

Destabilizing preformed fAbeta / tannic acid

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Anti-amyloidogenic activity of tannic acid and its activity to destabilize Alzheimer's beta-amyloid fibrils in vitro.

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Inhibition of the accumulation of amyloid beta-peptide (Abeta) and the formation of beta-amyloid fibrils (fAbeta) from Abeta, as well as the destabilization of preformed fAbeta in the CNS would be attractive therapeutic targets for the treatment of Alzheimer's disease (AD). We previously reported that nordihydroguaiaretic acid (NDGA) and wine-related polyphenols inhibit fAbeta formation from Abeta(1-40) and Abeta(1-42) as well as destabilizing preformed fAbeta(1-40) and fAbeta(1-42) dose-dependently in vitro. Using fluorescence spectroscopic analysis with thioflavin T and electron microscopic studies, we examined the effects of polymeric polyphenol, tannic acid (TA) on the formation, extension, and destabilization of fAbeta(1-40) and fAbeta(1-42) at pH 7.5 at 37 degrees C in vitro. We next compared the anti-amyloidogenic activities of TA with myricetin, rifampicin, tetracycline, and NDGA. TA dose-dependently inhibited fAbeta formation from Abeta(1-40) and Abeta(1-42), as well as their extension. Moreover, it dose-dependently destabilized preformed fAbetas. The effective concentrations (EC50) of TA for the formation, extension and destabilization of fAbetas were in the order of 0-0.1 microM. Although the mechanism by which TA inhibits fAbeta formation from Abeta as well as destabilizes preformed fAbeta in vitro is still unclear, it could be a key molecule for the development of therapeutics for AD.

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