EFFECTS OF FEMALE STEROID HORMONE DEPLETION ON TAU PATHOLOGY IN THE THY-
TAU22 TRANSGENIC MODEL

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Introduction: At menopause, there is a depletion of estrogens and progesterone that may increase risk for the development of Alzheimer’s disease (AD) in women. Nevertheless, epidemiological studies are still controversial. In AD animal models, many data are available on the effects of steroid hormones on cognition and amyloid pathology after ovariectomy. However, their effects on Tau pathology, the other neuropathological hallmark, are not fully uncovered. Tau pathology is characterized by Tau hyperphosphorylation and aggregation.

Aim: In the present work, we addressed the effects of estrogen depletion on Tau phosphorylation in a Tau transgenic model, namely THY-Tau22.

Methods: THY-Tau22 mice were ovariectomized or not at 4.5 months and then sacrificed at 8 months. Brains were used for biochemical and immunohistochemical analyses using different phosphorylation-dependent anti-Tau antibodies (AT8, AT270, AT100, AT180, AP422, AD2, Tau-1).

Results: Tau proteins as identified by AD2 and AT8 were more hyperphosphorylated in ovariectomized animals brain homogenates than in controls. However, there was no difference with other antibodies raised against pathological epitopes such as AT100. Finally, no major change in neurofibrillary degeneration was identified after ovariectomy.

Conclusion: These data indicate that ovariectomy is not strongly deleterious for Tau pathology.