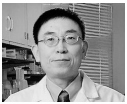
SEPIA lecture series

Thursday, June 29
11:00 CEST

Skin misfolded protein-seeding activity as a diagnostic biomarker across neurodegenerative diseases



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Parkinson's disease (PD) and Alzheimer's disease (AD) are the two most common neurodegenerative diseases that have recently been proposed to share the pathogenic mechanism similar to that of prion diseases (PrD) associated with an infectious prion protein (PrPSc).

Their definitive diagnosis mainly relies on the examination of the pathologically misfolded α-synuclein (α-SynP) containing Lewy bodies, amyloid β (Aβ) plaque and phosphorylated tau tangles, or PrPSc aggregates in the brain.

By using ultrasensitive seed-amplification assays (SAA) with real-time quaking-induced conversion (RT-QuIC) and protein misfolding cyclic amplification (PMCA) technologies, we recently reported for the first time that the seeding activity of the skin PrPSc or α-SynP (PrPSc- or α-SynP-SA) could be a biomarker for diagnosis of prion diseases or PD.

Our new study also revealed that similar to brain tissues, skin tissues of patients with AD and other tauopathies showed detectable seeding activity of misfolded tau by RT-QuIC assay. Our studies provided evidence that prion-like seeding activity of misfolded proteins including PrPSc, α-SynP and tau can be detectable in the skin tissues of different neurodegenerative diseases. The seeding activity of these prion-like misfolded proteins could serve as a diagnostic biomarker across neurodegenerative diseases.

This lecture will be held online:

- **by registering via Livestorm** with this link : **https://app.livestorm.co/fontenay-aux-roses/lecture-wen-quen-zou**

or by scanning the QR-code

For any question about the event please contact emmanuel.comoy@cea.fr