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The relationship between childhood psychosocial stressor level and telomere length: a meta-analysis

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Abstract

This meta-analysis examined the association between the level of childhood psychosocial stressors and telomere length, an important health biomarker. The meta-analysis, including 27 samples and 16,238 participants, found a significant association of ~0.08 between a higher level of childhood stressors and shorter telomere length at a mean age of 42 across studies. Moderator analyses showed a trend in the direction of effect sizes being significantly larger with shorter times between the stressors and telomere measurement. Moderator analyses showed significantly higher effect sizes for studies that used a categorical method for assessing child stressor level and for assays completed with qPCR rather than with the Southern blot method. There was no significant moderation of effect size by whether study assayed leukocytes or buccal cells, whether the study assessed child stressor level by memory-based recall versus archival records, and whether the study controlled for age, sex, or additional variables. The results, focused on childhood events, add to prior findings that perceived stress and negative emotions are associated with telomere length.

Introduction

Psychosocial stressors experienced during childhood, such as maltreatment or neglect, predict an increased risk of negative health outcomes across the life span (Felitti et al., 1998; Shonkoff, Boyce, & McEwen, 2009; Shonkoff & Garner, 2012; Wegman & Stetler, 2009). Experiencing high levels of childhood psychosocial stressors is associated with the later development of depression, bipolar disorder, post-traumatic stress disorder, and substance abuse, as well as cardiovascular disease, gastrointestinal disorders, metabolic disorders, and respiratory problems (Green et al., 2010; Wegman & Stetler, 2009). High levels of childhood psychosocial stressors are associated with an increased risk of premature death (Brown et al., 2009). Stressors experienced during early developmental windows may have epigenetic effects and enduring influences on biomarkers and nervous and immune system functioning (Shonkoff & Garner, 2012). For example, high levels of childhood stressors alter physiologic, cellular, and immune stress responses (Drury et al., 2014).

Telomere length may link childhood psychosocial stressors with later health developments. Telomeres are a biomarker associated with various aspects of health (Rode, Nordestgaard, & Bojesen, 2015). Telomeres are the nucleoprotein complexes at the end of chromosomes that preserve genetic information, regulate cellular replicative capacity, and prevent end-to-end fusion (Blackburn, Greider, & Szostak, 2006). Telomere length erosion can occur through repeated cell division and through exposure to oxidative stress and inflammation (O’Donovan et al., 2011). The general trend is for telomeres to shorten with aging; however, telomere biology is dynamic (Blackburn, Epel, & Lin, 2015) and telomeres can lengthen as well as shorten over time (Epel, 2012). Short telomere length is associated with or predicts many of the common diseases of aging, such as cardiovascular disease, stroke, cancer, vascular dementia, osteoporosis, obesity and diabetes (Blackburn et al., 2015; Rode et al., 2015), and all-cause mortality (Rode et al., 2015).

Systematic reviews (Niess & Kirkengen, 2015; Oliveira, et al., 2016) of studies of stressors and telomere length suggest that greater exposure to stressors may be associated with shorter telomeres. However, the evidence is mixed, with not all studies finding a significant relationship between exposure to stressors and telomere length. Exposure to stressors, which are events, may lead to greater perceived stress, a psychological phenomenon. Meta-analyses of effect sizes of the relationship across studies of perceived stress and telomere length reported a significant meta-analytic association (Schutte & Malouff, 2014; Mathur et al., 2016). To date no meta-analysis of effect sizes of the relationship between childhood psychosocial stressors and telomere length across studies has been published. Such a meta-analytic investigation could provide an overall effect size of relationship between...
chose these potential moderators because virtually every study not feasible to examine specific measures as potential moderators. These moderator variables included (1) type of tissue assayed, (2) whether the level of stressors was measured as categorical or continuous, (3) what type of assay was used, (4) whether childhood stressor level was based retrospectively on memory of events or not, whether (5) age and (6) sex were controlled, (7) whether additional variables were controlled, and (8) whether telomere length was log-transformed due to non-normal distribution of data.

Literature Search
We systematically searched PsychINFO, Pubmed, EMBASE, CINAHL Complete, Cochrane Central, Research Gate, and Google Scholar to identify all articles, completed at any time, reporting on childhood psychosocial stressors and telomere length. The search included high as well as low status and did not focus on children in very low SES families. Thus, SES was not a pure measure of psychosocial stress level. We also excluded reports that provided the same results as a report we included in the meta-analysis; Brody, Yu, Beach & Philibert (2015) and Révész, Milaneschi, Terpstra & Penninx (2016). Three studies fit the inclusion criteria but did not provide the data needed for meta-analysis (Zhang et al., 2014; Robles, Carroll, Bai, Reynolds, Esquivel, & Repetti, 2016; Theall et al., 2013). We attempted unsuccessfully to obtain the needed information from the corresponding authors.

Coding process
Coding involved recording three types of information relating to effect size: r or some other statistic that indicates effect size, N for the key analysis, and the direction of the association between stressor level and telomere length. Coding also included entering data for each study about the possible moderators of effect size. When studies reported results for more than one measure of level of childhood stressors, we calculated the average effect size across the measures.

Two of us completed the initial coding together. Then a third...
member of our research group independently coded the effect sizes and moderators. A comparison of the independent coding showed agreement on 95% of the decisions. For all disagreements regarding coding, we made final decisions by consensus.

**Relevant studies identified**

The literature search retrieved 2,122 potentially relevant articles. Figure 1 shows the study selection process that resulted in the 27 samples that met all inclusion criteria.

**Meta-analytic methods**

We report effect sizes below as $r$. When studies reported standardized beta weights with other variables included in the regression, we used the results that controlled for sex and age and as few other variables as possible. It is sensible to include age and sex controlled effect sizes because studies have found that women tend to have longer telomeres (Gardner et al., 2014) and that younger individuals tend to have longer telomeres (Marioni et al., 2016). Most of the studies did control for those variables, either statistically or by comparing high and low stressor groups that were very similar with regard to the variables.

The Comprehensive Meta-Analysis Program (Borenstein, Hedges, Higgins & Rothstein, 2014) calculated the overall weighted effect size. We used a random effects model in order to allow for between-studies variation. The Q statistic assessed effect-size homogeneity across studies. Finally, trim and fill method and fail-safe $N$ assessed the impact of possibly missing studies.

**Results**

Table 1 shows the key characteristics of each included sample. Figure 2 shows graphically the effect size for each sample. The overall meta-analytic association between level of childhood psychosocial stressors and telomere length, with 27 samples, including 16,238 total participants, was $r=-0.082$ (95%CI $-0.122$, $-0.042$), $P<0.001$. There was a significant level of heterogeneity among effect sizes, $Q(26)=109$, $P<0.001$, $I^2=76$, suggesting the possibility of finding moderators of effect size.

The fail-safe $N$ was 338, indicating that 338 studies with 0 effect size would be needed to reduce the overall effect size to a nonsignificant level. Duval and Tweedie’s trim-and-fill statistic indicated that the overall effect size was not significantly affected by the results of small $N$ studies and that no adjustment in effect size was needed. See Figure 3 for the funnel plot of effect sizes.

The mean age at telomere measurement in the studies was 42 years. The only directional hypothesis regarding potential moderators of effect size, that the younger the participants at measurement of telomere length, the higher the effect size, showed a trend towards significance, slope estimate $=0.002$ (95%CI $0.000$, $0.004$),
Table 1. Descriptive data, including effect size, for studies in the meta-analysis.

| Author                  | Childhood psychosocial stressor/s | No. | Mean age at telomere collection | Memory-based retrospective assessment of stressor | TL cell type | TL assay type | Categorical stressor | Age controlled | Sex controlled | Other variables controlled for | Log transformed | r    |
|-------------------------|-----------------------------------|-----|---------------------------------|-----------------------------------------------|--------------|--------------|--------------------|---------------|---------------|-------------------------------|----------------|------|
| Asok et al. (2015)      | Neglect, family violence etc     | 89  | 4.9                             | No                                           | Buccal mucosa | PCR          | Yes                 | No            | Yes           | Yes                           | No             | -0.22*|
| Beach et al. (2014)     | Life stress                       | 183 | 21.8                            | Yes                                          | Leukocyte    | PCR          | No                  | No            | No            | No                            | No             | -0.04|
| Bersani et al. (2016)   | Abuse, general trauma            | 76  | 34.6                            | Yes                                          | Leukocyte    | PCR          | No                  | Yes           | Yes           | All same sex                  | No             | -0.43**|
| Chen et al. (2014)      | Abuse, neglect etc                | 20  | 35.9                            | Yes                                          | Leukocyte    | PCR          | No                  | Yes           | Yes           | No                            | No             | -0.13|
| Chen et al. (2014)      | Abuse, neglect etc                | 20  | 35.9                            | Yes                                          | Leukocyte    | PCR          | No                  | Yes           | Yes           | No                            | No             | -0.61*|
| Drury et al. (2012)     | In institutional care             | 100 | 8.4                             | No                                           | Buccal mucosa | PCR          | No                  | Yes           | Yes           | Yes                           | No             | -0.05|
| Drury et al. (2014)     | Adverse events                   | 80  | 10.2                            | Yes                                          | Buccal mucosa | PCR          | Yes                 | Yes           | Yes           | Yes                           | No             | -0.28**|
| Glass et al. (2010)     | Physical abuse, sexual abuse     | 1090| 47.8                            | Yes                                          | Leukocyte Southern blot |             | Yes                  | No            | No            | No                            | Yes            | .002 |
| Jockzik et al. (2014)   | Interparent violence, physical abuse etc | 677 | 29.0                            | Yes                                          | Leukocyte    | PCR          | No                  | All same age  | Yes           | Yes                           | No             | -0.01|
| Kananen et al. (2010)   | Parental substance abuse/mental illness etc | 974 | 40.8                            | Yes                                          | Leukocyte    | PCR          | No                  | Yes           | Yes           | Yes                           | No             | -0.09*|
| Kiecolt-Glaser et al. (2011) | Abnormality                   | 132 | 65.9                            | Yes                                          | Leukocyte Southern blot |             | Yes                  | Yes           | Yes           | Yes                           | No             | -0.06|
| Koffer et al. (2016)    | Abuse, neglect etc                | 58  | 71.9                            | Yes                                          | Buccal mucosa | PCR          | No                  | Yes           | Yes           | No                            | No             | 0.21 |
| Koffer et al. (2016)    | Abuse, neglect etc                | 62  | 76.2                            | Yes                                          | Buccal mucosa | PCR          | No                  | Yes           | Yes           | No                            | No             | 0.12 |
| Lewandowski et al. (2010) | Childhood adversity              | 87  | 28.6                            | Yes                                          | Blood        | PCR          | Yes                 | No            | All same sex | No                            | No             | -0.41**|
| Mason et al. (2015)     | Physical abuse, sexual abuse     | 1130| 45.5                            | Yes                                          | Leukocyte    | PCR          | No                  | Yes           | All same sex | No                            | Yes            | -0.01|
| O’Donovan et al. (2011) | Physical abuse, physical neglect etc | 41  | 30.2                            | Yes                                          | Leukocyte    | PCR          | No                  | Yes           | No            | No                            | No             | -0.42*|
| Ostert et al. (2016)    | Parental illness/loss, separated from home etc | 324 | 57.0                            | Yes                                          | Leukocyte    | PCR          | No                  | All same age  | All same sex | No                            | No             | -0.02|
| Sarlahinen et al. (2014) | Absent parent                    | 1406| 61.5                            | No                                           | Leukocyte    | PCR          | Yes                 | Yes           | Yes           | Yes                           | Yes            | -0.05|

Continue on next page.
P=0.068, two-tailed. The association would be significant at P=0.034 with a one-tailed test. The other moderator analyses were all categorical comparisons. Table 2 shows the results. Two variables showed significant moderation of effect size: Studies that compared groups, e.g., being abused or not, showed higher associations between level of childhood stressor and telomere length than studies that treated stressor level as a continuous variable. Also, studies that used qPCR had higher effect sizes than studies that used Southern Blot. If we apply a Bonferroni correction to control for alpha inflation in the analyses of the eight categorical variables, these findings would not meet the adjusted P standard of 0.05/8 or 0.006.

Table 1. Continued from previous page.

| Author                     | Childhood psychosocial stressor/s | No. | Mean age at telomere collection | Memory-based retrospective assessment of stressor | TL cell type | TL assay type | Categorical stressor | Age controlled | Sex controlled | Other variables controlled for | Log transformed | r   |
|----------------------------|----------------------------------|-----|---------------------------------|-------------------------------------------------|--------------|---------------|----------------------|----------------|----------------|---------------------------------|----------------|-----|
| Schaaks et al. (2015)      | Adverse events, trauma           | 496 | 70.6                            | Yes                                             | Leukocyte    | PCR           | Yes for adverse events; No for trauma | Yes            | Yes            | Yes                             | Yes            | No  | 0.32** |
| Shale et al. (2013)        | Family violence, physical abuse etc | 236 | 10.0                            | No                                              | Buccal mucosa| PCR           | Yes                  | All same age | Yes            | Yes                             | Yes            | No  | -0.05  |
| Surtees et al. (2011)      | Emotional abuse, 444I physical abuse etc | 62.0 | Yes                             | Leukocyte                                      | PCR           | No            | Yes                  | No             | No             | No                             | Yes            | No  | -0.01  |
| Tyrka et al. (2010)        | Physical neglect, emotional neglect | 31  | 26.9                            | Yes                                             | Leukocyte    | PCR           | Yes                  | No             | No             | No                             | Yes            | No  | -0.31  |
| Tyrka et al. (2015)        | Parental loss, separation from family | 179 | 31.0                            | Yes                                             | Leukocyte    | PCR           | Yes                  | No             | Yes            | Yes                             | Yes            | No  | -0.09  |
| van Ockenburg et al. (2015) | Parental loss, separation etc | 445 | 55.5                            | Yes                                             | Leukocyte    | PCR           | Yes                  | No             | Yes            | Yes                             | Yes            | Yes | -0.00  |
| Verhoeven et al. (2015)    | Emotional neglect, emotional abuse etc | 296 | 41.8                            | Yes                                             | Leukocyte    | PCR           | Yes                  | Yes            | Yes            | No                             | Yes            | No  | -0.02  |
| Zalli et al. (2014)        | Parental loss, separation, household substance use etc | 434 | 63.2                            | Yes                                             | Leukocyte    | PCR           | Yes                  | Yes            | Yes            | Yes                             | Yes            | No  | -0.01  |

*Effect size based on both abused and neglected. **Effect size based on age-adjusted results. * Used sociodemographic adjustment results. ** Used sociodemographic adjustment results for emotinal neglect, emotional abuse, physical abuse, sexual abuse. *P<0.05, **P<0.001.

Figure 2. Graphical representation of effect size for each sample.
Discussion and Conclusions

The meta-analysis found a small but significant association (-0.08) between level of childhood psychosocial stressors and telomere length, across 27 samples that included 16,238 participants. The association was significant regardless of whether stressor level was based on recall or more objective documentation, and regardless of whether the cells assayed for telomere length were leukocytes or buccal. Childhood stressors can have a long-term impact on telomere length, as indicated by the significant association between exposure to childhood stressors and telomere length at a mean age of 42 years for participants included in the present meta-analysis.

The results provide a possible mediational explanation for the finding that psychosocial stressors experienced during childhood predict negative health outcomes in adulthood (Felitti et al., 1998; Shonkoff & Garner, 2012; Wegman & Stetler, 2009). There are a number of biological and behavioural pathways that early trauma affects, such as inflammation and changes in health behaviours, and these also interact with telomere length; thus the causal factors linking early trauma and later disease are likely due to a variety of inter-related factors. (Danese & McEwens, 2012). Telomere length appears to be one of the causal factors, as recent mendelian genetic studies of telomere length have shown direct prediction of earlier onset of certain diseases of aging (Codd et al., 2013; Zhan et al., 2015).

The findings of the present meta-analysis extend findings of previous research on psychological states and telomere length in focusing on the relationship between actual events experienced in childhood and later telomere length. Prior meta-analyses reported significant associations between perceived stress and telomere length (Mathur et al., 2016; Schutte & Malouff, 2014). Prior meta-analyses also found significant associations between anxiety levels and telomere length (Malouf & Schutte, in press) and between depression and telomere length (Schutte & Malouff, 2015).

Childhood psychosocial stressors predict telomere shortening, and negative psychological states (perceived stress, anxiety, and depression) may operate as mediators linking stressors and telomere shortening. Some studies have found that recent psychosocial stressors in adults are also associated with shorter telomeres (e.g., Schaeck et al., 2015). Telomere functioning is dynamic (Blackburn et al., 2015) and shortened telomeres may recover as time passes after exposure to a stressor (Verhoeven et al., 2015), and thus we predicted that time would moderate effect size. We found a moderation trend consistent with this view. However, remarkably, childhood stressors were still significantly associated with shortened telomeres decades later. It is unknown whether childhood psychosocial stressors are more or less associated with telomere length than stressors experienced by adults, but it is possible that childhood stressors have more impact because childhood is a critical period of development of biological systems (Shonkoff & Garner, 2012) or because of the limited coping ability of children. Studies have found exposure to other environmental factors, such as pesticides, to be associated with telomere length (Hou et al., 2013). The present findings add meta-analytic results for early-life psychosocial environmental factors. One interesting meta-anal-

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Table 2. Categorical moderator analysis.

| Moderator | k | r | CI 95% | Homogeneity Analysis |
|-----------|---|---|-------|----------------------|
|           | Lower | Upper | P    | Q   | df | P    |
| Memory-based retrospective assessment of stressor | | | | | | |
| No        | 4   | -0.06 | -0.1 | 0.01 | 0.02 | 2.57 | 3 | 0.46 |
| Yes       | 22  | -0.09 | -0.14| -0.04| <0.001 | 105.96 | 21 | <0.001 |
| TL cell type | 0.04, P=0.84 | | | | | |
| Buccal    | 6   | -0.06 | -0.19| 0.07 | 0.39 | 12.55 | 5 | 0.03 |
| Leukocyte | 20  | -0.07 | -0.11| -0.03| <0.001 | 81.97 | 19 | <0.001 |
| TL assay type, Q(1)=4.49, P=0.03 | | | | | | |
| Southern blot | 2 | 0.01 | -0.07| 0.08 | 0.90 | 0.64 | 1 | 0.42 |
| qPCR      | 25  | -0.10 | -0.13| -0.05| <0.001 | 106.77 | 24 | <0.001 |
| Categorical stressor Q(1)=4.39, P=0.04 | | | | | | |
| Yes       | 11  | -0.14 | -0.23| -0.06| <0.001 | 57.19 | 10 | <0.001 |
| No        | 16  | -0.04 | -0.08| -0.00| 0.04 | 38.59 | 15 | 0.001 |
| Age controlled Q(2)=4.95, P=0.08 | | | | | | |
| All same age | 4 | -0.02 | -0.07| 0.03 | 0.34 | 0.35 | 3 | 0.95 |
| No        | 6   | -0.15 | -0.28| -0.02| 0.03 | 21.00 | 5 | <0.001 |
| Yes       | 17  | -0.09 | -0.14| -0.03| <0.001 | 85.81 | 16 | <0.001 |
| Sex controlled Q(1)=0.32, P=0.85 | | | | | | |
| All same sex | 6 | -0.10 | -0.18| -0.01| 0.02 | 30.19 | 5 | <0.001 |
| No        | 5   | -0.11 | -0.23| 0.02 | 0.10 | 11.34 | 4 | 0.02 |
| Yes       | 16  | -0.08 | -0.13| -0.02| 0.01 | 63.91 | 15 | <0.001 |
| Other variables controlled for Q(1)=0.89, P=0.35 | | | | | | |
| No        | 15  | -0.06 | -0.12| -0.01| 0.02 | 44.62 | 14 | <0.001 |
| Yes       | 12  | -0.10 | -0.17| -0.04| <0.001 | 61.35 | 11 | <0.001 |
| Log transformed Q(1)=3.39, P=0.07 | | | | | | |
| Yes       | 4   | -0.04 | -0.08| 0.00 | 0.03 | 4.29 | 3 | 0.23 |
| No        | 23  | -0.10 | -0.15| -0.05| <0.001 | 104.67 | 22 | <0.001 |

1Shales et al. (2014) excluded because study used mixed methods; 2Levandowski et al. (2016) excluded because study used "blood."
lytic moderator finding involved the significantly greater effect size for comparison of extreme groups on level of childhood stressors than for correlational studies with various levels of stressors. Similarly, a meta-analysis of the association between anxiety level and telomere length found that analyses of extreme groups showed much greater effect sizes than correlational studies, although the difference was not significant (Malouff & Schutte, 2016). It could be that only extreme levels of childhood psychosocial stressors have long-term effects on telomere length.

Studies that used qPCR assays had significantly higher effect sizes than Southern blot studies. Because only two studies in the meta-analysis used Southern blot, that finding may be a statistical fluke.

The moderator results are best viewed as suggestive. First, with only 27 samples included, the moderator analyses had limited power to identify significant differences. Second, moderator analyses are always quasi-experimental — no one randomly assigned some studies to use one method and other studies to use another. Third, the statistical significance of some moderators in this meta-analysis varies with how conservative one wants to be regarding using one-tailed tests and controlling for alpha inflation.

Future research on child psychosocial stressors and telomere length might systematically compare different types of psychosocial stressors and examine the role of possible mediators and moderators, including potential buffers such as social support. In addition, it will be important to examine in more depth the characteristics of the stressors and symptoms of distress. This type of research will help identify both predictors of vulnerability and resilience to the lifelong effects of severe childhood stressors.

Figure 3. Funnel plot of standard error by effect size (Fisher’s Z).

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