

Age associated with amyloid-beta kinetics

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physiological and pathophysiological changes and may be applicable to other proteinopathies."

Several authors disclosed financial ties to C2N Diagnostics, which has licensed related patents from Washington University.

More information: <u>Abstract</u> <u>Full Text (subscription or payment may be required)</u>

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(HealthDay)—Increasing age is associated with slowed amyloid-? (A?) turnover, according to a study published online July 20 in the *Journal of Neurology*.

Noting that <u>age</u> is the single greatest risk factor for Alzheimer's disease, Bruce W. Patterson, Ph.D., from Washington University in St. Louis, and colleagues examined the correlation between age, amyloidosis, and A? <u>kinetics</u> in the central nervous system of humans. A? kinetics were assessed in 112 participants.

The researchers found that increasing age was significantly associated with slowed turnover rates of A? (2.5-fold longer half-life over five decades of age). Specifically in participants with <u>amyloid</u> deposition, there were independent effects on A?42 kinetics. Amyloidosis correlated with an increased irreversible loss of soluble A?42 (more than 50 percent) and a A? reversible exchange rate that was 10-fold higher.

"These findings reveal a mechanistic link between human aging and the risk of amyloidosis, which may be owing to a dramatic slowing of A? turnover, increasing the likelihood of <u>protein misfolding</u> that leads to deposition," the authors write. "This study provides an example of how changes in protein turnover kinetics can be used to detect



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