10th Alzheimer's disease/Parkinson's disease (AD/PD) Conference

March 9-13, 2011, Barcelona, Spain

The 10th AD/PD 2011 conference was held from 9-13, March 2011 at the Centre Convencions Internacional Barcelona (CCIB), Spain and aimed to present the cutting edge advances to unravel the mechanisms involved in Alzheimer's (AD), Parkinson's (PD) and other related neurodegenerative diseases like frontotemporal dementia (FTD), amyotrophic lateral sclerosis (ALS), progressive supranuclear palsy (PSP), dementia with Lewy bodies (DLB) etc. An international panel of experts in neurodegenerative diseases shared and compared exciting new discoveries on the role of pathogenic protein aggregates in the pathogenesis, diagnosis, and imaging as well as their impact on therapy and prevention in the above mentioned diseases. Another important and relevant area was Genome Wide Association (GWA) study in identifying new genes as susceptibility factors in AD and PD. The conference agenda included over 300 lectures (including 7 plenary talks) and 1218 poster presentations. It witnessed a global participation from academia and industry with over 2750 participants from 71 countries attended the meeting.

The first day had back-to-back plenary lectures from two renowned neuroscientists- a protein chemist and a neurogeneticist. The first one on ubiquitin proteolytic system was delivered by the 2004 Nobel Prize winner in Chemistry, Prof. Aaron Ciechanover from Israel. Next, Prof. Christine Van Broeckhoven, Belgium talked on the genetics of late-onset neurodegenerative diseases where she focused on AD and FTD. She also shared her recent findings on GWA study that implicate *BIN1* and *CR1* genes to be associated with AD risk. As a trend of the AD/PD meeting one plenary lecture took us to the history of a specific research of the host country relevant to the conference theme and this year Dr. I. Ferrer reviewed the history of neurology and research in AD and PD in Spain.

There were 44 symposia that separately covered topics on animal models for AD, PSP, PD and Prion diseases, inflammatory mediators and neuroinflammation in neurodegeneration; pathogenic proteins like alpha synuclein, tau, Parkin and

LRRK2, TDP-43, a-beta toxicity mediator, gamma-secretase and progranulin etc. In addition, there were symposia on clinical (methodologies, management of neurogenic orthostatic hypotension in PD, imaging, cognitive deficits in PD, PET, MRI, cerebral amyloid angiopathy, DLB) and genetic aspects in neurodegeneration. Innovative approaches and biomarkers, treatment and prevention (immunotherapy, affitope vaccines), neurogenesis, stem cells and growth factors, current theories and mechanisms being studied for treatment; mitochondrial impairments and treatments; A β protofibrils and the search for a disease modifying therapy in AD, propagation of misfolded proteins and opportunities in treatment, APOE, treatments and lipid metabolism; challenges and potential treatments in AD and PD, treatment options in AD, PD and ALS) as well as drug discovery and development in neurodegenerative diseases (pyroglutamate A β , AD, PD and alpha synuclein aggregation, advanced drug development, drug discovery for AD) were some of the other symposia of this meeting.

Prof. Virginia M.-Y. Lee, USA presented her well established cellular system that can robustly develop NFT (neurofibrillary tangles)-like tau aggregates thereby providing mechanistic insights into NFT pathogenesis and a potential tool for identifying tau-based therapeutics for AD in future. Dr. Athena Andreadis, USA presented a very interesting work where she demonstrated that RNA processing proteins located on chromosome 21 cause errors in the splicing regulation of tau exon 10 and therefore it might contribute to early dementia among individuals with Down's syndrome.

Prof. Christian Haas and Dr. Anja Capell, Germany, described a promising approach for a possible treatment against FTD. FTD can be caused due to loss-of-function mutation in the *Progranulin* (*PGRN*) gene which encodes a growth factor. They have now shown that various drugs that are already on the market to treat diseases like malaria, angina pectoris or heart rhythm disturbances as well as some highly selective inhibitors of vacuolar ATPases can increase the production of progranulin and this occurs by a translational mecha-

nism that is independent of autophagy, endocytosis or lysosomal degradation. Accordingly, these drugs might well act as good candidates for therapeutic use to prevent progranulin-dependent neurodegeneration.

The lectures by Prof. John Hardy, UK and Dr. Andrew Singleton, USA featured on the next stage to understand the mechanism for those genes showing significant association. Dr. Singleton presented, on behalf of the International Parkinson's Disease Genomics Consortium (IPDGC), a meta-analysis of 5 PD GWA studies and replicated the significantly associated loci in additional samples. Apart from the reported association at *MAPT*, *SNCA*, *HLA-DR*, *BST1*, *GAK* and *LRRK2*, 5 new loci (*ACMSD*, *STK39*, *MCCC1*, *LAMP3*, *SYT11*, *CCDC62* / *HIP1R*) showed significant association with PD. Interestingly, individuals at the top quintile of the genetic risk spectrum with respect to the 11 identified loci had 2.5 times the risk of PD compared to those in the bottom quintile. The biological consequences of these loci in terms of gene expression and DNA methylation were also studied from frontal and temporal cortices, pons and cerebellar regions in 150 brain samples. Presently, age at onset GWA study is in progress that might lead to the identification of newer susceptibility genes in PD.

There was informal networking with Professors where specific areas were covered by as many as 6 different eminent scientists each day. In addition, there were "Meet the expert" where 3 parallel sessions were held for an hour. For the first time, this conference's winners of Cornelli awards, Leda Hanin award, Greenberg family awards and Junior faculty awards were scheduled to deliver their oral presentations on their research and were allotted the same time as the other oral presentations of 20 minutes. India's Dr. Bhavani Shankara Bagepally, National Institute of Mental Health & Neurosciences, Bangalore, was one of the proud recipients of the Junior faculty award and his study on *ApoE4* genotype status and white-matter structural integrity in AD was well appreciated. The poster session was held in 2 sessions and featured a wide range of topics commensurate with

the theme of the conference as eluded to earlier.

Overall, the 10thAD/PD conference was a huge success and advancement in the research activities in neurodegenerative

diseases all around the world showed great promise for better understanding of these diseases as well as the possible future therapies. The next conference (AD/PD 2013) will be held at Florence, Italy during March 6-10, 2013.

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Erratum

The manuscript published in Ann Neurosci, 17(1) 18-21, 2010 had wrongly mentioned rats instead of mice under the "Methods" section. The authors have clarified that the study was conducted on mice with required Institute Animal Ethical Committee approval no. 01/032/08. This error is deeply regretted.