

Abstract

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Greater endogenous estrogen exposure is associated with longer telomeres in postmenopausal women at risk for cognitive decline.

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OBJECTIVE: Longer duration of reproductive years of life and thus greater exposure to endogenous estrogen may be associated with a lower risk of age-related diseases in women. The present study examined the relationship between estimated endogenous estrogen exposure and telomere length (TL) and telomerase activity, two biomarkers of cellular aging, in a sample of postmenopausal women at risk for cognitive decline.

METHODS: Telomere length was measured using a quantitative PCR method and telomerase activity by TRAP (Telomere-Repeats Amplification Protocol) assay in peripheral blood mononuclear cells (PBMCs). Study subjects were 53 postmenopausal women (35 with natural and 18 with surgical menopause) receiving hormone therapy (HT) for at least one year or longer. Length of reproductive years of life, computed as the difference between age at menopause and age at menarche, was used as a proxy of duration of exposure to endogenous estrogen. Length of time on HT was the measure used for duration of exogenous estrogen exposure.

RESULTS: We found that longer endogenous estrogen exposure was associated with greater TL (standardized $\beta=0.06$, Wald $\chi(2)=3.7$, $p=0.04$) and with lower telomerase activity (standardized $\beta=-0.09$, Wald $\chi(2)=5.0$, $p=0.03$). Length of reproductive years was also inversely associated with the combination of short TL and high telomerase (OR=0.78, 95% CI: 0.63, 0.97, $p=0.02$). Length of HT use was not associated with TL or telomerase activity in this study.

CONCLUSION: The results suggest that the endogenous estrogens may be associated with deceleration of cellular aging. This is the first study to examine associations between endogenous estrogens, telomere length and telomerase activity.

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