

UC Irvine

UC Irvine Previously Published Works

Title

Prenatal programming of newborn and infant telomere length

Permalink

<https://escholarship.org/uc/item/187866jv>

Journal

European Journal of Psychotraumatology, 3(0)

ISSN

2000-8198 2000-8066

Authors

Entringer, Sonja
Epel, Elissa S
Lin, Jue
[et al.](#)

Publication Date

2012-09-10

DOI

10.3402/ejpt.v3i0.19477

Copyright Information

This work is made available under the terms of a Creative Commons Attribution License, available at <https://creativecommons.org/licenses/by/4.0/>

Peer reviewed

EUROPEAN JOURNAL OF

PSYCHO-



TRAUMATOLOGY

Supplement 1, 2012



Effects of Traumatic Stress
Molecular and Hormonal Mechanism

Abstracts from 42nd Annual Conference
New York, September 11–14, 2012

**42nd ANNUAL CONFERENCE
ON EFFECTS OF
TRAUMATIC STRESS**

**MOLECULAR AND
HORMONAL MECHANISMS**

Program chair: Tom Hildebrandt

President: Rachel Yehuda

Editor: Miranda Olf

**New York
September 11–14, 2012**

ISPNE ABSTRACT BOOK

SYMPOSIUM

Sonja Entringer, Elissa S. Epel, Jue Lin, Claudia Buss, Elizabeth H. Blackburn, Hyagriv N. Simhan, Pathik D. Wadhwa. University of California, Irvine, CA, USA

Prenatal programming of newborn and infant telomere length

Rationale/statement of the problem: Substantial evidence suggests conditions in intrauterine life may play a critical role in subsequent health and disease susceptibility related outcomes (i.e., the concept of fetal or developmental programming of health and disease). The elucidation of biological mechanisms underlying these effects is an area of active investigation. We suggest that telomere biology may represent a novel mechanism underlying the effects of a disparate set of suboptimal intrauterine exposures on various health and disease risk phenotypes. From an evolutionary-developmental perspective, energy substrate availability (i.e., nutrition) and challenges that have the potential to impact the structural or functional integrity and survival of the organism (i.e., stress) likely represent the most important environmental considerations underlying natural selection and developmental plasticity. Maternal stress and nutrition in pregnancy therefore represent attractive candidate processes in the context of fetal programming of telomere biology. Our previous work has established an important role for prenatal stress and stress-related processes in adult telomere biology.

Methods: In two longitudinal birth cohorts, stress- and nutrition-related processes were assessed during pregnancy and telomere length (TL) was subsequently measured in newborns (cord blood) and infants (buccal cells).

Results: (1) Among the nutrition-related factors, maternal lower folate levels (an essential methyl donor) and higher triglyceride concentrations in early pregnancy were significantly and independently associated with shorter newborn TL. (2) Among psychosocial stress-related measures, higher maternal pregnancy-specific stress was associated with shorter newborn TL. (3) Maternal estrogen (E3) levels during early pregnancy were associated with longer infant TL.

Conclusion: Taken together, our findings provide the first evidence in humans that maternal nutrition and stress-related processes during pregnancy may exert a programming effect on the newborn and infant telomere biology system. *In utero* telomere biology represents a potential molecular mechanism whereby different exposures in this critical developmental period *before* birth could impact subsequent health and disease susceptibility related outcomes over the life span, including aging and longevity.

Keywords: telomere biology; telomere length; newborn; health; stress; aging

Citation: European Journal of Psychotraumatology Supplement 1, 2012, 3 - <http://dx.doi.org/10.3402/ejpt.v3i0.19477>

Idan Shalev^{1,2}, Terrie E. Moffitt^{1,2,3,4}, Avshalom Caspi^{1,2,3,4}. ¹Department of Psychology and Neuroscience, Duke University, Durham, NC, USA; ²Institute for Genome Sciences & Policy, Duke University, Durham, NC, USA; ³Department of Psychiatry and Behavioral Sciences, Duke University, Durham, NC, USA; ⁴Social, Genetic, and Developmental Psychiatry Centre, Institute of Psychiatry, King's College London, UK

Childhood trauma and telomere maintenance

Rationale/statement of the problem: Stress in early life is known to have a powerful direct effect on poor health in later life. This direct effect requires one or more underlying mechanisms that can maintain it across the life-course. It is therefore essential to characterize the biological mechanisms through which children may acquire such lasting vulnerability to disease, namely, the mechanisms of biological embedding. One plausible mechanism may lie in changes to DNA. New research suggests that stress exposures can accelerate the erosion of DNA segments called telomeres.

In the past 2 years, six studies provided support for an association between telomere length (TL) and childhood stress. Although these studies advance understanding of the link between childhood stress and TL, almost all studies have relied on adult measures of TL and retrospective recall of stress years after the stress was experienced raising important questions about the true nature of these findings. Interpretation of findings from cross-sectional studies of TL is ambiguous in light of recent longitudinal analyses of repeated TL measurements. These recent findings indicate that the temporal process of telomere erosion is more complex than initially assumed, and that repeated measures (not just length at one time point) are needed to measure true telomere erosion in individuals who are experiencing stress. Moreover, given the elapsed time between the putative stress exposure and the measurement of