Differential Effects of Environmental, Relational, and Biological Adversity on Autonomic Regulation in Young Children

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Background
• Research documents deleterious effects of risk exposure on behavioral, emotional, and, more recently, physiological adjustment (e.g., Blandon, Calkins, Keane, & O’Brien, 2008; Miller, Chen, & Zhou, 2007).
• In the domain of physiological regulation, and in particular cardiac functioning, subsystems (i.e., Respiratory Sinus Arrhythmia [RSA], which indexes parasympathetic functioning; Pre-ejection Period [PEP], which indexes sympathetic functioning) underlie global indices such as heart rate (HR), and may be differentially affected by adversity.
• Further, different types of risk (i.e., environmental, relational, biological) may exert unique effects on development (e.g., Bendersky & Lewis, 1994; Kerr, Black, & Krishnakumar, 2000).
• Therefore, the present study examined relations between specific types of adversity and children’s autonomic regulation with respect to parasympathetic (RSA), sympathetic (PEP), and global (HR) systems during a startle task.

Method
• Adversity exposure was assessed among 174 4-year-olds (51% female) via caregiver reports of environmental risk (i.e., poverty, residential mobility, mother’s education), relational risk (i.e., child physical abuse and/or excess punishment, child emotional abuse and/or harsh verbal punishment, child neglect and/or parental substance abuse), and biological risk (i.e., no prenatal care, prenatatal substance exposure, pregnancy/birth complications), and dichotomous indicators were summed within type. For group level comparisons, variables endorasing 2 or more indicators were considered high risk within each type.
• Children’s autonomic regulation was assessed at age 6 during a startle task. Task-specific baseline measures of HR, RSA, and PEP were collected while the examiner read a story corresponding to a set of puppets (2 minute baseline). Next, the examiner left and the mother followed previous instructions to lift a puppet, causing marbles to crash onto a metal tray (1 minute startle). The examiner then returned and completed the story (2 minute recovery).

Results
• Although an index of cumulative risk across domains was not significantly related to autonomic regulation, relations between adversity and regulation were variable across domains (Table 1b; Figures 1a-1c).
• Surprisingly, environmental adversity was related to better regulation as indicated by increases in reactivity (i.e., RSA decrease/HR increase).
• Conversely, relational adversity was associated with a muted pattern of responding, indicated by both less reactivity and recovery in RSA.
• Biological adversity was associated with higher baseline heart rate and lower baseline RSA, as well as less heart rate recovery.
• Although not significant, patterns of PEP responses mirrored RSA.
• Relational risk was associated with lower baseline PEP and less PEP recovery, while environmental risk was related to more PEP reactivity.

Discussion
• This study demonstrates the importance of accounting for unique relations between specific domains of adversity and individual subsystems of cardiac regulation.
• For example, biological risk exposure was associated with lower baseline adjustment, while environmental and relational risks were more related to reactivity and recovery.
• Further, relational risk appeared to be most strongly related to RSA functioning.
• Most notably, relational risk was associated with a flattened response pattern, but environmental risk with a highly reactive pattern.
• Ongoing work is needed to clarify the meaning of these differential response patterns.
• For example, environmental adversity may be less severe than relational adversity, such that the improved responsiveness of the environmental risk group may reflect benefits associated with moderate risk exposure.
• Alternatively, children in the high environmental risk group may actually be maladaptive in their hyperreactivity, such that the optimal response pattern lies between that expressed by the environmental and relational risk groups.
• Apparent differences in mean level (rather than pattern) of response between biological and relational risk groups highlight the need for future investigations to further clarify the meaning of these risk groups, their respective influences on regulatory development, and patterns of comorbidity across risks.
• In future research, we will examine potential moderators of these results (e.g., gender), and evaluate the consistency of behavioral patterns that occur in conjunction (and in contrast) with these physiological responses.

References

Table 1a. Bivariate Correlations between ANS Functioning and Adjustment

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Reactivity</th>
<th>Recovery</th>
</tr>
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<tbody>
<tr>
<td>HR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RSA</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>PEP</td>
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<td></td>
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</tbody>
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Table 1b. Bivariate Correlations between ANS Functioning and Variable Risk Exposure

<table>
<thead>
<tr>
<th></th>
<th>HR</th>
<th>RSA</th>
<th>PEP</th>
<th>HR</th>
<th>RSA</th>
<th>PEP</th>
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</thead>
<tbody>
<tr>
<td>Environmental Risk</td>
<td>-.031</td>
<td>.016</td>
<td>.028</td>
<td>.131</td>
<td>-.154</td>
<td>-.126</td>
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<td>Relational Risk</td>
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<td>.080</td>
<td>-.122</td>
<td>-.052</td>
<td>.141</td>
<td>.037</td>
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<tr>
<td>Biological Risk</td>
<td>.189</td>
<td>.207</td>
<td>.061</td>
<td>.043</td>
<td>.065</td>
<td>.071</td>
</tr>
</tbody>
</table>

*p≤.01, *p≤.05, #p≤.10; Reactivity and Recovery values are standardized residuals, where positive values for HR and negative values for RSA and PEP indicate more reactivity and less recovery.