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What Constitutes Effective Coping and Efficient Physiologic Regulation following Psychosocial Stress Depends on Involuntary Stress Responses

Jason J. Bendezú, Sarah E. D. Perzow, and Martha E. Wadsworth

The Pennsylvania State University, 208 Moore Building, University Park, PA 16801, United State of America

Abstract

This study utilized a random-assignment experimental design to examine the interactive contributions of youth-reported trait involuntary stress responses (ISRs) and effortful coping on physiologic reactivity and recovery patterns in preadolescent boys and girls. Fourth- and fifth-grade child-parent dyads ($N=126$) participated in this study. Children were exposed to the Trier Social Stress Test (TSST-C) and then to one of two randomly-assigned experimental coping conditions: behavioral distraction and cognitive avoidance. Children's ISRs were examined as predictors of salivary cortisol and alpha-amylase (sAA) reactivity as well as moderators of the effect of coping condition on cortisol and sAA recovery trajectories. Multi-level modeling analyses did not link ISRs to physiologic reactivity patterning. ISRs and coping condition interacted to predict differential physiologic recovery trajectories. In the distraction condition, children reporting high ISR levels displayed less efficient cortisol and sAA recovery than children reporting low ISR levels. Surprisingly, the opposite was found for children reporting high ISR levels in the avoidance condition. These children displayed more efficient physiologic recovery relative to their high ISR level peers in the distraction condition. Findings suggest that the efficiency of preadolescents' physiologic recovery following stress may depend on *regulatory fit* between children's ISR levels and cues from their coping environment.

Keywords

coping; preadolescence; cortisol; alpha-amylase; stress

1. Introduction

Coping, which entails effortful emotional, cognitive, and behavioral responses that help manage acute or ongoing stress, is a critical aspect of healthy child development. Extensive evidence links coping to an array of health and psychosocial outcomes (Compas et al., 2001; Grant et al., 2003), which may in part reflect the ability of coping to mitigate against stress-linked physiologic dysregulation (Wadsworth, 2015). However, little is known about for whom and under what conditions effortful coping works to help children effectively manage

the physiological effects of stress. Involuntary stress responses (ISRs) are immediate, automatic cognitive, behavioral, emotional, and physiologic responses to stress, which the individual perceives but does not enact intentionally (Compas et al., 1997; Lazarus, 1999). High ISR levels can interfere with effortful coping attempts after stress exposure (Compas et al., 1999; Wadsworth & Berger, 2006), but little research has empirically demonstrated this effect with rigorous experimental design and biological measures that capture the full time course of the stress response as it unfolds. The current study examined the degree to which ISRs enhance or constrain children's effortful coping and effect reactivity and recovery stress physiology patterning in a community sample of preadolescents.

The Responses to Stress (RTS) framework defines ISRs as biologically-based or overlearned responses to stress (Connor-Smith et al., 2000). Youth-reported ISRs demonstrate cross-time and -situation consistency (Valiente et al., 2014), suggesting they reflect enduring predispositions toward physiologic or psychological stress responses. ISRs include arousal and related behaviors that keep a child engaged with a stressor (intrusive thoughts, impulsive actions) as well as responses that direct a child or their reactions away from a stressor (escape, numbing). Children reporting high ISR levels experience automatic negative thoughts, physical symptoms such as muscle tension and increased heart rate, and difficulty concentrating when facing stressors. Left unmanaged, ISRs can contribute to health problems and emotional sequela (Kiecolt-Glaser et al., 2002; Lovallo & Gerin, 2003; Primo et al., 2000).

ISRs are, therefore, similar to constructs used in research with adults such as "stress reactivity," which is defined as an individual's disposition to respond to a stressor cognitively, behaviorally, and physiologically (Lovallo & Gerin, 2003; Schlotz, 2013). Thus, as with stress reactivity, the ISR construct captures processes that may originate from any number of brain systems or peripheral tissues involved in the stress response (Lovallo, 2011). In addition, ISRs, much like stress reactivity, are postulated to arise from both temperamental characteristics such as behavioral inhibition and disinhibition (Gray, 1991; Rothbart, 1991) as well as responses to novelty or threat that have become habitual (Compas et al., 1999). The ISR construct, which stems from developmental research, however, focuses more narrowly on a set of unplanned responses that have risen to the level of conscious awareness, but are not under conscious control. Importantly, in the developmental literature ISRs are fundamentally distinguished from "coping," which is an individual's conscious, volitional, and effortful response to stress (Compas et al., 1999).

Extensive evidence links effortful coping and ISRs *independently* to health and psychosocial outcomes (Compas et al., 2001; Kiecolt-Glaser et al., 2002). However, little research has focused on their *interactive* contribution, despite conceptualizations of coping as a process which entails effortful attempts directed toward managing involuntary, prepotent reactivity (Connor-Smith & Compas, 2004). This distinction between effortful adaptation and automatic, prepotent reactions is critical to the study of their interplay and, thus, what constitutes *effective* and *ineffective coping* (Compas et al., 1999). For example, ISRs are posited to place constraints on children's engagement in effortful coping, a process referred to here as *regulatory interference* (Compas et al., 1999; Wadsworth & Berger, 2006). Whether moving children toward or away from a stressor, ISRs may increase arousal and

attention to task irrelevant cues (worry, negative thoughts), consuming energy and executive resources that could otherwise be devoted to performing goal-relevant tasks or reallocating attention to distraction coping opportunities (Lazarus, 1999; Sarason et al., 1996; Wenzlaff & Wegner, 2000). Thus, individual differences in ISRs may play a role in defining for whom and under what conditions certain coping skills are ultimately effective.

If ISRs contribute to reactivity and *regulatory interference*, it should be evident at the physiologic level of analysis. Research demonstrating that child ISRs get “under the skin” (McEwen, 2012) is lacking. Children’s management of stress can be modeled as rise and fall patterns of hypothalamic-pituitary-adrenal axis (HPA) and sympathetic adrenal medullary system (SAM) by-products following a stressor (Hostinar & Gunnar, 2013). Studies with adults utilizing salivary cortisol (sC) and alpha-amylase (sAA) as biomarkers of HPA and SAM activity, respectively, indicate that ISRs such as rumination and negative cognitions are associated with elevated sC levels (Byrd-Craven et al., 2011) and increased physiologic *reactivity* in both the HPA (Zoccola & Dickerson, 2012) and SAM (Engert et al., 2014) systems.

Recent stress physiology models suggest that the phase of the stress response following reactivity – the *recovery* period – is as important as the reactivity phase for understanding stress-health links (Brosschot & Thayer, 1998; Javaraas et al., 2012). By definition, protracted recovery periods expose the body to elevated stress hormone levels for longer periods of time as the return to pre-stressor baseline levels is much slower. Children’s effortful coping manifests in the recovery period following exposure to an acute stressor and influences the efficiency with which stress hormones return toward pre-stressor baseline levels (Wadsworth et al., 2016). Thus, the recovery period following peak stress activity seems ideal for examining whether ISRs impede efficient cortisol and sAA recovery.

The Trier Social Stress Test for Children (TSST-C; Buske-Kirschbaum et al., 1997) is a reliable experimental procedure which can help demonstrate how ISRs contribute to individual differences in physiologic reactivity and recovery (Gunnar et al., 2009). We extend this methodology to further study *regulatory interference*; determining whether ISRs impede efficient cortisol and sAA recovery particularly for children given the opportunity to behaviorally distract themselves. Immediately following the TSST-C, children were experimentally primed with either behavioral distraction or cognitive avoidance coping. Distraction is believed to combat stress’ damaging effects by helping children consciously reengage their attention to productive or soothing activities while avoidance is thought to exacerbate these effects by inadvertently focusing attention to the source of stress (Wadsworth et al., 2016). Though not the explicit focus of the current study, we anticipated that children primed to distract themselves would demonstrate more efficient physiologic recovery patterning relative to those primed to avoidance. By limiting access to requisite executive resources (Compas et al., 1999), ISRs are thought to affect the type of effortful coping (e.g., distraction) a child can successfully enact and, thus, hamper efficient regulation of stress physiology. As such, we anticipated that children with high ISR levels primed to distract themselves post-TSST-C would be limited in their ability to effectively do so as evidenced by less efficient physiologic recovery patterns relative to low-ISR peers given the same prime.

Preadolescence is a critical period for studying ISRs and associated physiologic responses for many reasons. Burgeoning meta-cognitive and self-reflective capacities help alert children to the presence of distress and allow for more sophisticated forms of coping (Losoya et al., 1998; Seiffge-Krenke et al., 2009). These capacities also increase susceptibility to more automatic dysfunctional thinking processes (Ellis & Hudson, 2010; Wells, 1995). Lastly, preadolescent HPA and SAM systems are particularly sensitive to environmental cues, and poorly-managed stressors may contribute to dysregulated response profiles that continue into adulthood (Gustafsson et al., 2010; Hankin et al., 2010).

A clearer view of how preadolescent ISRs contribute to regulatory interference is needed to elucidate for whom and under what conditions coping is effective at down-regulating stress physiology. This knowledge may improve coping intervention efficacy and, in turn, thwart maladaptive psycho-biological development. The current study tests two specific predictions. H_1 : Children with higher ISR levels would display exaggerated cortisol and sAA *reactivity*, operationalized as steeper or more curvilinear reactivity trajectories. H_2 : Children with higher ISR levels would display more protracted cortisol and sAA *recovery* trajectories compared to their low-level peers, and this effect would be most evident for children primed to engage in distraction. That is, we expected ISR level and coping condition main effects to be qualified by their interaction, with ISRs differentially contributing to cortisol and sAA recovery trajectories in distraction and less so in the avoidance condition. We operationalize protracted recovery patterning as less steep, more curvilinear trajectories that are slower to return towards baseline.

2. Methods

2.1 Participants

Preadolescent children ($N = 126$, $M_{\text{age}} = 10.75$ years, $SD = .71$, 51.6% male) and a parent (89.7% mothers) were recruited from elementary schools in a large metropolitan area in the western U.S. ($n = 30$) and a semi-rural area in the northeastern U.S. ($n = 96$). Median annual household income for the sample was \$75,000. The majority of participants identified as White (96.03% of children and caregivers).

2.2 Procedures

Interested parents enrolled their child and completed questionnaires online. Children were then scheduled to complete the 90 minute (min) experiment between the hours of 3:00 and 5:30 pm. Parents were instructed to have their children refrain from brushing their teeth, consuming a large meal, dairy, or sugary and acidic foods within an hour of their study arrival time. Saliva samples were collected throughout the experiment via passive drool into vials at six time points (T1–T6). Upon arrival, children rinsed their mouth with bottled water and an experimenter collected a saliva sample (T1). Next, the experimenter administered questionnaires to the child for 40 min and then collected a second saliva sample (T2). Children then completed the TSST-C, a 15 min paradigm in which participants are asked to prepare (5 min) and deliver (5 min) a speech and complete a mental subtraction task (5 min) in front of an unresponsive “panel of experts.” Experts were confederates unknown to participants and unaware of their randomly-assigned¹ coping condition. A third saliva

sample was taken immediately after the TSST-C (T3). Children were then taken to one of two randomly-assigned coping condition rooms: a distraction room ($n = 66$) filled with musical instruments, art supplies, and toys where they were prompted to play with the materials and an avoidance room ($n = 60$) free of distractions where they were prompted to try not to think about their TSST-C performance. After 10 min, a fourth saliva sample was taken (T4). Children were then interviewed for 10 min about the coping strategies they used during and after the TSST-C. A fifth saliva sample was collected (T5). Next, children engaged in a 10 min guided progressive muscle relaxation exercise. Then, a final sixth saliva sample was collected (T6). Experimenters debriefed children after the experiment and families received \$50 for participating. Study procedures were conducted with the adequate understanding and written assent/consent of child/parent participants.

2.3 Measures

2.3.1 Salivary cortisol and alpha amylase (sAA)—Six saliva samples were collected via passive drool (Davis et al., 2002) to measure HPA and SAM reactivity and recovery. Saliva samples were stored in a medical grade ultra-low temperature freezer and transported on dry ice to the CORE Biomarker Lab at Penn State University (Salimetrics, LLC, State College, PA). Cortisol levels were determined using a commercial expanded-range high-sensitivity enzyme immunosorbent assay kit (No. 1-3002/1-3012; Salimetrics, LLC, State College, PA) that detects levels in the range of 0.003 to 3.0 Kg/dL (range, 0.08Y82.77 nmol/L).

sAA levels were determined using a commercial kinetic reaction assay kit (No. 1-1902; Salimetrics, LLC, State College, PA). sAA activity present in a sample is proportional to the increase (over a 2 min period) in absorbance at 405 nm. Results are computed in U/mL of sAA using the formula: $[\text{Absorbance difference per min} \times \text{total assay volume (328 ml)} \times \text{dilution factor (200)}] / [\text{millimolar absorptivity of 2-chloro-p-nitrophenol (12.9)} \times \text{sample volume (.008 ml)} \times \text{light path (.97)}]$. Cortisol and sAA were extracted from the same samples, run in duplicate, and batched in the same order as random assignment.

2.3.2 Characteristic involuntary stress responses (ISR)—Children completed the social stress version of the Responses to Stress Questionnaire (RSQ; Connor-Smith et al., 2000), a 57 item self-report measure of three effortful coping (primary control, secondary control, disengagement) and two involuntary stress response factors. Involuntary engagement stress response items assess rumination, intrusive thoughts, physiologic and emotional arousal, and involuntary action (e.g., “When I have problems with friends...I can’t stop thinking about how I am feeling or about the problem” and “I feel it in my body (check all that apply): my heart races, I feel hot and sweaty, my breathing speeds up, my muscles get tight”). Involuntary disengagement stress response items assess emotional numbing, cognitive interference, inaction, and escape (e.g., “When I have problems with friends...I can’t think at all”, “I just have to get away, I can’t stop myself”). Both ISR factors had acceptable internal consistency ($\alpha = .89$ and $.86$, respectively) and were strongly

¹Children were randomized to condition within gender and investigators were blind to this process.

correlated ($r = .86$). To create a more robust ISR index, we created a composite by aggregating both ISR factor scores.

Connor-Smith et al. (2000) observed that adolescents have a propensity for endorsing either higher or lower overall levels of all RSQ coping and stress response items. To control for these individual differences in item endorsement base rates and following guidelines set forth by the RSQ measure developers, ISR factor scores were computed as ratio proportion scores prior to analyses. ISR factor scores were divided by the sum of all five factor scores, with final scores reflecting the proportion of a child's stress responses allocated to ISRs. Both RSQ factor and ratio scores demonstrate convergent and discriminant validity with other measures of coping, behavioral adjustment, and physiologic reactivity in both adolescent and adult samples (Connor-Smith & Compas, 2004; Connor-Smith et al., 2000).

2.3.3 Coping condition manipulation check—Children reported what they did in their respective coping rooms to calm down, make themselves feel better, or make the situation better, even if they thought it did not work. These free responses were coded (yes/no) for distraction (active attempts to feel better or solve the problem) strategies and avoidance strategies (and/or associated intrusive thoughts). There was a significant relationship between children's assigned coping condition and their reported strategies, $\chi^2(1, 126) = 48.21, p < .001$ suggesting that children complied with respective prompts in the distraction (89%) and avoidance (72%) conditions.

2.3.4 Covariates—Covariates include child gender, puberty, income-to-needs ratio (INR) and medication use. Child gender was coded as 0 (boy) and 1 (girl). Pubertal status was assessed using the Pubertal Development Scale (Petersen et al., 1988). Female pubertal status was indexed by menarche, height, body hair growth, and breast growth ($\alpha = .72$). Male pubertal status was indexed by voice changes, height, body hair growth, and facial hair growth ($\alpha = .49$). INR is an index of annual household income relative to national poverty line norms. An INR of 1.0 indicates poverty while middle income status is indicated by an INR of 3.0. The median INR for the present sample was 2.91. Medication use (e.g., steroids and other anti-inflammatory medications known to affect cortisol activity) was scored for each child following recommendations outlined in Granger et al. (2009).

3. Data Analysis

3.1.1 Data reduction and preprocessing—Participants whose age was atypical for fourth and fifth grade ($n = 1$) and who did not participate in the 90m experiment ($n = 1$) were excluded from the present analyses ($N = 126$). Cortisol ($n = 20$) and sAA ($n = 17$) values ± 3 SD from the sample mean were excluded (Dozier et al., 2006; Gunnar et al., 2001). Cortisol and sAA values remained positively skewed and a fourth-root transformation normalized those data (Miller & Plessow, 2013). A $\ln$ transformation normalized INR values. MANOVA post-hoc tests of geographic location (western US, northeastern US) indicated each differed only on INR ($F = 22.86, p < .001$). This variable was controlled for in all subsequent analyses.

3.1.2 Missing data—Missing value analysis (SAS 9.3) was conducted for all key demographic and study variables used in the present set of analyses. Little's (1988) missing completely at random (MCAR) test was non-significant; $X^2(183) = 189.04, p = .36$. Thus, the data could be MCAR. Markov Chain Monte Carlo (MCMC) multiple imputation methods (PROC MI, SAS 9.3) were used which circumvented power loss associated with listwise deletion. As 2.94% of data values were missing, five imputations were performed (Graham et al., 2007) which included all variables to be used in analyses. Parameter estimates were pooled across the five imputations using PROC MIANALYZE (SAS 9.3).

3.1.3 Bivariate associations—Fisher's Z-adjusted correlations were used to examine bivariate associations. As research has demonstrated an association between gender, pubertal status, and physiologic responses (Stroud et al., 2011), partial correlations controlling for these variables in addition to family INR were conducted.

3.1.4 Multilevel models—Cortisol and sAA reactivity and recovery patterns were analyzed with multilevel modeling (MLM) methods (SAS 9.3, PROC MIXED), using reactivity (sAA: T1-T3; cortisol: T2-T4) and recovery (sAA: T3-T6; cortisol: T4-T6) phase sample time as a level-one within-subjects factor and ISR and coping condition (0=avoidance, 1=distraction) as level-two between-subjects factors. Baseline measures for sAA (T1) and cortisol (T2) differed as sAA reactivity can be observed within moments of stress exposure whereas cortisol peak is delayed (De Kloet et al., 2005; Takai et al., 2004). Random intercepts and linear slopes models were used (model fit data available upon request) with Kenward-Rogers corrected degrees of freedom. Child gender, pubertal status, INR², and medication use were included as covariates and retained despite statistical nonsignificance. As acute reactivity and inefficient down-regulation can be operationalized by significant linear and quadratic effects, we examined both in all analyses.

4. Results

Descriptive and bivariate statistics are shown in Table 1. Girls were further along in pubertal development than boys; Welch's $t(61.61) = -4.64, p < .001$. Twelve participants endorsed steroid or anti-inflammatory medication use (sample average score = .40, $SD = 1.26$)³. Cortisol levels during the experiment were highly correlated, as were sAA levels. ISR level was marginally positively associated with T2 sAA levels. Otherwise, ISR and coping condition were not correlated with cortisol or sAA levels.

4.1 H₁: Reactivity Trajectories

4.1.1 Analyses—To determine whether ISRs influenced reactivity cortisol and sAA trajectories, we conducted two separate MLM analyses. In each analysis, the following mean-centered two-way interaction terms were added to covariates and main effects: ISR x linear time (time), ISR x quadratic time (time²). To examine linear effects that were possibly

²Excluding INR from analyses did not alter MLM results or conclusions.

³Except for baseline sAA, $t(32.97) = 2.63, p < .05$, reactivity and recovery sC and sAA for children who were taking and did not take medication were not significantly different (all $p > .22$). Excluding participants who endorsed medication use from analyses did not alter MLM results or conclusions.

masked by quadratic terms, non-significant quadratic effects were systematically removed from each model. Table 2 displays parameter estimates for each reactivity MLM.

4.1.2 Cortisol—In the first step, family INR ($\beta = -0.29$, $p < .01$) was the only covariate significantly associated with overall reactivity-phase cortisol levels. Significant main effects emerged for only linear ($\beta = -0.02$, $p < .001$) and quadratic ($\beta = -0.03$, $p < .001$) time, suggesting that children's salivary cortisol levels increased during the reactivity phase and that the shape of the effect was curvilinear. All two-way interactions were non-significant.

4.1.2 sAA—Apart from significant linear ($\beta = 0.13$, $p < .001$) and quadratic ($\beta = .05$, $p < .001$) time effects, no significant main effects or two-way interactions emerged. Contrary to hypotheses, there was no evidence to suggest that children's ISR influenced reactivity cortisol or sAA trajectories.

4.2 H₂: Recovery Trajectories

4.2.1 Analyses—Similar MLM analyses were conducted to examine whether ISR was differentially associated with cortisol (T4-T6) or sAA recovery⁴ (T3-T6) trajectories, and whether this relation differed by coping condition. Table 3 provides parameter estimates for the two final recovery MLMs. Significant three-way interactions were probed at 2 *SD* above and below mean ISR values (Roisman et al., 2012). In cases where ISRs interacted with coping condition and time to predict recovery trajectories, we only report and interpret the contribution of those effects within the context of the significant three-way interactions.

4.2.2 Cortisol—Covariates were not significantly associated with overall recovery cortisol levels. Significant main effects for only linear ($\beta = -0.02$, $p < .001$) and quadratic ($\beta = -0.01$, $p < .05$) time emerged, suggesting that children's cortisol levels decreased in a curvilinear manner during the recovery phase. Furthermore, the interaction between ISR, coping condition, and quadratic time was significant; $\beta = -0.06$, $p < .05$ (Figure 1)⁵. As expected, children with high, compared to low, ISR levels who were primed to distract themselves following the TSST-C had more negative quadratic, less efficient cortisol recovery trajectories. Cortisol levels for high ISR children continued to increase during the 10 min following T4, which was then followed by a moderate decrease. Conversely, children with low ISR levels also primed to distract exhibited a less quadratic, more efficient cortisol recovery trajectory.

Surprisingly, the opposite was true for children in the avoidance condition (Figure 1). Recovery cortisol trajectories for children with high ISR levels were less quadratic and more efficient when they were primed to actively avoid thinking about their TSST-C performance, relative to children with the same ISR levels primed to distract. Recovery cortisol trajectories for children with low ISRs primed to avoid were more quadratic and less efficient compared to low-level peers primed to distract.

⁴Cubic time and interaction effects were explored, found to be non-significant, and were thus removed.

⁵The inclusion of this three-way cross-level interaction reduced level-1 residual variance by 1%, level-2 intercept variance by 5%, and level-2 slope variance by 6%.

4.2.3 sAA—Family INR ($\beta = -1.69, p < .05$) was the only covariate significantly associated with overall reactivity-phase cortisol levels. Significant main effects emerged for only linear ($\beta = -0.04, p < .001$) and quadratic ($\beta = 0.05, p < .01$) time, suggesting that children's salivary cortisol levels decreased during the recovery phase in a curvilinear manner. A significant three-way interaction between ISR, coping condition, and quadratic time emerged; $\beta = -0.70, p < .05$ (Figure 2)⁶. As expected, children with high, compared to low, ISR levels who were primed to distract themselves following the TSST-C had less steep and efficient sAA recovery trajectories. For these children, sAA levels exhibited a more linear slow decrease between T3 and T6. Conversely, children with low ISR levels who were also primed to distract exhibited a more curvilinear sAA recovery trajectory. That is, sAA levels quickly and efficiently decreased from T3 to T4 and maintained this decrease between T4 and T6.

Again, the opposite was true for children in the avoidance condition (Figure 2). Children with high ISR levels had unexpected curvilinear sAA trajectories. Relative to their high ISR level peers primed to distract, these children had steeper decreases between T3 and T4 that were maintained between T4 and T6. Recovery sAA trajectories for children with low ISR levels were more stably elevated between T3 and T6 and thus less efficient relative to their low-level peers in the distraction condition.

5. Discussion

The present study utilized a novel experimental design to examine associations between preadolescents' trait ISRs and physiologic reactivity and recovery patterning when exposed to a standardized psychosocial stressor. We measured HPA and SAM reactivity and recovery and compared patterns between children reporting higher ISR levels and those reporting low ISR levels, relative to all stress responses. We also investigated the effect of priming distraction and avoidance coping strategies for physiologic response trajectories of children with different ISR levels. Findings revealed *regulatory interference* in both HPA and SAM systems during the recovery, but not reactivity, phase of stress response. Further, primed coping strategies traditionally thought to be universally adaptive (e.g., distraction) appear to be effective in managing stress physiology, but only for some children and under certain conditions. We suggest that future studies consider the notable possibility of *regulatory fit* in order to best understand how match between child-level characteristics and coping strategies influence children's physiologic stress response profiles (Wadsworth et al., 2016). This knowledge is critical to identifying factors which may influence the efficacy of coping-based interventions (Compas et al., 1999).

5.1 No relationship between ISR levels and physiologic reactivity

Cortisol and sAA levels increased over the course of the reactivity phase, but this increase did not differ as a function of children's ISR levels. This finding was unexpected and is partially inconsistent with previous adult research indicating that ISR-related constructs are linked to HPA and SAM reactivity. Specifically, previous studies with adults have found that

⁶The inclusion of this three-way cross-level interaction reduced level-1 residual variance by 1%, level-2 intercept variance by 1%, and level-2 slope variance by 11%.

physiological reactivity is positively associated with rumination, intrusive thoughts, and worry (Brosschot et al., 2006). These specific ISRs resulted in prolonged arousal that is initiated in anticipation of a threat and is maintained even after the threat has passed (Engert et al., 2014; Zoccola & Dickerson, 2012).

Three considerations with respect to the nonsignificant reactivity findings are worth noting. First, preadolescence is a heterogeneous period of development with respect cortisol reactivity (Gunnar et al., 2009). Thus, additional child-level moderating variables (e.g., age, sex, and working memory) which the current study did not consider may differentiate children who are more or less susceptible to ISRs and reactivity stress physiology (Gur et al., 2012; Ji et al., 2015). Second, the current study focused on “trait” ISRs. A few studies with adults have found associations between trait ISR-related constructs and cortisol reactivity (e.g., perceived stress reactivity, Schlotz et al., 2011). Those which have used observational, video-mediated recall, and experience sampling methods to assess “state” ISRs during laboratory based stressors have found positive associations with physiologic “reactivity,” as evidenced by elevated post-stressor cortisol and sAA levels (Byrd-Craven et al., 2011; Engert et al., 2014; Zoccola et al., 2010). Methods designed to capture in the moment, “state” ISRs with preadolescents may be needed to detect this relationship. Third, this divergence may stem from different operational definitions of “reactivity.” Many prior studies operationalized reactivity as elevated cortisol or sAA levels over the entire stress response (Brosschot et al., 2006; Zoccola & Dickerson, 2012) including the post-peak period which we and others (e.g., Ji et al., 2015) consider to be recovery, not reactivity. The positive associations among ISRs and “reactivity” in some but not all (Schlotz et al., 2011) prior studies may reflect a combination of reactivity and recovery effects in their measures of reactivity.

5.2 Differences in physiologic recovery efficiency: Regulatory interference

As noted by Wadsworth et al. (2016), the recovery phase of the stress response appears to be particularly well-suited for demonstrating the contribution of ISRs and their interaction with coping on cortisol and sAA responses to acute psychosocial stress. According to RTS theory (Connor-Smith et al., 2000), effortful coping takes place during the recovery period and influences the speed and efficiency of physiologic-down regulation. Thus, while ISRs did not on their own directly influence cortisol and sAA recovery profiles as expected, this is not surprising given that *regulatory interference* is thought to occur in the presence of effortful coping attempts. Indeed, *regulatory interference* effects on physiologic recovery patterns emerged when both ISRs and effortful coping were examined interactively.

As expected, children reporting high ISR levels primed to distract themselves displayed less efficient cortisol and sAA recovery patterns compared to children with low ISR levels in the same condition. ISRs are thought to set limits on the types of effortful coping responses a child can effectively engage in while managing a stressor (Compas et al., 1999, Wadsworth & Berger, 2006). It may be that ISRs in part reflect overlearned, automatic cognitive processes that deplete effortful processing reserves needed for effective problem-solving (e.g., preparing and delivering a speech) or volitional reallocation of attention (e.g., distraction). It is also possible that unsuccessful attempts to manage ISRs when given ample

distraction options may have exacerbated ISR influences, contributing to feelings of being out of control and heightened perceived distress (Kelly & Kahn, 1994). These interpretations are consonant with an emerging literature on the effects of worry and self-generated thoughts on task performance and stress physiology (DeCaro et al., 2011; Smallwood, 2013).

5.3 Differences across coping conditions: Regulatory fit

Our investigation into associations between preadolescent stress physiology, ISRs and regulatory interference revealed an unexpected and intriguing finding. High ISR children primed to avoid thinking about their TSST-C performance displayed more efficient cortisol and sAA recovery trajectories compared to high ISR children who were primed to engage in behavioral distraction. Our account of regulatory interference thus far might predict the opposite. That is, elevated ISRs should consume processing resources needed for effortful coping and efficient physiologic down-regulation, perhaps including those presumably needed for effortful avoidance. In fact, engaging in active avoidance efforts has been shown to *exacerbate* involuntary responses such as rumination and intrusive thoughts (Wenzlaff & Wegner, 2000), which together should hamper, not facilitate, the efficiency of physiologic recovery.

How, then, do children with elevated ISR levels evince more efficient regulation across two physiologic systems when primed to avoidance coping relative to distraction? A plausible explanation, which draws on our assessment of characteristic ISRs as opposed to in-vivo ISRs, is that avoidance may have provided a better *regulatory fit* for this subgroup of children (Wadsworth et al., 2016). Compas and colleagues (1999) argued two points which are germane to this supposition. First, effective coping can only be understood when examined in relation to child-level characteristics and the contexts in which it is enacted. Mismatches (i.e., poor regulatory fit) between coping strategies and child-level characteristics (e.g., temperament) or contexts may contribute to increased, rather than decreased, distress. Second, ISRs in childhood and adolescence have some basis in temperament individual differences that emerge early in development. Thus, preadolescent ISRs may be a product of temperamental *heterotypic continuity* (Caspi, 1998), with implications for how children typically cope with stressors. Indeed, infants who are highly reactive and display elevated negative affect to novel stimuli typically engage in avoidance behaviors (e.g., avoiding peers, not speaking in front of others) in early and middle childhood (Fox et al., 2001; Kagan & Snidman, 2004). Also, children who report characteristically elevated involuntary physiologic arousal and intrusive thoughts often use avoidant forms of coping (e.g., disengagement; Connor-Smith et al., 2000; Wadsworth et al., 2005).

As our assessment of children's ISRs was more reflective of dispositional characteristics (as opposed to situation-specific responses), our findings also demonstrate the concept of *regulatory fit*. That is, encouraging children who characteristically experience high levels of ISRs to avoid thinking about their performance on the speech and math tasks may have actually been beneficial for physiologic recovery because avoidance is more consistent with strategies they tend to use. This may have been particularly important for these children after

finishing the TSST-C because, by virtue of the task's approach-related nature, it offers little opportunity to behaviorally disengage or avoid stress-related cognitions. Further, prompting these high-ISR children to engage in unaccustomed behavioral distraction may have increased mental load by introducing them to additional novelty, placing demands on executive resources that would otherwise be typically allocated to suppressing ISRs (Wenzlaff & Wegner, 2000). The distraction condition, therefore, may have provided a worse regulatory fit for high ISR children.

5.4 Implications, limitations, and future directions

The present experimental study is the first to empirically demonstrate that different coping strategies and contexts contribute to *regulatory interference* and provide better *regulatory fit* depending on whether children experience high or low levels of ISRs. Though effects were small and require replication, the findings suggest that what constitutes effective coping may in part be determined by *regulatory fit*, or the match between child-level characteristics, coping strategies, and available environmental coping resources (Compas et al., 1999). There may not be universally adaptive or maladaptive coping strategies that work equally well at regulating stress physiology for all children in all situations (Wadsworth, 2015). Rather, our findings suggest that behavioral distraction may work for preadolescents with low ISR levels whereas cognitive avoidance may work for preadolescents who experience high ISR levels in the face of psychosocial stress. These relations, however, may not hold across development. As preadolescents mature cognitively, they may become better equipped to use secondary control strategies (e.g., focused distraction) to overcome their predispositions towards involuntary responding to stress and associated physiologic arousal. Longitudinal research is needed to clarify this. Furthermore, as ISRs appear to constrain how effective preadolescents' distraction efforts are, they may also influence how children acquire this and other coping skills (Compas, 2009).

Thus, coping intervention research with this age group is needed to explore the extent to which limits posed by ISR-based regulatory interference and fit also apply to how efficiently skills can be learned and implemented. Our results support the need for patient-centered intervention efforts where children's typical ways of coping and responding involuntarily to stress are used to tailor intervention content specifically for their regulatory strengths and weaknesses. Additionally, these findings suggest that there is no one "right" way to cope with stress; that curricula should emphasize that what works for one individual in one circumstance might not work for that person in a different circumstance or for someone else in the same circumstance. Hence, being taught a broad array of strategies and learning the importance of flexibility and persistence are likely to be beneficial for all children.

An additional inquiry motivated by our findings as well as the regulatory fit hypothesis is whether alternative coping strategies, such as *cognitive distraction* or *behavioral avoidance*, would provide better or worse regulatory fit for children who report high ISR. Take, for example, *behavioral distraction*. Children reporting high ISR continued to display decreases in sAA levels from T5 to T6, immediately following the guided progressive muscle relaxation (PMR) exercise. PMR may have been effective in helping these children recover from TSST-C where distraction behaviors were not. Indeed, priming individuals to *think*

about something else (e.g., guided imagery) is thought to help manage involuntary thought processes (Wenzlaff & Wegner, 2000). Future studies are needed to test this hypothesis and should incorporate PMR at earlier time points where there are higher physiology levels to recover from.

There are limitations of the present study that should be addressed in future research. First, children in our study constitute a relatively homogenous sample, which limits the generality of the findings. However, our aim was to provide empirical evidence of a physiologic component to children's ISRs and possible *regulatory interference* processes. A non-at risk sample is well-suited for a proof-of-principle study such as this. Research which demonstrates the applicability of these novel findings in more diverse samples (e.g., lower-income, ethnically diverse) is warranted (Brenner et al., 2013). Second, our sample included children who took medications known to influence stress hormone levels. However, these children, for the most part, did not significantly differ in cortisol or sAA sample levels and the contribution of medication use scores to cortisol and sAA reactivity and recovery patterning was non-significant. Additionally, though important covariates were considered, this study did not control for other variables known to influence cortisol levels in challenge paradigms (e.g., BMI; Francis et al., 2013). Third, though children's retrospective reports suggest that our coping manipulation was successful in producing the intended strategies, we did not assess or analyze children's in-the-moment use of distraction or avoidance. Future studies could consider momentary assessment and experience sampling methods for more fine grained analysis of children's in-vivo coping and influence of state ISRs on preadolescent stress physiology. Fourth, it is possible that children's perceptions of how effective their assigned strategies were in making themselves feel better also influenced physiologic recovery efficiency. Future studies should examine perceived coping effectiveness and efficacy as predictors of physiologic recovery patterning. Finally, our regulatory interference and fit findings are limited to child-level characteristics. As suggested by Compas et al. (1999), research is needed to understand how contextual factors (e.g., daily stressors, chronic stress, poverty) dictate what constitutes effective and ineffective coping and associated physiologic response profiles. Despite these limitations, this study provides novel empirical evidence that preadolescents' involuntary stress responses get "under the skin" and elucidates mechanisms that explain individual differences in child reactivity and post-stressor recovery.

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Figure 1.

Predicted cortisol concentrations for high (red line, square markers) and low (blue line, circle markers) involuntary stress responses (ISR) by coping condition during the reactivity and recovery phase (shaded region). Predicted trajectories were plotted two standard deviations above and below mean ISR scores and reverse transformed for illustrative purposes. Nonsignificant reactivity trajectories plotted for visual clarity and ease of interpretation.

Figure 2.

Predicted alpha-amylase concentrations for high (red line, square markers) and low (blue line, circle markers) involuntary stress responses (ISR) by coping condition during the reactivity and recovery phase (shaded region). Predicted trajectories were plotted two standard deviations above and below mean ISR scores and reverse transformed for illustrative purposes. Nonsignificant reactivity trajectories plotted for visual clarity and ease of interpretation.

Descriptives and Partial Correlations for Child Involuntary Responses, Coping Condition, and Stress Hormone Concentrations

Table 1

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	<i>M</i>	<i>SD</i>	<i>Min</i>	<i>Max</i>
(1) Child ISR	—	.11	.08	.15 [†]	.08	.07	.13	.03	—	—	—	—
(2) Coping condition ^a	.11	—	.02	-.09	.01	-.01	.01	-.01	—	—	—	—
(3) Saliva sample - T1	.03	.02	—	.59*	.50*	.20*	.21*	.29*	80.10	68.84	6.30	413.83
(4) Saliva sample - T2	-.12	-.08	.65*	—	.73*	.38*	.37*	.42*	81.27	80.33	6.23	604.70
(5) Saliva sample - T3	.03	.01	.53*	.72*	—	.65*	.62*	.69*	114.35	96.18	2.30	596.40
(6) Saliva sample - T4	-.02	-.01	.24*	.39*	.63*	—	.94*	.89*	89.66	84.00	10.16	603.90
(7) Saliva sample - T5	.05	.01	.24*	.38*	.57*	.94*	—	.93*	88.93	85.31	7.21	619.10
(8) Saliva sample - T6	.06	.01	.31*	.45*	.63*	.87*	.93*	—	80.16	69.57	7.40	571.20
<i>M</i>	0.23	0.52	0.08	0.08	0.08	0.12	0.11	0.09				
<i>SD</i>	0.04	0.50	0.06	0.06	0.05	0.08	0.09	0.07				
<i>Min</i>	0.13	0.00	0.01	0.01	0.01	0.01	0.01	0.01				
<i>Max</i>	0.34	1.00	0.36	0.37	0.28	0.41	0.42	0.32				

Note. *N* = 126; ISR = Involuntary Stress Response composite; T = Time. Coefficients above the diagonal reflect correlations between alpha-amylase values. Coefficients below the diagonal reflect correlations between cortisol values. Descriptives to the right of the correlation matrix correspond to alpha-amylase levels. Descriptives underneath the correlation matrix correspond to cortisol levels.

^aSpearman's rho

[†]*p* = .09.

**p* < .05.

Table 2

Parameter Estimates, (Standard Errors), and Fit Statistics for Regulatory Interference Multilevel Models Predicting Reactivity Phase Salivary Cortisol and Alpha Amylase Growth Trajectories

	Cortisol	Alpha-Amylase
Main effects		
Intercept	0.53 [*] (0.01)	2.89 [*] (0.05)
Child gender	−0.03 (0.01)	0.01 (0.11)
Child puberty	−0.01 (0.01)	−0.01 (0.03)
Family income-to-needs ratio	−0.29 [*] (0.10)	−1.47 (0.86)
Child medication use	−0.01 (0.01)	−0.01 (0.04)
Child ISR	−0.05 (0.12)	0.82 (0.81)
Linear time (time)	0.03 [*] (0.01)	0.13 [*] (0.02)
Quadratic time (time ²)	0.01 [*] (0.02)	0.05 [*] (0.01)
Two-way interactions		
Child ISR x linear time (time)	0.07 (0.08)	0.05 (0.31)
Child ISR x quadratic time (time ²)	−0.04 (0.02)	−0.18 (0.13)
Random effects		
Intercept	0.01 [*] (0.01)	0.26 [*] (0.04)
Slope	0.01 [*] (0.01)	0.02 [*] (0.01)
Covariance (Intercept, Slope)	0.01 [*] (0.01)	0.02 [*] (0.01)
Residual	0.01 [*] (0.01)	0.05 [*] (0.01)

Note. N=126; ISR=involuntary stress response

^{*}
p < .05.

Table 3

Parameter Estimates, (Standard Errors), and Fit Statistics for Regulatory Interference Multilevel Models Predicting Recovery Phase Salivary Cortisol and Alpha Amylase Growth Trajectories

	Cortisol	Alpha-Amylase
Main effects		
Intercept	0.55 [*] (0.01)	2.97 [*] (0.07)
Child gender	−0.03 (0.02)	0.11 (0.11)
Child puberty	0.01 (0.01)	−0.01 (0.03)
Family income-to-needs ratio	−0.27 (0.14)	−1.69 [*] (0.79)
Child medication use	0.01 (0.01)	0.02 (0.04)
Child ISR	0.05 (0.23)	−0.32 (1.20)
Coping condition	−0.01 (0.02)	−0.11 (0.10)
Linear time (time)	−0.02 [*] (0.01)	−0.04 [*] (0.01)
Quadratic time (time ²)	−0.03 [*] (0.01)	0.05 [*] (0.02)
Two-way interactions		
Child ISR x coping condition	0.01 (0.31)	1.61 (1.60)
Child ISR x linear time (time)	0.06 (0.06)	0.05 (0.12)
Coping condition x linear time (time)	0.01 (0.01)	0.01 (0.01)
Child ISR x quadratic time (time ²)	0.02 (0.02)	0.21 (0.26)
Coping condition x quadratic time (time ²)	0.01 (0.01)	−0.02 (0.02)
Three-way interactions		
Child ISR x coping condition x linear time (time)	0.01 (0.08)	−0.21 (0.17)
Child ISR x coping condition x quadratic time (time ²)	−0.06 [*] (0.03)	−0.70 [*] (0.35)
Random effects		
Intercept	0.01 [*] (0.01)	0.25 [*] (0.03)
Slope	0.01 [*] (0.01)	−0.01 [*] (0.01)
Covariance (Intercept, Slope)	0.01 (0.01)	0.01 (0.01)
Residual	0.01 [*] (0.01)	0.05 [*] (0.01)

Note. N=126; ISR=involuntary stress response

^{*}
p<.05.