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Psychiatric disorders and leukocyte telomere length: Underlying mechanisms linking mental illness with cellular aging.

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Abstract

Many psychiatric illnesses are associated with early mortality and with an increased risk of developing physical diseases that are more typically seen in the elderly. Moreover, certain psychiatric illnesses may be associated with accelerated cellular aging, evidenced by shortened leukocyte telomere length (LTL), which could underlie this association. Shortened LTL reflects a cell's mitotic history and cumulative exposure to inflammation and oxidation as well as the availability of telomerase, a telomere-lengthening enzyme. Critically short telomeres can cause cells to undergo senescence, apoptosis or genomic instability, and shorter LTL correlates with poorer health and predicts mortality. Emerging data suggest that LTL may be reduced in certain psychiatric illnesses, perhaps in proportion to exposure to the psychiatric illnesses, although conflicting data exist. Telomerase has been less well characterized in psychiatric illnesses, but a role in depression and in antidepressant and neurotrophic effects has been suggested by preclinical and clinical studies. In this article, studies on LTL and telomerase activity in psychiatric illnesses are critically reviewed, potential mediators are discussed, and future directions are suggested. A deeper understanding of cellular aging in psychiatric illnesses could lead to re-conceptualizing them as systemic illnesses with manifestations inside and outside the brain and could identify new treatment targets.

KEYWORDS: Aging; Antidepressant; Anxiety; Bipolar affective disorder; Depression; Disease; Early life adversity; Inflammation; Leukocytes; Major depressive disorder; Manic-depression; Mortality; Neurotrophic; Oxidative stress; Post-traumatic stress disorder; Psychosis; Schizophrenia; Stress; Telomerase; Telomeres

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