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PRESS RELEASE

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EARLIER DIAGNOSIS AND BETTER TREATMENT FOR ALZHEIMER'S DISEASE

As the most frequent neurodegenerative disorder worldwide, Alzheimer's Disease (AD) is one of the most devastating diagnoses a patient can receive. Almost 50 million people currently live with AD or other dementia, with rates predicted to exceed 131.5 million by 2020. In work presented at the FENS Forum of Neuroscience in Berlin today (10 July), scientists may now be closer to identifying the disease earlier than before and treating it more effectively, possibly with a simple dietary intervention.

The main clinical symptom of AD is dementia, the successive loss of control over cognitive abilities, yet this is often only detectable several years after onset of the disease. This means it may be too late to intervene and that treatment given is targeting the effects of the disease rather than its cause. A major challenge in AD therefore lies in identifying signature changes in the brain to allow earlier diagnosis and more effective treatment.

In an international effort including laboratories from Germany, Spain, and Switzerland, **Dr Jose Sanchez-Mut** and colleagues from École Polytechnique Fédérale de Lausanne (EPFL), Switzerland, used cell culture, mouse models and human post-mortem tissue to explore whether a particular chemical modification of DNA, the body's genetic code, might underlie the risk of developing AD.

As Dr Sanchez-Mut explained, "We already know quite a lot about the amyloid pathway in Alzheimer's disease, whereby a microscopic protein fragment called beta-amyloid accumulates in the brain, disrupting communication between cells and eventually killing them. What we sought to find out is why this pathway is triggered, resulting in the devastating pathology we see in Alzheimer's disease."

The team's studies focused on a gene called PM20D1 which, when its activity was boosted in a genetically modified mouse model of AD, both reduced amyloid plaque formation and improved cognitive performance. Conversely, when the activity of PM20D1 was down-regulated, amyloid amounts increased and cognition was impaired.

Thought to work through modifying levels of harmful free radicals, which are by-products of normal cellular activity, the researchers believe that manipulating PM20D1 as simply as with a food-based drug could herald use of a dietary intervention in those at risk of AD.

Looking to possible links between their work and other nervous system disorders, Dr Sanchez-Mut added, "We are interested in related reports that PM20D1 is associated with obesity and diabetes, both risk factors for AD, but also to two other nervous

system disorders, multiple sclerosis and Parkinson's disease. Its possible involvement in conditions beyond Alzheimer's disease certainly warrants further study."

END

Symposium: S45 - Epigenetic remodeling of brain function: Addiction and neurodegeneration

Abstract: Allele-specific PM20D1 epigenetic regulation is associated with Alzheimer's disease

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NOTES TO EDITORS

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The 11th FENS Forum of Neuroscience, the largest basic neuroscience meeting in Europe, organised by FENS and hosted by the German Neuroscience Society will attract more than 7,000 international delegates. The Federation of European Neuroscience Societies (FENS) was founded in 1998. With 43 neuroscience member societies across 33 European countries, FENS as an organisation represents 24,000 European neuroscientists with a mission to advance European neuroscience education and research. <https://forum2018.fens.org/>

Further reading:

PM20D1 is a quantitative trait locus associated with Alzheimer's disease. J Sanchez-Mut, H Heyn, B Silva, L Dixsaut, P Garcia-Esparcia, E Vidal, S Sayols, L Glauser, A Monteagudo-Sánchez, J Perez-Tur, I Ferrer, D Monk, B Schneider, M Esteller, J Gräff *Nature Medicine*, 2018; 24 (5): 598 DOI: [10.1038/s41591-018-0013-y](https://doi.org/10.1038/s41591-018-0013-y)