

The Effect of Childhood Stress on Telomeres (A Literature Review)

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Abstract

Telomeres are DNA repeat structures that are bound by protein and found at the chromosomal end, they regulate the ability of cells to replicate, prevent fusion of chromosomes from forming together and prevent genetic data leakage. Although telomere shortening takes place naturally as one ages, several research studies suggest that there is a correspondence between physiological environment and length changes of telomeres. This study highlights on how childhood stress such as maternal depression, low economic status, violence, and family disruption impact the length of telomeres. Studies by the National Institute of Health confirm that exposure to childhood stress accelerates the shortening of telomeres. Additionally, this review assesses how each type of childhood stress at different developmental stages alters telomeres' length. To further understand how these factors affect telomere length, mechanisms contributing to telomere shortening have also been discussed.

Keywords: Childhood stress, Telomere length, Biological aging, Telomere length (TL), Leukocyte Telophase Length (LTL)

1. Introduction

Changes in telomeres such as shortening are very visible during early development. However later at maturity visibility will be low (Frenck et al., 1998). The physical, social, and emotional development of children during childhood directly affects their overall and how their adult life will be. Studies by Clemente et al. (2019) have demonstrated the importance of early development years where an increase in exposures to nitrogen dioxide while at pregnancy was linked to shortening of telomere in samples of blood collected from children of 5-12 years.

As body cells differentiate and divide, telomeres are yet to replicate fully due to the difficulty of the DNA polymerases to complete the multiplication of linear molecules' ends causing telomere shortening in every multiplication. Telomeres shorten in humans with age in somatic cells replicating (Kananen et al., 2010). The activity of telomerase increases in T cells of individuals suffering acute antigen exposure; however, its activity reduces with exposure to repeated antigens simulation as the cells near maturation. Rare genetic diseases such as dyskeratosis congenital reduce the body's ability to synthesize a sufficient amount of telomerase enzyme.

This leads to shortened telomeres, leading to premature death from gradual bone marrow failure and eventual exposure to infection-causing pathogens. Childhood stress, another major cause of telomere shortening, can be linked to premature deaths from a weak immune system (Willis et al., 2019). Ranking of telomeres between childhood and early adulthood was conversely related to exposure to residential traffic at the address of birth of children. (Bijnens et al., 2017). Different childhood stress affects telomere structure differently.

2. Maternal stress

According to a study by Angela et al. (2014), maternal stress during pregnancy significantly affects telomeres. The article categorizing maternal stress as a contributor to telomere structure is based on two studies assessing the link between stress during pregnancy and the telomere length of a born child. In the first study by Entringer et al., 2011, exposure to stress during the prenatal period was found to be a leading contributor to adult offspring leukocyte telomere length.

The authors evaluated maternal stress considering other variables, age, sex, postnatal early-life adversity, birth weight percentile, and BMI were controlled.

In the second study by Entringer et al. (2013), maternal psychological stress on the leukocyte telomere length of a newborn was assessed in research on 27 mother-newborn pair; the authors reported a linear, independent and significant effect on stress from pregnancy on newborn leukocyte telomere length accounting for a quarter of change in telomere length. Other factors affecting leukocyte telomere length of a newborn, especially the risk factors of the mother like age at conception and cardiovascular disease, were however not considered in the analysis (Broer et al., 2013)

3. Chronic psychological stress at childhood on telomeres

The study by Angela et al. (2014) shows that extremely high levels of psychological stress cause shorter telomeres, reduced activity of telomerase enzyme and increased oxidative stress. This was concluded from a study by Epel et al. (2004), where leukocyte telomere length was evaluated in (PBMCs), the activity of telomerase, and stress from air pollution of fifty-eight caregiving mothers for either a chronically ill child or healthy. The duration of caregiving was also significantly related to shorter leukocyte telomere length, suggesting that chronic stress was a key determinant on telomeres. A weakness of the study was that the results were from the short analysis. The size of the sample was also another weakness.

Another study by Dmajanovic et al. (2007) in Serbia compared cytokine production, peripheral blood mononuclear cells (PBMCs) composition, depressive symptoms, Leukocyte telomere length, and activity of telomerase enzymes between caregivers of control and caregivers of children suffering Alzheimer's disease. They also had a short leukocyte telomere length in peripheral blood mononuclear cells than caregivers of controls. Telomerase activity in peripheral mononuclear cells and T cells had a significantly high control.

Finally, a research study by Humphreys et al. (2012), revealed that reported shorter Leukocyte telomere length among children born to mothers with highly elevated levels of psychological stress linked to domestic violence. The study also revealed that the duration of time in a toxic relationship and bearing kids in the relationship was associated with shorted telomeres.

4. Oxidative stress on telomers

The study by Rebecca et al. (2021) analyzed 20-30 ml sampled blood obtained from an umbilical cord and placental tissue directly after birth in 296 participants for telomere length showed that shortening and dysfunction of telomere were increased by oxidative stress. Infants born of participants that smoked, especially in their third trimester, showed a significant difference in telomere length than other participants. This can be attributed to the fact that the placenta has a thin membrane during the third trimester, allowing some substances to go across (Mayer et al. 2019). This includes; pro-oxidant species, nicotine found in tobacco, promethium, pro-inflammatory cytokines, and. The study suggests that entry of promethium into the circulatory system of a fetus may cause short telomeres.

Air pollution exposure by pregnant mothers leads to pro-oxidant and pro-inflammatory environments. The pro-oxidant environment is related to the shortening of telomere. Mothers that inhale promethium while pregnant risk exposing a fetus to it as its ability to pass through the placenta (Perera et al., 2018; Bov et al., 2019). Experimental evidence by Moller et al. (2014) shows air pollution expose one to oxidative stress. Studies have also shown nitrogen dioxide from gas stoves and tobacco smoking exposures by pregnant mothers were related to shortening of Telomere length in samples of blood collected from children of five to twelve years old. (Clemente et al., 2019)

Arranging of telomere length at infancy and maturity was oppositely related to exposure at birth to residential traffic. One is that its explanation of the effect of childhood stress on telomers is narrow and majors only on oxidative stress as a determinant of telomere length. Another weakness is that the participants also spent time during daily commutes where they risk more exposure to air pollution from their residential places to occupational addresses. Finally, the relevance of associations between newborn telomere length and environmental factors can be challenging to foresee, and several other factors could also influence telomeres.

5. Childhood adversities stress

The by Antti-Jussi et al. (2021) discusses childhood adversities' relationship to telomere length. It confirmed earlier results of an inverse relationship between telomere leukocyte length and childhood. Childhood adversities were assessed in three ways.

First, the loss of either parent or both parents before 16 years of age was considered adversity and a possible cause of stress.

Second, the childhood socioeconomic level of both parents was based on the level of education of the parents.

Third, questionnaires were issued to participants with yes/no answers to different questions such as; unemployment in the family, alcoholism by either parent, divorce, bullying at school, chronic illness, mental health problem, and disability. The study results proved that the length of telomere was contrarily related to age. Also, shorter leukocyte telomere length was more where the score on the adversities questionnaire was high (having a lot of yes). Childhood effect on telomere at later stages of growth of puberty and adulthood remained remarkable after adjusting psychosis, depression, alcoholism, and anxiety disorder diagnosis for the past 12 months. The weaknesses of the study were that stress alone in itself was not significant enough but emotional and behavioral responses were necessary.

A significant effect on telomere cannot be caused by one stressor (Puterman et al., 2016). The study concludes that stress during childhood may promote telomere shortening which is significant in adulthood. This is backed by Eli et al. (2015). The study suggests that stress over one's lifespan promotes accelerated aging and early disease resulting from the shortening of telomeres. The study examined 4598 individuals, both male and female in the US Health and Retirement Study. Only one adverse effect on telomere length was not corresponding. Lifetime cumulative adversity showed a prediction of 6% higher odds of short telomere length., while in adjusted models, the presence of each extra adversity predicted 11% more odds of telomeres shortening. It concludes that later adulthood and telomere length are significantly affected by childhood stress.

6. Conclusion

Stress is not a monolith, and several other external factors during childhood must be considered that may affect telomeres. The study by Angela et al. (2014) gives the most accurate and detailed explanation of childhood stress association with telomeres. Eli et al. (2015) focus more on lifetime cumulative adversity overlooking childhood stress.

For oxidative stress during childhood and its effect on telomeres, the study by Rebecca et al. (2021) gives the most compressive description.

7. References

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