución de los pacientes. Hablamos del Centro Alzheimer Fundación Reina Sofía (CAFRS). El director de Informativos Telecinco ha entrevistado allí al doctor Pascual Sánchez-Juan , director científico del Centro de Investigación de Enfermedades Neurológicas (CIEN).

Las familias que cuidan de sus seres queridos

Asimismo, el equipo de Informativos también ha estado con familiares que cuidan de sus seres queridos . Entre otros, Montse , una mujer con una mirada fuerte, pero triste . "Lo que más temo es el día en el que (mi marido) no me reconozca y se ponga agresivo porque crea que soy una extraña voy a hacerle daño".

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Temas

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20/09/2024

El doctor Pascual Sánchez-Juan, desde el Centro Alzheimer Fundación Reina Sofía: Es el momento de mayor esperanza

Pacientes, familiares y doctores de 26 países completan el Camino de Santiago para abordar la demencia

Pascual Sánchez-Juan , director científico de la Fundación CIEN: " La visión del alzhéimer ha sido muy lineal"

Muchos ciudadanos se preguntan qué es lo que distingue al alzhéimer de otras enfermedades neurodegenerativas. " Clásicamente era un cuadro de demencia , donde normalmente se empieza por la afectación de la memoria, pero luego afecta a otras áreas cognitivas. Y lo que vemos en el cerebro de esos pacientes, cuando se estudia, son dos proteínas, fundamentalmente, depositadas fuera de las neuronas y dentro de las neuronas. Esto era cómo se concebía la enfermedad hasta hace no mucho", detalla el doctor Sánchez-Juan.

" La visión del alzhéimer ha sido muy lineal . 'Esta proteína se acumula, esto altera a esta proteína y esto produce la enfermedad'. Ahora sabemos que, probablemente, es mucho más complejo que eso. Es todo un reto entender cómo funciona el cerebro. Es uno de los problemas que tenemos para avanzar en el tratamiento de estas enfermedades", agrega el director científico de la Fundación CIEN.

Es todo un reto entender cómo funciona el cerebro

Un dato importante que conocer es que hay diferencias que se pueden apreciar a la vista entre un cerebro sano y un cerebro que está sufriendo alzhéimer . "La parte más media, que sería el hipocampo , eso es la diana, la zona cero de la enfermedad del Alzheimer, donde vamos a ver primero los cambios en forma de atrofia en toda una estructura, y es lo que se relaciona con la memoria , por eso en los pacientes, normalmente, los primeros síntomas son que no recuerdan lo que han desayunado o lo que hicieron ayer. Porque son incapaces de fijar nueva información", comenta el experto, que ha analizado una radiografía de un cerebro con Carlos Franganillo. "Todo lo que se ve en negro es el líquido cefalorraquídeo, es vacío. Se ve en negro porque se ha atrofiado y, por lo tanto, esto está ocupado por el líquido".

El Banco de Cerebros, de la Fundación CIEN, "la joya" del Centro Alzheimer Fundación Reina Sofía

El doctor Sánchez-Juan asegura que "la joya del centro es el Banco de Cerebros ", el cual ayuda de forma crucial a la investigación del alzhéimer: "Se nutre, por un lado, de los pacientes de las residencias -diríamos que es el programa interno-, y luego también de donantes externos".

"Para procesar un cerebro , normalmente, lo que hacemos es separarlo en dos mitades , una vez que se ha extraído el órgano. Una se va a congelar para estudios, por ejemplo, bioquímicos y otra se va a fijar en formol. Las escaneamos y, una vez están digitalizadas, podemos cuantificar o buscar patrones", precisa el doctor.

Para procesar un cerebro, normalmente, lo que hacemos es separarlo en dos mitades

Otra de las cuestiones tratadas por el doctor es la conducta de los pacientes con alzhéimer , cómo es su evolución. ¿Cómo se pasa de un estado de ánimo inicial de miedo y de incertidumbre a después perder la noción de la realidad? "Lo primero que solemos ver, y a veces se asocia con los problemas de memoria, es la apatía . A eso se puede añadir o no un componente depresivo cuando uno es consciente de los síntomas. Y, conforme avanza la enfermedad, podemos ver alteraciones psiquiátricas más severas, como los delirios o alucinaciones. Puede haber agresividad incluso. Va generando que la familia realmente tenga que hacer frente a una situación muy dura ",

indica el experto.

El objetivo actual: "De momento, lo razonable es intentar detener o ralentizar el curso de la enfermedad"

La ciencia comienza a tener herramientas para hacer frente a la enfermedad. Pero ¿tiene suficientes para predecir que una persona que no tiene ningún síntoma puede desarrollar la enfermedad 10 o 20 años después ?

"Los fármacos que modifiquen el curso de la enfermedad van a ser mucho más activos en cuanto los apliquemos . Por lo tanto, sería ideal en estados donde la enfermedad no da la cara. Ahora tenemos herramientas que son mucho más sensibles y nos pueden ayudar a detectar, no solo componentes del alzhéimer, sino otros componentes que se asocian al alzhéimer y que determinan el pronóstico", explica al respecto el doctor Sánchez-Juan.

Ahora tenemos herramientas que son mucho más sensibles

" De momento , lo razonable es intentar detener o ralentizar el curso de la enfermedad . Cronificar la enfermedad, como se ha hecho con el cáncer. Y en este sentido hay algunas buenas noticias. En Estados Unidos han aprobado fármacos cuyos efectos son, de momento, modestos, pero consistentes", agrega el experto, que considera que el planeta vive un momento de gran prosperidad dentro de este campo: "Creo que es el momento de mayor esperanza . Esto, por desgracia, aún no ha llegado a los pacientes. Están intentando atajar distintas partes del problema y que, probablemente, en el futuro, de forma combinada, nos ayuden realmente a avanzar en esa intención de retrasar el curso de la enfermedad".

Temas

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20/09/2024

Montse: Yo temo mucho el día que no me conozca, el día que piense que soy una extraña y se ponga agresivo

Redacción digital Informativos Telecinco

20/09/2024 22:13h.

La tensión y la tristeza en los ojos de los cuidadores: los familiares que conviven con un enfermo de Alzheimer

El doctor Pascual Sánchez-Juan, desde el Centro Alzheimer Fundación Reina Sofía: "Es el momento de mayor esperanza"

Montse mira con ojos fuertes pero tristes. "Lo que mas temo es el día que no me conozca, que no me reconozca y se ponga agresivo porque crea que soy una extraña voy a hacerle daño". Es el gran miedo de las familias que conviven con familiares que sufren Alzheimer . Ese momento en el que el ser querido no sea él, sino un desconocido que no lo es, por eso tratan de disfrutar del presente. "Algún síntomas de ansiedad se manifiestan".

Álvaro reconoce que algunas veces la jefa se enfada porque no llega a las cosas. Es consciente del futuro. Y del presente. La medicación la toma Alvaro en parches y como él dice a veces necesita ayuda porque son en la espalda. Montse sabe que habrá que tomar decisiones a futuro. Y que para ello tendrá que contar con sus hijos. Sabe que será un momento duro para todos. "Difícil de digerir para ellos". Álvaro es disciplinado.

MÁS

Medio: cadenaser.com
Publicado: 24/09/2024 **Audiencia**: 13.220.896
Edición: Digital **Valor**: 16.393€
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URL: cadenaser.com/nacional/2024/09/25/macaco-presenta-la-me...

24/09/2024

Macaco presenta 'La memoria del corazón', una canción dedicada a su madre con Alzheimer

El artista catalán Dani Carbonell, artísticamente conocido como Macaco, emociona al público con la canción más personal de su carrera en homenaje a su madre, que padece esta enfermedad Las enfermedades cobran un matiz totalmente diferente cuando las vivimos en primera persona o tocan a un familiar cercano. El cantante Dani Carbonell, más conocido como Macaco, recibió hace un tiempo la dura noticia de que su madre padecía Alzheimer. A raíz de la convivencia con ella, y gracias a intentar preservar su memoria a través de la música y los sentimientos, ha nacido 'La memoria del corazón' una canción que ve la luz hoy, 25 de septiembre de 2024, en homenaje a su madre y a todas las personas que combaten esta enfermedad. La música como gran conexión "Estoy muy feliz de poner ese homenaje a mi madre y compartir mi experiencia con todas esas personas que tengan familiares con esta enfermedad", comenta emocionado Macaco. El cantante explica que su madre venía de una familia muy humilde con mucho gusto por el arte. Todos los hermanos cantaban, pero su madre destacaba desde muy pequeñita. Ella es Teresa María, la voz de las canciones de Mary Poppins en España, y en otras películas reconocidas como My Fair Lady . Macaco cuenta que los primeros síntomas leves de la enfermedad empezaron hace aproximadamente 6 años y que se fueron acentuando con el pasar de los meses. El carácter de su madre se tornaba cada vez más complicado y la comunicación con ella se hacía más difícil. En su afán por acompañarla y tratar de ayudar lo máximo posible descubrió que la música tenía un poder sanador contra el Alzheimer que podía servir para conectar emocionalmente con ella. "Uno de los días que salí a pasear con ella me puse a tararear la melodía de Mery Poppins. Entonces, de repente, mi madre empezó a cantarla y a conectar algunos recuerdos. Yo me emocioné y empecé a llorar. Surgió un flujo de conexiones a través de las canciones que no frenaba", cuenta Macaco en La Ventana. De esta anécdota va surgiendo la idea de crear una canción para su madre. Fue un proceso de casi un año en el que, mientras tanto, iba grabando notas de voz cantando con su madre para el grupo de WhatsApp de la familia. Escribió una poesía que luego se convirtió en canción: "Apareció la idea de coger una parte de alguna canción que hubiera cantado mi madre, y me decidí por la de la película My Fair Lady . Con las herramientas que existen ahora, fui capaz de extraer la voz limpia y pudimos incorporarla a los instrumentos de mi canción. Cuando le puse la canción a mi madre se quedó flipando". Todos los derechos de 'La memoria del corazón' de Macaco han sido cedidos para la fundación Alzheimer España, con el objetivo de que las investigaciones y las ayudas prosperen. "Creo que mi madre no se olvidará de nosotros hasta que se vaya. Tiene una conexión muy fuerte con sus hijos", afirma Dani Carbonell, Macaco. Los avances contra el Alzheimer, cada vez más cerca Pascual Sánchez Juan es neurólogo y director científico de la Fundación CIEN, Centro de Investigación de Enfermedades Neurodegenerativas y centro de Alzheimer Fundación Reina Sofía, y ha venido también a La Ventana para aportar el punto de vista científico, siempre necesario cuando se habla sobre Alzheimer. "Me gustaría lanzar un mensaje de esperanza. Aunque el Alzheimer es una enfermedad terrible y sin paliativos, los avances científicos nos hacen albergar esperanzas de que, en un futuro a medio plazo, seremos capaces de ofrecer más cosas a los pacientes", anuncia Sánchez Juan. Hoy en El País se publicaba una noticia que anunciaba una "rebelión científica" para que Europa apruebe la administración de un fármaco que otros países ya dispensan y que, en casos de detección precoz de Alzheimer, puede frenar hasta un 30% la progresión de la enfermedad. El neurólogo explica que este fármaco nuevo actúa contra una proteína clave en la creación de la enfermedad, eliminándola, y lo califica como "un avance robusto". Sin embargo, sigue existiendo una gran brecha en Europa con el Alzheimer. "Los sistemas sanitarios no ven la detección precoz del Alzheimer como una prioridad. Hay muchas herramientas para esto que no se utilizan", afirma Sánchez Juan. El neurólogo cuenta el caso concreto de un paciente que él mismo atendió: "Ayer diagnostiqué a un paciente de Alzheimer y decidió ir a tratarse en Estados Unidos. Él puede permitírselo, pero habrá muchísima gente que no pueda y esto va a seguir pasando. Habrá ciudadanos que tengan acceso

a unos fármacos y otros que no". Para tratar de solucionar este problema, desde la fundación CIEN con Pascual Sánchez Juan se ha puesto en marcha un gran proyecto multicéntrico para reclutar a voluntarios que les aporten diagnósticos precoces y precisos de Alzheimer para investigar y allanar el terreno sobre el que aterrizarán los fármacos y tratamientos que llegarán en el futuro para combatir la enfermedad. "Este estudio pretende, con un análisis de sangre, hacer un diagnóstico de la enfermedad mucho más preciso, precoz y frecuentemente", afirma el neurólogo. Prevenir o retrasar los efectos del Alzheimer es posible. Sánchez Juan recomienda cualquier tipo de estimulación cognitiva, cualquier forma de entrenar el cerebro. Estudiar música, idiomas, matemáticas, hacer crucigramas, resolver adivinanzas... Todas estas actividades ayudan a muscular el cerebro y lo hace más resiliente al deterioro. "Yo siempre recomiendo a los pacientes que estas actividades las disfruten, que no les torturen. Si uno disfruta haciendo sopas de letras, adelante, si no mejor que haga otra cosa, pero siempre disfrutando de la actividad", recomienda Sánchez Juan.

Medio: Agencia de noticias EUROPA PRESS
Publicado: 20/09/2024
Edición: Servicio de Salud
Sección: Sociedad Sindicatos, Asociaciones

Agencia de noticias EUROPA PRESS
20/09/2024

La Reina Sofía participará en una cumbre internacional sobre Alzheimer organizada por España y Portugal

MADRID, 20 (EUROPA PRESS)

El Centro de Investigación en Enfermedades Neurológicas (CIEN) ha informado de que la Reina Doña Sofía presidirá el acto oficial de celebración del Día Mundial del Alzheimer, que se conmemora cada 21 de septiembre, en el marco del Congreso Internacional sobre Enfermedades Neurodegenerativas, que se celebra en Lisboa.

El Congreso está organizado por la Fundación Reina Sofía, CIEN y la Fundación Champalimaud, y reúne a investigadores de referencia mundial en una plataforma para compartir los últimos avances en la investigación y tratamiento de patologías neurodegenerativas. En el acto, la Reina Doña Sofía estará acompañada por Leonor Beleza, presidenta de la Fundación Champalimaud de Lisboa y exministra de Salud de Portugal.

Según los organizadores, se trata de "un esfuerzo conjunto de España y Portugal", que reafirma en un momento "singular y esperanzador, marcado por la medicina de precisión", los avances en la detección precoz con biomarcadores, la experimentación con inteligencia artificial para el diagnóstico o el horizonte de nuevas terapias farmacológicas.

El Congreso Internacional sobre Enfermedades Neurodegenerativas cuenta con un programa estructurado en siete sesiones científicas y los aportes de más de treinta investigadores y representantes de asociaciones de varios países, en un esfuerzo por integrar la investigación y los cuidados asistenciales.

Entre los ponentes más destacados del congreso se encuentra Cath Mummery, neuróloga del University College London y directora del Dementia Research Centre de la institución británica, donde lidera innovadores ensayos clínicos para tratamientos que podrían modificar el curso de enfermedades neurodegenerativas como el Alzheimer; David Wolk, director del Penn Alzheimer's Disease Research Center de la Universidad de Pensilvana, reconocido por su trabajo pionero en la investigación sobre cognición y envejecimiento, y Ed Lein, investigador senior en el Allen Institute for Brain Science de la Universidad de Washington, quien aportará su experiencia en la creación de atlas cerebrales y en el uso de genómica para comprender mejor estas patologías.

Por su parte, Pascual Sánchez Juan, director científico de CIEN; Joaquim Ferreira, director del Laboratorio de Farmacología Clínica y Terapéutica de la Universidad de Lisboa; Alberto Lleó, director del Servicio de Neurología del Hospital de la Santa Creu i Sant Pau y responsable de su Unidad de Memoria, y Agustín Ruiz, director científico del ACE Alzheimer Center Barcelona, son también algunos de los expertos españoles y portugueses que participarán con ponencias y conferencias magistrales.

El objetivo de la cumbre es la puesta en común de los avances que se están produciendo en dos áreas bien diferenciadas pero complementarias: el de la investigación científica y el de la intervención terapéutica en el ámbito sociosanitario. Cada uno de los campos cuenta con un programa de conferencias y ponencias, dentro de un modelo traslacional en el que la investigación y los cuidados se conciben de manera integrada.

Medio: elespanol.com
Publicado: 21/09/2024
Edición: Digital
Sección:
URL: elespanol.com/ciencia/salud/20240921/sanchez-juan-neuro...
Audiencia: 29.676.742
Valor: 36.799€

21/09/2024

Sánchez-Juan, neurólogo: España no está a la altura del reto mayúsculo que supone el alzhéimer

"Diagnosticar el alzhéimer antes de que aparezcan los síntomas no se justifica de ningún modo" / "Un indicio de alzhéimer puede ser no recordar qué has desayunado" / "Deberíamos tener un pacto con el alzhéimer al igual que con la ELA" / "El alzhéimer es como la 'peli' del meteorito: no se quiere mirar, pero viene" Dice Pascual Sánchez-Juan (Elche, 1973) que no se especializó en demencias porque tuviese algún caso en su familia que le marcara . Lo hizo"por vocación", porque era - y sigue siéndolo - " un apasionado del cerebro ". Aunque ha cambiado su forma de mirarlo. Antes, lo observaba a través del paciente, como neurólogo en el hospital. Ahora, trata de descifrarlo 'desde dentro', con la investigación.

LA RAZON

Medio: La Razón
Publicado: 23/09/2024
Edición: Madrid
Sección: REGIONAL Madrid

Audiencia: 78.000
Difusión: 26.259 Ejemplares
Valor: 12.409€

EFE

El lunes pasado visitó el Parque Natural de la Montaña Palentina para conocer el estado de conservación del gato montés

Su interés trasciende el deber regio

El lunes pasado visitó la Montaña Palentina para conocer las investigaciones sobre la conservación del gato montés, cuya población han disminuido o incluso desaparecido en varias partes de Europa. Además de observar la fauna silvestre en su hábitat, recorrió la comarca para conocer las características del paisaje, la ecología del gato montés y sus problemas. En este contexto, la Reina habla con todo el mundo y deja patente que su implicación trasciende el deber institucional.

La madre de Felipe VI despliega su faceta más humana en las causas vinculadas con la salud y el planeta

Las otras luchas de la **Reina Sofía** que ocupan su agenda diaria

Marian Benito. MADRID

A la Reina Sofía su compromiso con la vida le elimina cualquier arruga de la mente y del ánimo. A punto de cumplir 86 años, no se dobla más que para trabajar duro recogiendo plásticos y residuos en entornos naturales, como viene haciendo desde hace años. Es una tarea más en una agenda apabullante, siempre a la medida de sus deberes, inquietudes o desasosiegos, que son muchos.

Podríamos pensar que posee el don de la ubicuidad, pero lo que ocurre es que está allí donde se la necesita. En agosto no faltó a su cita en París para apoyar al Comité Olímpico en representación de la Familia Real. Y el viernes y sábado, 20 y 21 de septiembre, viajó a Lisboa para participar en el Congreso Internacional de Enfermedades Neurodegenerativas, un encuentro científico que reunió a expertos de todo el mundo para compartir los últimos avances en la investigación y tratamiento de estos males que tanto aterran.

Una vez terminadas las conferencias, la Reina recorrió una exposición científica junto a Pascual Sánchez, investigador de la Fundación del Centro de Investigación de Enfermedades Neurológicas (CIEN), quien respondió a su interés por la medicina de precisión, la detección precoz, la experimentación con inteligencia artificial y las nuevas terapias farmacológicas que cambiarán el rumbo de las enfermedades neurodegenerativas. Incansable, ese mismo día por la tarde asistió al concierto de la cantante de fados Katia Guerreiro. Doña Sofía es una gran melómana, por lo que la intérprete le brindó la ocasión de apreciar la riqueza lírica de este género.

Doña Sofía, en el Congreso de Enfermedades Neurodegenerativas

Un día después, ejerció como madrina en el acto oficial que se celebró en este mismo Congreso con motivo del Día Mundial del Alzheimer. Fue la encargada de presidir el evento y, aunque no pronunció ningún discurso, introdujo a los ponentes. Entre ellos, Ana Paula Martins, ministra portuguesa de Sanidad, y Leonor Beleza, presidenta de la Fundación lusa Champalimaud, organizadora del encuentro junto a la Fundación Reina Sofía y CIEN. Los participantes insistieron en la importancia de la prevención y una mayor investigación.

Sin diamantes y con las tiaras esperando en el joyero real, Doña Sofía alivia su timidez en este tipo de tareas que le permiten ser espontánea y mostrarse curiosa y sensible con los colectivos más vulnerables, como la infancia y las personas afectadas por una enfermedad neurodegenerativa. En ese querer saber y participar, demuestra firmeza en su propósito.

En sus eventos solidarios se muestra espontánea y curiosa

Una de sus preocupaciones es la enfermedad neurodegenerativa y todos sus trastornos, desde el alzhéimer o párkinson a la esclerosis múltiple. Como presidenta de la Fundación Reina Sofía, apoya aquellas iniciativas que tienen por objeto la investigación y la mejora de vida de los pacientes. Este contrato personal consigo misma la llevó el pasado mes de abril a Cracovia (Polonia) para presidir la sesión inaugural de la 36th Global Conference of Alzheimer's Disease International. Acababa de recibir el alta después de su ingreso en el hospital Ruber Internacional de Madrid, debido a un problema del tracto urinario que generó preocupación. La madre de Felipe VI mostró que conserva una extraordinaria salud de hierro y, una vez más, antepuso su deber a cualquier molestia.

Amante de la naturaleza

No son las únicas causas en las que está involucrada. Ama la naturaleza y le preocupa especialmente el cuidado del planeta, lo que le lleva a remangarse cada temporada para aportar su granito de arena en la recogida de basuras en entornos naturales, demostrando la misma resistencia en su compromiso ecológico.

Su agenda no le impide ocuparse de su familia y junto a ella es habitual que aparezca su compañera de vida, Irene de Grecia, de 82 años, si bien en los últimos meses su salud se ha visto deteriorada, como se desprende de su imagen en silla de ruedas en las reuniones y actos religiosos familiares. La próxima cita real será en Atenas, con motivo de la boda de su sobrina la princesa Teodora, hija de Ana María de Grecia y el fallecido rey Constantino. Volveremos a ver la Reina Sofía, esta vez con zafiros y diamantes, y la misma sensibilidad a flor de piel.

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21/09/2024

Fado, arte y ciencia: el emotivo viaje de la Reina Sofía a Lisboa

La madre de Felipe VI mantiene su firme implicación en la lucha contra las enfermedades neurodegenerativas La rentrée de Doña Sofía , después de las vacaciones de verano, no ha podido ser más emotiva. Su viaje a Lisboa este fin de semana confirma su firme compromiso con la institución y con la sociedad . La Reina participa desde ayer en el Congreso Internacional de Enfermedades Neurodegenerativas, un evento organizado por la Fundación Reina Sofía, la Fundación CIEN y la Fundación Champalimaud en Lisboa. La convocatoria reúne a prestigiosos médicos y científicos de todo el mundo para compartir los últimos avances en la investigación y tratamiento de estas enfermedades. Su clausura, este sábado, coincide con la celebración del Día Mundial del Alzheimer y ha sido la Reina la encargada de presidir el acto oficial. El Congreso se ha desarrollado en la sede de la Fundación Champalimaud. El viernes, a su llegada, Doña Sofía fue recibida por la presidenta de la Fundación, Leonor Beleza, el embajador en la República Portuguesa, Juan Fernández Trigo y la secretaria general de Investigación, Eva Ortega-Paíno y el vicepresidente de la Fundación Champalimaud, Joao Silveira. A continuación, Doña Sofía ocupó su lugar en el salón de actos donde el maestro de ceremonias, el director clínico del Campus Neurológico de la Universidad de Lisboa, Joaquím Ferreira, presentó la sexta sesión científica "Intervenciones no farmacológicas". A esta le siguieron otras ponencias en las que se detallaron los avances en el tratamiento de la enfermedad neurodegenerativa, la neuromodulación personalizada y el manejo del alzhéimer más allá de la medicación. Tras la finalización de las ponencias, la Reina Sofía recorrió la exposición de posters científicos acerca de enfermedades neurodegenerativas, donde recibió las explicaciones por parte de un investigador de la Fundación del Centro de Investigación de Enfermedades Neurológicas (CIEN), el doctor Pascual Sánchez. Por la tarde, asistió al concierto a cargo de la cantante de fados Katia Guerreiro. El Congreso, nacido de un esfuerzo conjunto de España y Portugal, se reafirma en un momento singular y esperanzador, marcado por la medicina de precisión, los avances en la detección precoz con biomarcadores, la experimentación con inteligencia artificial para el diagnóstico o el horizonte de nuevas terapias farmacológicas. A finales del pasado abril, poco después de su ingreso hospitalario, hizo su primera aparición en la ciudad de Cracovi , con motivo de la 36.ª edición de la Global Conference of Alzheimer's Disease International-ADI, en la que expertos de todo el mundo compartieron información sobre nuevas prácticas e innovaciones. Hace un año por estas fechas fue también la encargada de presidir el Congreso Internacional sobre Investigación e Innovación en Enfermedades Neurodegenerativas en el Auditorio de Edgar Neville de Málaga.

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La Reina Sofía retoma sus actos oficiales con una visita a Lisboa

La Reina Sofía retoma sus actos oficiales con una visita muy especial a Lisboa

La Reina Sofía ya está en Portugal

Su Majestad ha puesto rumbo al país del sur de Europa para presidir el acto oficial de celebración del Día Mundial del Alzheimer

Lo ha hecho en el marco del Congreso Internacional sobre Enfermedades Neurodegenerativas

La Reina Sofía en un acto en Portugal. (Foto: Gtres)

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La Reina Sofía ya está en Portugal. ■Su Majestad ha puesto rumbo al país del sur de Europa para presidir el acto oficial de celebración del Día Mundial del Alzheimer , que se conmemora el 21 de septiembre, en el marco del Congreso Internacional sobre Enfermedades Neurodegenerativas. Organizado conjuntamente por la Fundación Reina Sofía, CIEN y la Fundación Champalimaud, esta convención se ha convertido con el paso de los años, en un evento imprescindible para los profesionales de la investigación en este campo. Con un programa estructurado en siete sesiones científicas de calidad excepcional, el congreso cuenta con la participación de más de 30 investigadores y representantes de asociaciones de varios países.

Para esta ocasión, la Reina Sofía ha optado por una original americana Escada con un diseño cuello solapa y en color lila que está protagonizada por un bonito y llamativo bordado floral. Se trata de una prenda que Doña Sofía ha lucido en más de una ocasión, como en los Premios Sociales Fundación Mapfre en octubre de 2021. La reina Sofía ha completado su estilismo con una cartera de mano, botines y unos collares largos llamativos.

La Reina Sofía en un acto en Portugal. (Foto: Gtres)

Este viaje de Doña Sofía a Portugal es el primer acto oficial de la madre del Rey Felipe VI tras la temporada de verano, pues no llevaba a cabo compromisos institucionales desde que a comienzos del mes pasado asistió a la ceremonia de clausura de los Juegos Olímpicos de París 2024. La reina Sofía representó a la Casa Real desde un lugar de honor en la tribuna. La griega se sentó al lado de los reyes de Suecia y del príncipe Alberto de Mónaco. En el Estadio de Francia, la Reina disfrutó del 'paseíllo' de todas las delegaciones y del espectáculo, así como de las actuaciones; mientras el Rey Felipe y Doña Letizia, acompañados de sus hijas, disfrutaban de sus vacaciones privadas.

El lado más solidario de la Reina Sofía

La implicación de la Reina Sofía con la investigación y el tratamiento de enfermedades de tipo neurodegerativo es constante y viene de lejos. De hecho, esta es una de las pocas cuestiones en las que doña Letizia no ha tomado el relevo de su suegra tras la proclamación de Felipe VI en 2014. La madre de Felipe VI es Embajadora Honoraria de Alzheimer's Disease International (ADI). Además, a través de la Fundación Reina Sofía se puso en marcha en 2001 el Proyecto Alzheimer y en el año 2007, se inauguró el Centro Alzheimer de su fundación.

La Reina Sofía en un acto en Portugal. (Foto: Gtres)

Como presidenta de esta institución, Doña Sofía apoya siempre que puede cualquier iniciativa que tenga por objeto la investigación y ayuda en estos ámbitos. De ahí que el pasado mes de abril viajara a Cracovia a presidir la sesión inaugural de la 36th Global Conference of Alzheimer's Disease International, que tuvo lugar en Polonia. A la Reina Sofía, sea como fuere, las enfermedades neurodegenerativas le han tocado muy de cerca. En el año 1989, la princesa Eugenia de Grecia, madre de su prima Tatiana Radziwill , falleció a consecuencia del Alzheimer.

Comentar

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La reina Sofía asiste en Lisboa a un foro de expertos en neurociencia

La reina Sofía asiste en Lisboa a un foro de expertos en neurociencia

Por Newsroom Infobae

20 Sep, 2024 09:17 a.m. EST

Guardar

Nuevo

Lisboa, 20 sep (EFE).- La reina Sofía asistió este viernes en Lisboa al Congreso Internacional sobre Enfermedades Degenerativas, que durante tres días reúne en la capital lusa a decenas de expertos para dar a conocer a la ciudadanía los avances en la investigación contra el Alzheimer y otras dolencias.

Durante su visita a la segunda jornada del foro, celebrado en las instalaciones de la Fundación Champalimaud, la reina emérita saludó a algunos asistentes y, posteriormente, se sentó en primera fila como oyente en una de las sesiones, dedicada a las intervenciones no farmacológicas.

Durante la ponencia, presidida por el director del Laboratorio de Farmacología Clínica y Terapéutica de la Universidad de Lisboa, Joaquim Ferreira, diversos expertos hablaron de las distintas técnicas disponibles para abordar determinados factores de las enfermedades neurodegenerativas.

Los investigadores participantes de la sesión, de referencia mundial en neurología, fueron Pablo Martínez Lage, del centro de investigación Fundación CITA Alzheimer en San Sebastián (España); Giacomo Koch, del Departamento de Neurología Clínica de la Fundación Santa Lucía en Roma (Italia); y Arianne Gravesteijn, del Centro Médico Universitario de Ámsterdam (Países Bajos).

Tras la charla, la monarca pudo ver una serie de paneles expuestos en las instalaciones que mostraban los resultados de varios estudios sobre neurología.

Está previsto que esta tarde asista además a un concierto de fado que será celebrado en las instalaciones de la fundación.

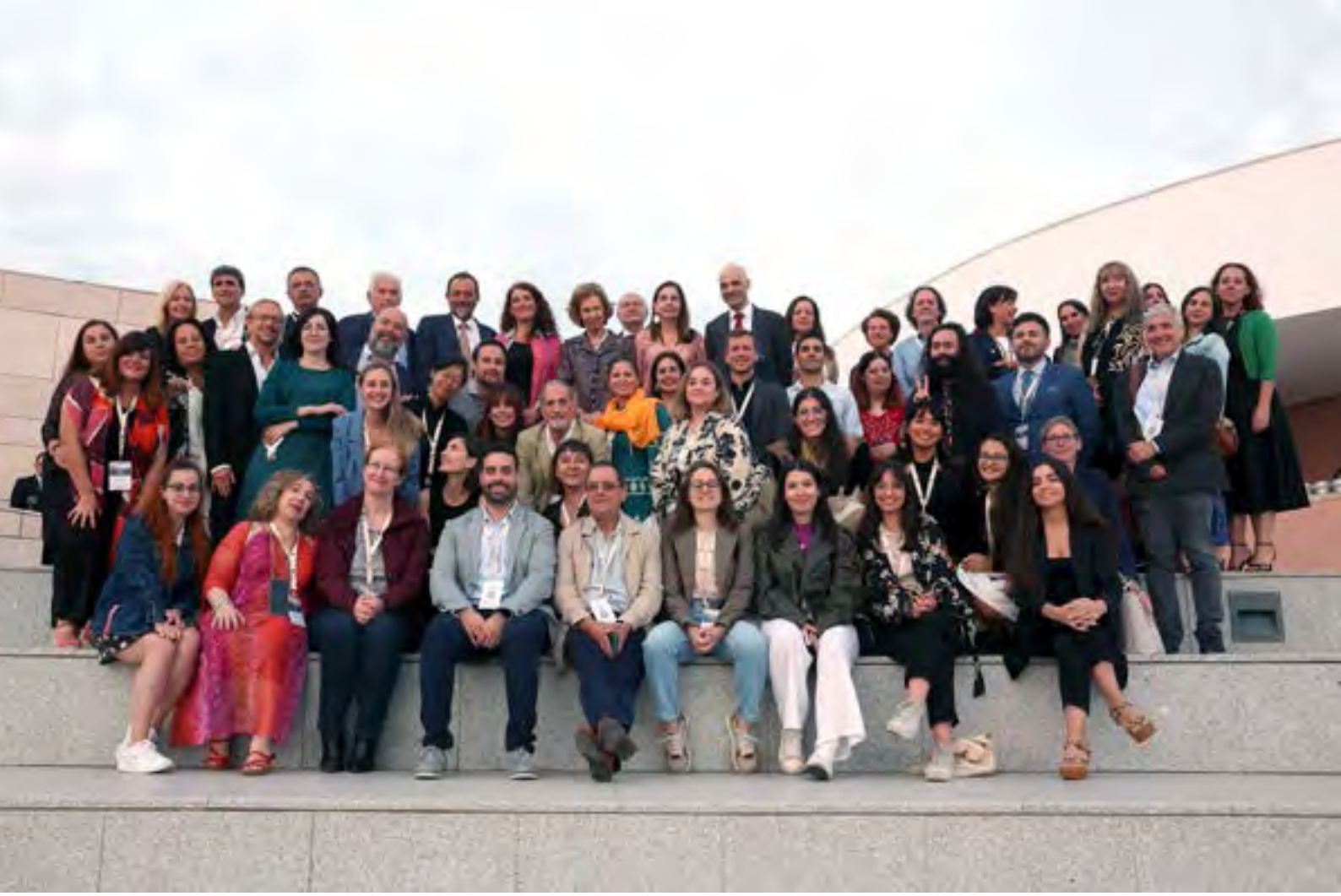
Este sábado, cuando finaliza el foro, la reina emérita presidirá el acto central del congreso, realizado con motivo del Día Mundial del Alzheimer, acompañada por Leonor Beleza, presidenta de la Fundación Champalimaud de Lisboa y exministra de Salud de Portugal.

Este evento se celebra desde 2013, organizado por la Fundación Reina Sofía y el Centro de Investigación en Enfermedades Neurológicas, a los que este año se suma la Fundación Champalimaud lusa.

El objetivo de la cita es dar a conocer los avances en la investigación contra el Alzheimer y otras dolencias neurodegenerativas y concienciar para encontrar soluciones y respuestas globales, dadas las consecuencias sociales que tienen.

Lisboa servirá como plataforma para que investigadores de todo el mundo compartan experiencias y descubrimientos fundamentales para la comprensión y tratamiento de estas enfermedades, en especial el Alzheimer.

El programa incluye siete sesiones científicas en las que participan más de 30 investigadores y representantes de asociaciones de varios países y abarca aspectos de detección temprana, variabilidad genética, neuroimagen, biomarcadores, medicina personalizada o terapias no farmacológicas. EFE rmj/cch/ads



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