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PET IMAGING OF PROTEINOPATHIES IN NEURODEGENERATIVE DISEASE

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Traditional nuclear medicine ligands were designed to target cellular receptors or transporters with a binding pocket and a defined structure activity relationship. More recently, tracers have been developed to target pathological protein aggregations. Aggregations of proteins such as tau, α-synuclein, and β-amyloid (Aβ) have been identified in neurodegenerative diseases, including Alzheimer's disease (AD) and other dementias, and Parkinson's disease (PD). Indeed, Aβ deposition is a hallmark of AD, and detection methods have evolved from coloured dyes to modern ^{18}F-labelled positron emission tomography (PET) tracers. Such tracers are becoming increasingly established in routine clinical practice for evaluation of Aβ neuritic plaque density in the brains of adults who are being evaluated for AD and other causes of cognitive impairment. While similar in structure, there are key differences between the available compounds in terms of dosing/dosimetry, pharmacokinetics, and interpretation of visual reads. In the future, quantification of Aβ-PET may further improve its utility. Tracers are now being developed for evaluation of tau protein, which is associated with decreased cognitive function and neurodegenerative changes in AD, and is implicated in the pathogenesis of other neurodegenerative diseases. While no compound has yet been approved for tau imaging in clinical use, it is a very active area of research. Development of tau tracers comprises in-depth characterisation of existing radiotracers, clinical validation, a better understanding of uptake patterns, test-retest/dosimetry data, and neuropathological correlations with PET. Tau imaging may allow early, more accurate diagnosis, and monitoring of disease progression, in a range of conditions. In conclusion, several PET tracers for detection of pathological protein depositions are now available for clinical use, particularly PET tracers that bind to Aβ plaques. Tau-PET tracers are currently in clinical development. These tracers will continue to change our understanding of complex disease processes.

Biography

Stephens Andrew W has received an MD and a PhD in Biochemistry, Biophysics and Genetics from the University of Colorado. He was Board certified in Internal Medicine and had a Clinical Practice before entering Pharmaceutical Development. He is a Founder and the Chief Medical Officer at Piramal Imaging, GmbH, responsible for all clinical research and development activities including the approval of ^{18}F-florbetaben (NeuraCeq). He has more than 25 years of experience in the Pharmaceutical Industry, primarily in the areas of translational medicine, and diagnostic imaging of neurodegenerative, oncological and cardiovascular diseases. He began his pharmaceutical industry career investigating RNA Aptamers at NeXagen/NeXstar, and Gilead. As a Senior Director of Translational Medicine at OSI Pharmaceuticals, he was responsible for early clinical studies of a number of anti-cancer oral signal transduction inhibitors. Most recently, he was VP, Head of Experimental Medicine Oncology/Diagnostic Imaging for Bayer Pharma.

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