

Abstract

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Leukocyte telomere length in major depression: correlations with chronicity, inflammation and oxidative stress--preliminary findings.

Wolkowitz OM, Mellon SH, Epel ES, Lin J, Dhabhar FS, Su Y, Reus VI, Rosser R, Burke HM, Kupferman E, Compagnone M, Nelson JC, Blackburn EH.

Department of Psychiatry, University of California San Francisco School of Medicine, San Francisco, California, United States of America.

BACKGROUND: Depression is associated with an unusually high rate of aging-related illnesses and early mortality. One aspect of "accelerated aging" in depression may be shortened leukocyte telomeres. When telomeres critically shorten, as often occurs with repeated mitoses or in response to oxidation and inflammation, cells may die. Indeed, leukocyte telomere shortening predicts early mortality and medical illnesses in non-depressed populations. We sought to determine if leukocyte telomeres are shortened in Major Depressive Disorder (MDD), whether this is a function of lifetime depression exposure and whether this is related to putative mediators, oxidation and inflammation.

METHODOLOGY: Leukocyte telomere length was compared between 18 unmedicated MDD subjects and 17 controls and was correlated with lifetime depression chronicity and peripheral markers of oxidation (F2-isoprostane/Vitamin C ratio) and inflammation (IL-6). Analyses were controlled for age and sex.

PRINCIPAL FINDINGS: The depressed group, as a whole, did not differ from the controls in telomere length. However, telomere length was significantly inversely correlated with lifetime depression exposure, even after controlling for age ($p < 0.05$). Average telomere length in the depressed subjects who were above the median of lifetime depression exposure (≥ 9.2 years' cumulative duration) was 281 base pairs shorter than that in controls ($p < 0.05$), corresponding to approximately seven years of "accelerated cell aging." Telomere length was inversely correlated with oxidative stress in the depressed subjects ($p < 0.01$) and in the controls ($p < 0.05$) and with inflammation in the depressed subjects ($p < 0.05$).

CONCLUSIONS: These preliminary data indicate that accelerated aging at the level of leukocyte telomeres is proportional to lifetime exposure to MDD. This might be related to cumulative exposure to oxidative stress and inflammation in MDD. This suggests that telomere shortening does not antedate depression and is not an intrinsic feature. Rather, telomere shortening may progress in proportion to lifetime depression exposure.

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