



**PRESS RELEASE** (2017/06/22)

**Critical roles of Cathepsin B in inducing Alzheimer's disease-like phenotypes**

A number of clinical and experimental studies have revealed a strong association between periodontal disease and the rate of cognitive decline in Alzheimer's disease (AD), however, the mechanism of the association still remains unknown. In the present study, the research groups of Associate Professor Zhou Wu and Professor Hiroshi Nakanishi have shown for the first time that systemic exposure to lipopolysaccharide from *Porphyromonas gingivalis* (*Pg*LPS) induces Alzheimer's disease (AD)-like phenotypes, including microglia-mediated neuroinflammation, accumulation of amyloid  $\beta$  ( $A\beta$ ) and impairment of learning and memory in middle-aged mice, but not in young mice. Furthermore, researchers found that cathepsin B (CatB), a lysosomal cysteine protease, plays critical roles for inducing these AD-like phenotypes.

The study confirmed that CatB plays a critical role in the link between periodontitis and AD. This led to the idea that CatB may be a therapeutic target for preventing periodontitis-associated cognitive decline in AD.

The research achievement was published online in *Brain, Behavior, Immunity* on June 10, 2017.

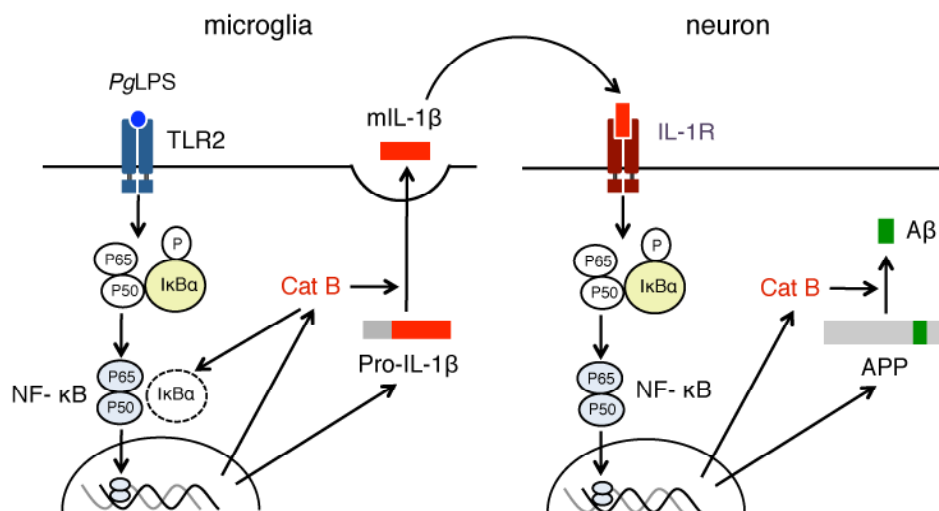


Figure: Schematic illustration of the critical roles of cathepsin B (CatB) in initiating AD-like phenotypes during chronic systemic *Pg*LPS exposure.

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