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Risky Family Processes Prospectively Forecast Shorter Telomere Length Mediated Through Negative Emotions

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Objective: This study was designed to examine prospective associations of risky family environments with subsequent levels of negative emotions and peripheral blood mononuclear cell telomere length (TL), a marker of cellular aging. A second purpose was to determine whether negative emotions mediate the hypothesized link between risky family processes and diminished telomere length. **Method:** Participants were 293 adolescents (age 17 years at the first assessment) and their primary caregivers. Caregivers provided data on risky family processes when the youths were age 17 years, youths reported their negative emotions at age 18 years, and youths' TL was assayed from a blood sample at age 22 years. **Results:** The results revealed that (a) risky family processes forecast heightened negative emotions ($\beta = .316, p < .001$) and diminished TL ($\beta = -.199, p = .003$) among youths, (b) higher levels of negative emotions forecast shorter TL ($\beta = -.187, p = .012$), and (c) negative emotions served as a mediator connecting risky family processes with diminished TL (indirect effect = -0.012 , 95% CI $[-0.036, -0.002]$). **Conclusions:** These findings are consistent with the hypothesis that risky family processes presage premature cellular aging through effects on negative emotions, with potential implications for lifelong health.

Keywords: adolescents, family process, health, negative emotions, telomere length

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A growing body of research has tested the hypothesis that family processes such as emotional climate and parent–child relationship quality across childhood and adolescence may contribute to chronic diseases later in life (Shonkoff, Boyce, & McEwen, 2009). This *risky family* model offers a psychosocial account of the impact of stress during childhood and adolescence on health later in life (Repetti, Taylor, & Seeman, 2002). It posits that some families confer risk for later health problems through chronically conflicted relationships, characterized by a lack of warmth, a lack of emotional support, and an unpredictable home environment. These family dynamics trigger a cascade of psychological vulnerabilities, including deficits in the regulation of emotions and the propensity to compensate for those deficits with health-compromising behaviors. Risky family dynamics also tend to increase reactivity of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS; El-Sheikh & Erath, 2011; Repetti et al., 2002). Frequent activation of these circuits triggers the release of

hormones such as cortisol, and catecholamines such as epinephrine and norepinephrine. Over time, these stress mediators may lead to wear and tear on bodily systems and to subsequent health problems (McEwen, 1998). This observation suggests that chronic stress inflicted by risky family environments may also accelerate aging processes.

The present study was designed to advance understanding of the association between risky family processes and health status by testing hypotheses involving prospective pathways among risky family processes—low parental emotional support, high levels of parent–youth conflict, and unpredictable home environments—negative youth emotions, and cellular aging (i.e., telomere length [TL] in peripheral blood mononuclear cells). Telomeres are DNA-protein complexes that cap the ends of chromosomes. Telomere length can provide a window into lifelong acceleration of aging processes (Shalev, Entringer, et al., 2013) as they shorten over time, and their shortness predicts subsequent morbidity and early mortality (Rode, Nordestgaard, & Bojesen, 2015). Specifically, TL may be viewed from a lifespan perspective because it reflects, in part, the cumulative number of cell divisions that have occurred and the long-term biochemical environment. Many studies find a negative association between age and TL (Monaghan & Haussmann, 2006), but substantial variation in TL exists among age-matched individuals. This suggests that factors other than chronological age affect telomere shortening. Shorter TL has been associated cross-sectionally with cardiovascular disease, cancer, stroke, diabetes, and autoimmune disease (D'Mello et al., 2015; Haycock et al., 2014) in older adults. Although TL in these conditions could be only an indicator of ongoing disease, other

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evidence suggests that TL predicts the likelihood of disease in the future despite current health status (Cohen et al., 2013). Prospective studies indicate that short TL predicts the occurrence of cancer (Ma et al., 2011; Weischer et al., 2013) and hypertension (Yang et al., 2009), as well as all-cause mortality (Rode et al., 2015).

Importantly, a growing body of evidence, some of which was obtained with adolescents, has demonstrated that chronic psychological stress (Epel et al., 2004), caregiving strain (Kiecolt-Glaser et al., 2011), social deprivation (Drury et al., 2012), family violence (Drury et al., 2014), neighborhood disorder (Theall, Brett, Shirliff, Dunn, & Drury, 2013), and exposure to extrafamilial violence (Shalev, Moffitt, et al., 2013) are associated with diminished TL. This suggests that TL may be useful in determining the ways in which stress associated with risky family processes forecasts biological aging at the cellular level. Furthermore, adverse childhood events that also have been found to be associated retrospectively with TL include maltreatment, trauma, parental death, familial mental illness, and parental unemployment (reviewed in Price, Kao, Burgers, Carpenter, & Tyrka, 2013; Shalev, 2012). One prospective study found that exposure to violence across time was associated with accelerated telomere erosion in children from age 5 to age 10 years (Shalev, Moffitt, et al., 2013), suggesting that the process of accelerated cellular aging may begin early in life. Other researchers have suggested that this process may already begin in utero (Entringer et al., 2013; Marchetto et al., 2016). The present analysis was designed to extend this research by examining prospective associations between risky family environments and TL. Specifically, exposure to risky family processes during adolescence, assessed at age 17, is hypothesized to forecast shortened TL during young adulthood, at age 22.

A second hypothesis proposes that negative emotions, operationally defined as elevated levels of anger and depressive symptoms, potentially will serve as a mediator connecting exposure to a risky family environment with shortened TL. One of the most prominent legacies of risky family environments concerns problems with emotion regulation, particularly with respect to worry, irritability, and reactivity (Brody, Yu, Beach, et al., 2014; Davies, Winter, & Cicchetti, 2006). These negative emotional processes can activate stress-related physiological systems (Brosschot, Gerin, & Thayer, 2006) that, over time, may contribute to the shortening of telomeres (Shalev, 2012). Thus, this study included the hypothesis that living in a risky family environment at age 17 will forecast elevated negative emotions at age 18, and that heightened negative emotions will serve as a mediator connecting risky family processes with TL.

Method

Participants

A total of 496 youths were recruited randomly from public school lists in six rural counties. Youths were enrolled in the study when they were about 17 years of age and provided self-report data at age 18 years. The data on TL were obtained from blood samples when the youths were 22 years of age. Data were collected in the context of a family-based prevention study (Brody, Yu, Chen, Kogan, & Smith, 2012). Assignment to the prevention or control condition was controlled in all data analyses. At Wave 1, median household gross monthly income was \$2,016.00 ($SD = 4,353.86$)

and mean per capita gross monthly income was \$887.54 ($SD = 1,578.98$). Although youths' caregivers worked an average of 38.5 hours per week, 42% of the families lived below federal poverty standards and another 15% lived within 150% of the poverty threshold; they could be described as working poor (Boatright, 2005).

Procedures

Families were contacted and enrolled in the study by community liaisons who resided in the counties where the participants lived. The community liaisons were African American community members, selected on the basis of their social contacts and standing in the community, who worked with the researchers on participant recruitment and retention. At all data collection points, parents gave written consent to their own and to minor youths' participation, and youths gave written assent or consent to their own participation. Each family was paid \$100 after each assessment. To enhance rapport and cultural understanding, African American university students and community members served as field researchers to collect data. During each assessment, one home visit lasting 2 hr was made to each family. At the home visit, self-report questionnaires were administered to the primary caregiver and youth on a laptop computer in a private place in each home. All procedures were approved by the Institutional Review Board at the first author's university.

Measures

Risky family environment. The risky family environment construct was measured at age 17 by using three observed variables obtained from parent ratings: parent-child conflict, chaos in the home, and parent support (Brody, Yu, Beach, et al., 2014; Brody, Yu, Chen, & Miller, 2014; Brody, Yu, Miller, & Chen, 2015). Parent-child conflict was assessed using a seven-item version of the Ineffective Arguing Inventory (IAI; Kurdek, 1994) adapted for use with parents and children. Parents rated statements about the conflicts they had with their children on a scale ranging from 0 (*disagree strongly*) to 4 (*agree strongly*), $\alpha = .79$. Examples include, "You and your child's arguments are left hanging and unsettled" and "You and your child go for days being mad at each other." Chaos in the home was assessed by the 15-item Confusion, Hubbub, and Order Scale (CHAOS; Matheny, Wachs, Ludwig, & Phillips, 1995). Parents rated as *true* (1) or *false* (0) statements designed to indicate the degree of disorganization and unpredictability in their home environments, $\alpha = .75$. Examples include, "There is often a fuss going on at our home" and "No matter what our family plans, it usually doesn't seem to work out." Parental support was assessed using the five-item emotional support subscale from the Family Support Inventory (FSI; Wills, Vaccaro, & McNamara, 1992). Parents rated statements about the support they provided to their children on a scale ranging from 0 (*not at all true*) to 5 (*very true*), $\alpha = .80$. Examples include, "My child can trust me as someone to talk to" and "When my child feels bad about something, I will listen."

Negative emotions. The negative emotions construct, measured at age 18, comprised the two observed variables of depression and anger. Self-reports of depressive symptoms were obtained using the Center for Epidemiologic Studies Depression scale

(CES-D; Radloff, 1977), which is widely used with community samples. Youths rated each of 20 symptoms on a scale ranging from 0 (*rarely or none of the time*), to 3 (*most or all of the time*), $\alpha = .85$. Anger was assessed using the 15-item State Anger subscale taken from the State-Trait Anger Expression Inventory that Spielberger and associates (Spielberger, Jacobs, Russell, & Crane, 1983) developed. Youths were asked about their feelings over the past 3 months and to rate discrete emotions (e.g., "I am furious"; "I feel angry") on a scale ranging from 1 (*always*) to 5 (*never*), $\alpha = .93$.

Telomere length. When the participants were age 22, certified phlebotomists went to each one's home to draw a blood sample. After the blood was drawn into serum separator tubes, it was frozen and delivered to the Psychiatric Genetics Lab at the University of Iowa for assaying. The measurement of TL involved several steps. Peripheral blood mononuclear cells (PBMC; e.g., lymphocytes and monocytes) were generated using Ficoll separation (see Philibert, Beach, & Brody, 2012). DNA was then extracted from PBMCs using Qiagen's QIAamp DNA Miniprep Kit (Valencia, CA) according to the manufacturer's instructions. Relative telomere (T) to single-copy-gene standard (S) ratios (T/S) for each sample were calculated using a minor adaption of the improved quantitative polymerase chain reaction (PCR) method that Cawthon (2009) developed. In brief, 40 ng of DNA were placed robotically into 384-well optical PCR plates. The resulting DNA was then amplified using a set of primers specific for either telomeric sequence or a single-copy-gene standard (albumin). The primers for the telomeric sequence are forward TGTTAGGTATCCCTATCCTATCCCTATCCCTATCCCTAACA and reverse ACAC-TAAGGTTTGGGTTTGGGTTTGGGTTTGGGTTAGTGT. For the single-copy-gene standard, albumin, the primers are forward GCCCGGCCCGCCGCGCCCGTCCCGCCGGAAGCATGGTCGCCTGTT and reverse CGGCGGCGGGCGGCGCGGGCTGGGCGGAAATGCTGCACAGAATCCTTG. The telomeric primers were used at a final concentration of 900 nM each, whereas the single-copy-gene standard primers were used at a final concentration of 500 nM each. In both cases, thermocycling was performed on an ABI 7900HT Genetic Analysis System (Life Technologies, Carlsbad, CA). SybrGreen® Power Master Mix (Life Technologies, Carlsbad, CA) was used to supply the buffer, DNA polymerase, and deoxynucleotides. The cycling parameters for each of the amplification stages were as follows: Stage I; 95° for 10 min; Stage II, two cycles of 15 sec at 95°, 15 sec at 49°, and 10 sec at 62°; and Stage III, 38 cycles of 15 sec at 95°, 15 sec at 62°, and 10 sec at 72°. The resulting data were cleaned, and quadruplicate replicates for each T or S value were used to calculate T/S ratios. The intraassay coefficients of variation (CVs) were 3.6% (T), 2.0% (S), and 3.0% (T/S ratio). The resulting ratios were normalized to the geometric mean of a set of three internal LC DNA standards plated 8 times on each plate.

Control variables. Youth gender, intervention status, family socioeconomic risk at youth age 17, and youth BMI and age at the time of the blood draw ($M = 22$, $SD = 1.00$, range = 20–25) were controlled in all analyses. Six dichotomous variables (1 = *present*, 0 = *absent*) formed a socioeconomic risk index (see Evans, 2003; Kim & Brody, 2005; Rutter, 1993): family poverty based on federal guidelines, primary caregiver unemployment, receipt of

Temporary Assistance for Needy Families, primary caregiver single parenthood, primary caregiver education level less than high school graduation, and caregiver-reported inadequacy of family income. The scores were summed to form the index ($M = 1.95$, $SD = 1.38$, range = 0–6). Intervention status and gender were dummy coded. Intervention participants were coded 1 and control participants were coded 0; male participants were coded 1 and female participants were coded 0. Each participant's weight and height were recorded and used to calculate body mass index (weight in kilograms divided by the square of height in meters).

Plan of Analysis

The hypothesized mediation model was tested with structural equation modeling (SEM) executed using Mplus 7.4 (Muthén & Muthén, 1998–2012) with full information maximum likelihood estimation on the data obtained at ages 17, 18, and 22. This estimation does not delete cases that are missing on endogenous variables, nor does it delete cases that are missing a variable within a wave of data collection. This method thus avoids problems, such as biased parameter estimates, that are more likely to occur if pairwise or listwise procedures are used (Acock, 2005). This analysis was performed to test the hypotheses that (a) living in a risky family environment at age 17 (measured as parent–child conflict, chaos in the home, and low parental support) would be positively associated with negative emotions at age 18 (measured as anger and depression), and negatively associated with TL at age 22; (b) negative emotions at age 18 would be negatively associated with TL at age 22; and (c) negative emotions at age 18 would serve as a mediator connecting risky family environments at age 17 with TL at age 22. In addition, nonparametric bootstrapping, which has been found to be sensitive in mediation analyses (Preacher & Hayes, 2004), was used to obtain the bias-corrected and accelerated confidence intervals (BCA) of the indirect effect for significance testing. The indirect, mediating effect was calculated 1,000 times using random sampling with replacement to build a sampling distribution.

Results

Attrition Analyses

Study hypotheses were tested with 293 youths (59% of the Wave 1 sample) who agreed to provide a blood sample from which TL was assessed at age 22. Comparisons, using independent *t* tests and chi-square tests, of the youths who provided TL data with those who did not provide them revealed one difference: The rate of missing TL data was higher for male (47.0%) than for female (36.7%) participants, $\chi^2(1) = 5.249$, $p = .022$. Therefore, gender was controlled in all analyses. An additional two-factor multivariate analysis of variance was executed to test group differences on the study variables for participants for whom TL data were or were not obtained at age 22, by gender. No significant main effects or interaction effects emerged for any study or confounder variables. The means for these comparisons are presented in the online supplementary material, in Table S1.

Hypothesis Testing

The structural model was specified with the control variables and risky family environment as the exogenous variables, not

predicted by any prior variable in the model. Negative emotions at age 18 were specified as an endogenous construct, which can be predicted by prior variables in the model, and TL at age 22 was specified as the criterion variable. Table 1 presents descriptive statistics along with bivariate correlations among the study variables. The measurement model fit the data well: $\chi^2(41) = 42.670$, $p = \text{ns}$; CFI = .996; RMSEA = .012 (0, .042). All indicators loaded on their respective constructs significantly and in the expected directions. Scatterplots illustrating the associations among the study variables—risky family environment (defined as the sum of the standardized scores for parent–child conflict, chaos in the home, and lack of parental support), negative emotions (defined as the sum of the standardized scores for depression and anger), and TL—are presented in Figure S1 in the online supplementary material.

Figure 1 presents the results of the tests of the hypothesized mediational model. The goodness-of-fit indices indicate a good fit between the proposed model and the data: $\chi^2(34) = 46.874$, $p = .070$; CFI = .951; RMSEA = .036 (0, .059). As expected, living in a risky family environment at age 17 forecast negative emotions at age 18 ($\beta = .316$, $p < .001$) and diminished TL at age 22 ($\beta = -.199$, $p = .003$). Also consistent with the study hypothesis, the results indicated that higher levels of negative emotions at age 18 forecast diminished TL at age 22 ($\beta = -.187$, $p = .012$). These results emerged with family SES risk, gender, intervention status, BMI, and age controlled. Finally, Figure 1 also shows the results of the mediational analysis. As hypothesized, the presence of the mediator, negative emotions, reduced the direct effect of risky family environments during adolescence on TL during young adulthood, from $\beta = -.199$, $p = .003$ to $\beta = -.139$, ns. The indirect effect of adolescent risky family environments on young adult TL via negative emotions was significant (indirect effect estimates = -0.012 , BCA = $-0.036 - -0.002$ with 1000 bootstrapping). The results thus support the hypothesis that negative emotions at age 18 mediated the effect of risky family environments at age 17 on TL at age 22.

Discussion

A longitudinal design was used to test hypotheses proposing that living in a risky family environment during adolescence would forecast cellular aging among African American young adults. As predicted, elevated levels of negative emotions during adolescence served as a mediator for the prospective association between life in a risky family environment and shortened TL. This is, to the authors' knowledge, the first prospective study showing that risky family environments and negative emotion are significantly associated with diminished TL. These patterns converge with evidence indicating that living in challenging family circumstances relatively early in the life span can undermine health, particularly when they contribute to habitual levels of distress characterized by negative emotions.

The mechanisms underlying the study's findings remain unclear. Navigating the challenges posed by life in a family characterized by low support, high conflict, and disorganization can be metabolically and behaviorally demanding. These experiences seem likely to cause persistent activation of stress-response systems, in particular the SNS, HPA axis, and immune system. The hormonal products of these systems, glucocorticoids and catecholamines, are elevated in youths who experience unsupportive parenting (Brody, Yu, Chen, et al., 2014; Brody et al., 2013) and can have downstream effects that could affect inflammatory processes (Miller, Rohleder, Stetler, & Kirschbaum, 2005).

Given this mechanistic analysis, it is notable that several studies in humans document significant associations among HPA axis indices, inflammation, and shorter TL (Epel et al., 2006; Kiecolt-Glaser et al., 2011; Révész et al., 2014; Tomiyama et al., 2012). Also meriting attention is evidence linking heightened cortisol activity with shorter TL in young daughters of mothers with recurrent episodes of depression (Gotlib et al., 2015). Indirect evidence that insensitive mothering may mediate the effect of maternal depression on TL can be found in Asok, Bernard, Roth, Rosen, and Dozier (2013). This research showed that sensitive, responsive parenting protected children growing up under high-

Table 1
Descriptive Statistics and Correlations Among Study Variables

Variables	1	2	3	4	5	6	7	8	9	10	11
1. Gender, male	—										
2. Intervention	-.115*	—									
Age 17											
3. SES-related risk	.004	-.013	—								
4. Parent–child conflict	-.012	.026	.109	—							
5. Chaos in the home	-.012	-.072	.051	.402***	—						
6. Parent support	.021	-.052	.107	-.476***	-.352***	—					
Age 18											
7. Depression	-.060	.028	.050	.169**	.113	-.157**	—				
8. Anger	-.020	-.034	.058	.186**	.125*	-.155*	.553***	—			
Age 22											
9. Telomere length	-.055	.045	-.004	-.124*	-.125*	.137*	-.179**	-.162**	—		
10. BMI	-.087	.122*	-.054	-.046	-.026	-.031	-.007	.033	-.066	—	
11. Age	.014	.202***	.182**	.079	.064	-.064	.101	-.053	.006	-.003	—
Mean	.37	.65	1.94	13.90	6.63	22.50	11.85	31.31	-.12	30.35	22.14
SD	.48	.48	1.39	5.00	3.95	2.97	8.65	11.52	.74	9.74	.99

Note. SES = socioeconomic status.

* $p < .05$. ** $p < .01$. *** $p < .001$.

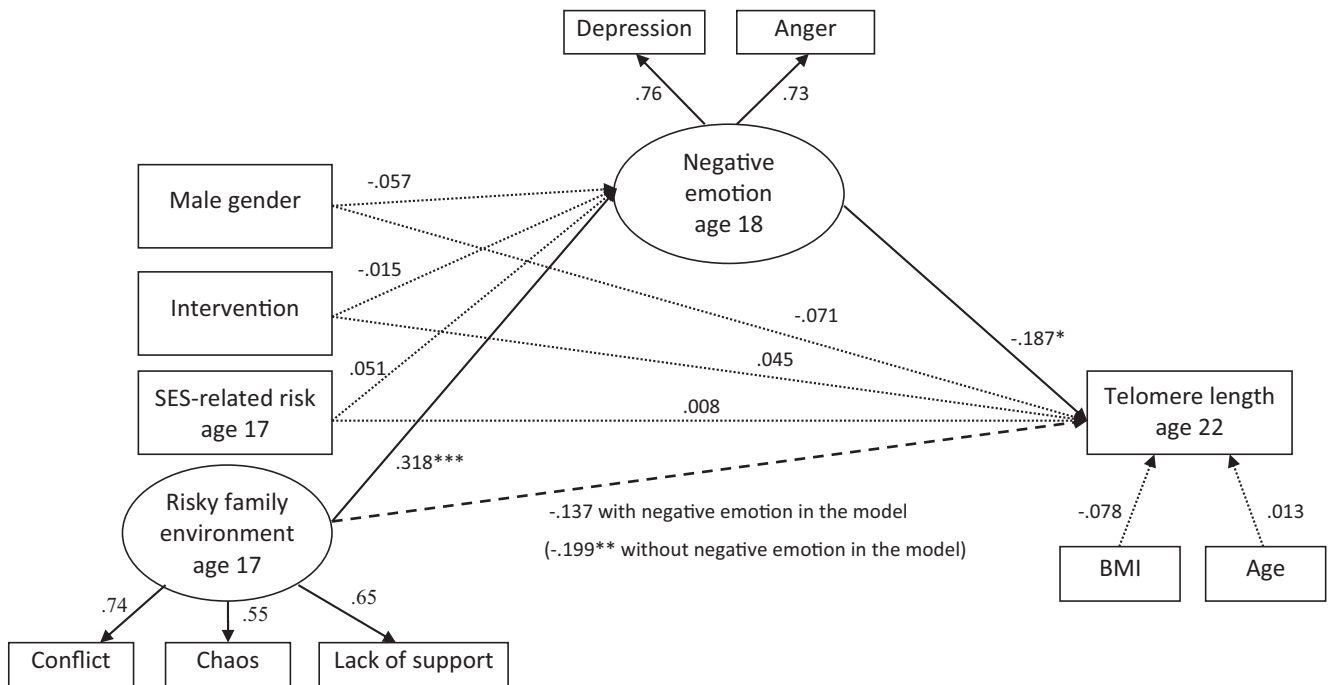


Figure 1. A mediation model of risky family at age 17, negative emotion at age 18, and telomere length at age 22 with socioeconomic-related risk, intervention status, gender, BMI, and age controlled. $N = 293$. Standardized coefficients are presented. SES = socioeconomic status. * $p < .05$. ** $p < .01$. *** $p < .001$.

risk conditions from the otherwise anticipated effect of early life stress on TL. Thus, some of the biological consequences of life in a risky family environment and the negative emotions it sponsors may result in complex interactions among frequent exposure to stress hormones, increased proliferation of immune cells, and other potential mediators (e.g., elevated oxidative stress and mitochondria dysregulation). All of these seem likely to influence rates of telomere erosion and cellular senescence, and thus, the rate of aging. Future research should test explicitly this mechanistic hypothesis with younger, nonclinical samples. This could not be done in the present study because no assessments were made of stress hormone output, indicators of inflammation, or indicators of oxidative stress. A follow-up study with multiple waves of psychosocial, hormonal, and other biomarker data would ideally be suited to pinpointing the mechanisms involved in these processes.

Limitations of the present study should be noted. Replications of this prospective study with younger children are indicated. Understanding the complex relationships among early family environments, negative affect, and biomarkers of premature cellular aging is highly relevant to public health. Also, it is not known whether the results of this study generalize to European American or Latino families living in either rural or urban environments. Finally, implications for prevention should be considered. Family-centered prevention that increases supportive parenting and reduces harsh parenting forecasts lower levels of proinflammatory cytokines (Miller, Brody, Yu, & Chen, 2014). Thus, family-centered prevention may be effective in deterring telomere erosion and the resulting health consequences among youths living in risky family environments.

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