CORRIGENDUM

Effects of pre- and postnatal maternal stress on infant temperament and autonomic nervous system reactivity and regulation in a diverse, low-income population—CORRIGENDUM

NICOLE R. BUSH, KAREN JONES-MASON, MICHAEL COCCIA, ZOE CARON, ABBEY ALKON, MELANIE THOMAS, KIM COLEMAN-PHOX, PATHIK D. WADHWA, BARBARA A. LARAIA, NANCY E. ADLER, AND ELISSA S. EPEL

aUniversity of California, San Francisco; bZuckerberg San Francisco General Hospital and Trauma Center; cUniversity of California, Irvine; and dUniversity of California, Berkeley

doi:10.1017/S0954579417001237, published by Cambridge University Press, 22 November 2017

We recently identified two important errors in the discussion in our original article. The first is in the first full sentence on page 1564, where higher in temperamental surgency should be lower, and the second is in the first sentence in the second paragraph on page 1566, where high surgency should be low. Both corrected pages are reprinted herein. We regret these errors and any problems they may have caused.

Reference


Address correspondence and reprint requests to: Nicole R. Bush, Psychiatry and Pediatrics, University of California, San Francisco, 3333 California Street, Suite 465, San Francisco, CA 94118; E-mail: Nicole.bush@ucsf.edu.
of maternal stress was “objective” (exposures) or “subjective” (appraisals), and also by whether the measure of infant reactivity and regulation was based on maternal perception or infants’ physiological responses to a standardized stressor. Overall, mothers who perceived themselves as being more stressed during pregnancy and postpartum reported that their infants were lower in temperamental surgency and had lower self-regulatory abilities, adjusting for exposure to SLE during pregnancy. These ratings of PS were unrelated to infant PNS stress reactivity and recovery. In contrast, higher counts of SLE during pregnancy were associated with greater infant PNS reactivity. Interaction findings suggest that the average effect of SLE on offspring physiology was significant, but that it was particularly salient among offspring of women with moderate to high levels of PS (i.e., PS appeared to moderate the effect of life event stress on offspring physiology). The findings are novel, in that there is relatively little data examining the unique contributions of both objective and perceived stress effects on offspring reactivity, and the majority of the few studies reporting tests of prenatal stress programming effects on offspring PNS reactivity have involved advantaged, Caucasian, non-US samples.

Given the uniqueness of the study population and the novelty of findings related to infant ANS reactivity, we focus our discussion first on these findings. Our ANS findings parallel those of Rash et al. (2015), who found that a higher maternal cortisol awakening response (a biological indication of greater stress, as well as other behavioral and biological processes) was associated with greater RSA reactivity for 6-month-old infants during a frustration paradigm. They also found it predicted lower baseline RSA, but that was not replicated in our study. Although more difficult to compare due to their use of multisystem profiles, Rash et al.’s (2016) finding from the same sample is also consistent with ours in that their mothers with relatively greater psychological distress during late pregnancy (in combination with decreasing daytime sAA slopes) were more likely to have infants who exhibited “coinhibition” of SNS and PNS during the stressors. Our findings are in contrast to Suurland et al. (2016), who found that a cumulative psychosocial risk score (including maternal

Figure 1. (Color online) (a) The interaction between stressful life events and perceived stress in the prediction of respiratory sinus arrhythmia reactivity, plotted at three levels of perceived stress. (b) The regions of significance for this interaction.
Corrigendum

here suggest that they may have different patterns of transmission to the fetus, at least in terms of ANS development.

In terms of stress paradigm methodology, our data are consistent with that of the two other studies we are aware of that have used two SF episodes to elicit RSA responses (Bosquet Enlow et al., 2014; Ritz et al., 2012). Our results were similar in that infants demonstrated PNS withdrawal to the SF episodes (with stronger reductions during the second SF) and some PNS recovery during reunion without full return to the original level during play. Other studies have found that infants from high-risk populations did not recover from the SF during reunion (Conradt & Ablow, 2010), or experienced even lower RSA in the reunion (Suuriland et al., 2016), suggesting that physiological effects of stress can be sustained, at least for a short while.

Our findings regarding maternal report of temperament are theoretically consistent with extant literature (Bosquet Enlow et al., 2009; Davis et al., 2011; Sandman et al., 2012), in that greater maternal pregnancy stress and postpartum stress have been associated with more difficult infant temperaments (such as low surgency and low regulation, found here), except that maternal stress did not predict negativity, which appears to be the most commonly documented association. Higher PS scores have been correlated with higher levels of cortisol (Pruessner, Hellhammer, & Kirschbaum, 1999); poor eating, drinking, and sleeping practices (Cohen & Williamson, 1988; Gibson, 2006); and general health behaviors during pregnancy (Guardino & Schetter, 2014), which can affect fetal development. In the prediction of infant regulation in our study, the effects of prenatal stress were larger than those of postnatal stress and the prenatal stress model accounted for 6% more of the variance, so it is tempting to infer that prenatal exposure to maternal PS is particularly relevant. Although important to examine perceptions, the stability of maternal report of PS across pregnancy and the postnatal period within this highly stressed sample may not be optimal for discerning prenatal from postnatal effects, as it prevented optimal modeling for determination of which exposure period was most important.

Extant theoretical and empirical literature suggests the timing of stress exposure is important for prenatal programming. Rash et al. (2015) found that maternal total cortisol assessed at 14 weeks of gestation, but not 32 weeks, was positively associated with infant RSA reactivity, and suggest that the effects of maternal cortisol on infant vagal tone appear to be sensitive to timing. Our assessment of exposure to SLE during pregnancy did not allow for determination of exposure timing. However, PS was assessed at two different time points during pregnancy (roughly 8 weeks apart). Although results were not presented here, exploratory analyses showed that the coefficients for “average prenatal stress” were stronger than those for either time point alone.

Limitations and strengths

In addition to the limitations described above, other factors merit consideration when interpreting findings presented here. First, although our sample size was larger than that of many ANS studies with infants (Bosquet Enlow et al., 2014; Feldman, Singer, & Zagoory, 2010; Moore, 2010; Ritz et al., 2012), funding time lines led to a relatively small sample, and a larger sample size is desirable. Second, the self-report measures of maternal stress and offspring temperament introduce potential bias and minimize confidence in those findings, yet others have found similar patterns using more objective measures of temperament. The setting for the assessment data described here also presents a possible limitation in that roughly half of assessments were completed in participant homes and the others were completed in our laboratory. This potential limitation is balanced by the successful completion of data collection with participants who were unable or unwilling to travel to our lab. Further, analyses revealed no difference in RSA values by home or clinic, as has been found in other home/clinic infant ANS studies (Haley, Handmaker, & Lowe, 2006). Third, as our focus was on understanding these phenomena within a multiethnic sample of low-income women, our study population did not have a full range of stress levels; specifically, it included few women with low levels of exposure to major adverse events. Despite this narrowed range, there was considerable variation in both of our stress predictors, and associations with offspring development were found.

These limitations are offset by a range of important study strengths. This study is one of few that examine infant RSA reactivity, and we used a gold-standard stress reactivity paradigm to assess reactivity and regulation. The study was conducted in a racially and ethnically diverse sample with a high level of exposure to life stressors, a population that is understudied and at increased risk for adverse infant development, including psychopathology. Moreover, the inclusion of both counts of adverse exposures and repeated measures of perceptions of stress provide an opportunity to investigate these unique sources of stress in vulnerable populations with complex challenges.

Implications and future directions

The specific role of PNS functioning within the etiology of early life psychopathology is still being understood, but weak PNS withdrawal to challenging contexts during infancy and early childhood has been shown to predict internalizing and externalizing symptoms; high levels of resting PNS activation and flexible withdrawal of the PNS in challenging contexts during early infancy and childhood have been shown to predict better regulation of attention and affect and more optimal social functioning (Beauchaine, 2001, 2015; Beauchaine, Gatze-Kopp, & Mead, 2007; Boyce et al., 2001; Calkins & Keane, 2004; Graziano & Derefeniko, 2013), although this can vary by sample type (Graziano & Derefeniko, 2013). The greater RSA withdrawal demonstrated by infants born to mothers with higher levels of exposure to adverse events may actually be adaptive, preparing the offspring for flexible responding to a stressful environment. Moreover,