

Early treatment is the key to beating Alzheimer's disease in later life

World-renowned neuroscientist Professor Bart De Strooper will deliver the 'Brain Prize Lecture' today at the 5th European Academy of Neurology (EAN) Congress.

OSLO, NORWAY, June 29, 2019 /EINPresswire.com/ -- World-renowned neuroscientist Professor Bart De Strooper will deliver the prestigious 'Brain Prize Lecture' today at the 5th European Academy of Neurology (EAN) Congress in Oslo, Norway, and outline why we need to intervene much earlier if we want to protect people against the symptoms of Alzheimer's disease in later life.



5th Congress of the European Academy of Neurology

This approach is based on decades of research on the causes of hereditary

forms of Alzheimer's disease and, unfortunately, disappointing clinical trial outcomes so far.

Over the past decades, Professor De Strooper has made important contributions to our understanding of the mechanisms of Alzheimer's disease. This included uncovering that gene

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Scientists need to shift their focus to the earlier stage of the disease and think about the cellular environment in which the disease develops" *Professor Bart De Strooper* Alzheimer's disease. This included uncovering that gene mutations in presenilin – part of the γ-secretase protein complex that 'cuts' other proteins into smaller pieces – lead to the production of abnormal amyloid to form plaques in the brains of people with Alzheimer's disease.

His discoveries mark breakthrough findings in the dementia field, furthering the understanding of how Alzheimer's disease may begin and provide potential new mechanisms to target with drug therapies in the future.

Professor De Strooper, Director of the UK Dementia Research Institute, London and group leader at the VIB-KU Leuven Center for Brain & Disease Research, Leuven, was awarded the Lundbeck Foundation Brain Prize for his work in this area last year and explained that understanding how these mutations drive dementia is important in developing new therapies. "We need to understand what is going on in the brain and how the brain operates while these changes occur", he commented. "Treating amyloid at a very early stage could protect against symptoms later on and we must target these processes if we want to make the most effective treatments."

The amyloid hypothesis works on the assumption that the accumulation of peptide amyloid- β plaques is the main cause of Alzheimer's disease by triggering neurogenerative processes. This leads to the loss of memory and cognitive ability and has guided research into dementia treatments for the past 25 years.

Professor De Strooper commented, "For decades we have studied Alzheimer's disease in its final stages. We now know that the disease process in the brain can start decades before the first symptoms arise. Studying the cognitive abnormalities in Alzheimer's disease patients has indeed taught us a great deal about the disease, but we are always looking at an advanced stage or even post-mortem. You could compare this to studying cancer but only reviewing the metastatic disease, completely missing the stage where the disease actually originates and begins to spread. Needless to say, in both cases the chance of clinical success and making a meaningful change in treatment for patients, would be in those earlier stages."

Trials for new Alzheimer's drugs have, until now, often been tested in patients with advanced disease and it is therefore difficult to change the disease course. Professor De Strooper argues that we need to understand what happens before the plaques form in the cellular phase. "Scientists need to shift their focus to the earlier stage of the disease and think about the cellular environment in which the disease develops", he observed.

Although budgets have been cut in dementia research, Professor De Strooper added there is still room for plenty of optimism due to the discoveries made on the neurodegenerative process. "Even though the situation is more complex than previously anticipated, if we compare the budgets and publication numbers in this disease area with those for cancer, it is simply not true that the success rate is exceptionally low. We just need to continue to invest heavily in new, innovative research to provide patients with optimal outcomes."

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Notes to Editors:

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About the Expert:

Professor Bart De Strooper is Director of the UK Dementia Research Institute (UK DRI) in London, UK, Professor of Alzheimer's Disease at University College London, UK, Professor of Molecular Medicine at the University of Leuven, Belgium) and Group Leader (and former Director) at the VIB-KU Leuven Center for Brain & Disease Research, Leuven, Belgium.

His scientific awards include a Brain Prize (2018), a European Grand Prix for Research (2018), and a Potamkin Prize (2002).

EAN - The Home of Neurology:

The European Academy of Neurology (EAN) is Europe's home of neurology. Founded in 2014, through the merger of two European neurological societies, EAN represents the interests of more than 45,000 individual members and 47 national institutional members from across the continent. This year, EAN celebrates its fifth year of fostering excellence in European neurology and will bring together more than 6,000 neurologists and related scientists to the biggest general neurology conference in Europe.

In Oslo, Norway, from June 29 to July 2, there will be an exchange of knowledge and promotion of best practice, with a focus on the main theme of neuroinflammation. The EAN Congress will also cover all neurological diseases and disorders, including the big 7: epilepsy, stroke, headache, multiple sclerosis, dementia, movement disorders, neuromuscular disorders.

References:

1. The Brain Prize Lecture: The prodromal, cellular phase of Alzheimer's Disease: towards a novel understanding of the disorder. Presented on Sunday, 30 June, 2019, at The 5th European Academy of Neurology (EAN) Congress in Oslo, Norway

2. Alzheimer's-Causing Mutations Shift Aβ Length by Destabilizing γ-Secretase-Aβn Interactions: <u>https://www.cell.com/cell/fulltext/S0092-8674(17)</u>30811-5

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