

COMMENTARY

SOME DIFFICULTIES IN INTERPRETING PSYCHOPHYSIOLOGICAL RESEARCH WITH CHILDREN

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Mona El-Sheikh and her colleagues have assembled an impressive series of studies for this monograph. To date, researchers interested in the consequences of marital conflict on children have focused primarily on the main effects of psychological variables. Although these studies have been invaluable in helping us to better understand risk and resiliency factors associated with children's exposure to marital conflict, future research will almost certainly be conducted at multiple levels of analysis, spanning genes to behavior (Cicchetti, 2008). The authors' work examining the conjoint effects of marital conflict and biological vulnerability on children's externalizing behaviors is therefore a welcome addition to the literature and likely marks a transition to a new generation of research.

Among the few authors who have examined biological moderators of outcomes among children exposed to marital conflict, respiratory sinus arrhythmia (RSA), an index of parasympathetic nervous system (PNS) functioning, has been the predominant measure used (e.g., El-Sheikh & Whitson, 2006; Katz & Gottman, 1995). In this monograph, El-Sheikh and colleagues extend this work in a number of important ways. First, they include a measure of sympathetic nervous system (SNS) functioning and examine interactions between the SNS and PNS in predicting children's externalizing behaviors. Second, they provide detailed descriptions of and comparisons among several alternative models of autonomic nervous system (ANS)-behavior relations, grounding their hypotheses firmly in theory. Finally, they conduct the same protocol with three separate samples of children (total $N = 577$), recognizing that three-way interactions, which are required to test their hypotheses, are often spurious and difficult to replicate. The authors should be applauded for this painstaking research effort. Although their pattern of results is complex, the findings indicate that children's physiological functioning is clearly related to their externalizing behaviors in the context of marital conflict.

THE DOCTRINE OF AUTONOMIC SPACE

It has been assumed traditionally that the SNS and the PNS function reciprocally, with increased activity in one branch accompanied by decreased activity in the other. This assumption underlies early theories linking individual differences in ANS functioning to psychopathology. For example, after observing low resting heart rate (HR) among delinquents, Eppinger and Hess (1910/1915) proposed that an autonomic imbalance favoring the inhibitory PNS was responsible for delinquent behavior. Given the assumption of reciprocal SNS and PNS activation, this was the only explanation available to account for low HR.

It is now widely accepted that the SNS and PNS can activate and/or deactivate reciprocally, independently, or coactively, depending on environmental conditions and both psychological states and traits (Berntson, Cacioppo, Quigley, & Fabro, 1994). Several studies conducted across a wide age range (see, e.g., Beauchaine, Gatzke-Kopp, & Mead, 2007) have demonstrated reduced SNS- and PNS-linked cardiac activity among conduct-disordered males, both at baseline and in response to rewarding stimuli. Thus, consistent with the hypotheses set forth in this monograph, these individuals exhibit a pattern of SNS and PNS *coinhibition*.

Although El-Sheikh and colleagues invoke the doctrine of autonomic space (among other models), their approach differs from most similar studies because their measure of SNS activity is electrodermal rather than cardiac. This is not a trivial difference. The concept of autonomic space was written to explain autonomic control of dually innervated target organs such as the heart (Berntson et al., 1994). Yet El-Sheikh and colleagues use a cardiac measure of PNS activity and an electrodermal measure of SNS activity. Electrodermal responding (EDR) is widely recognized as a nonspecific marker of psychological state, showing increases to an impressively wide range of stimuli, both positive and negative. Nevertheless, like SNS-linked cardiac activity, low EDR has been found repeatedly among delinquent males (Lorber, 2004). Thus, even though EDR provides different information about SNS responding than measures derived from cardiac function (e.g., cardiac preejection period), the authors' choice to use EDR does not invalidate their work. Indeed, the "proof is in the pudding," and the results reported by El-Sheikh and colleagues are consistent across three studies, three reporters, and two tasks—an impressive achievement given that confirmation of their hypotheses was dependent on detecting three-way interactions.

TESTING INTERACTIONS IN PSYCHOLOGICAL RESEARCH

The main hypotheses set forth by El-Sheikh and colleagues are bold. Based on the assumption that coupled (reciprocal) activation of the SNS and

PNS marks psychological health and that uncoupled activation (coinhibition or coactivation) marks psychological risk, El-Sheikh and colleagues predict that (a) coupled responding of the SNS and PNS will serve as a protective factor for developing externalizing behaviors following exposure to marital conflict, and (b) uncoupled responding of the SNS and PNS will serve as a risk factor for developing externalizing behaviors following exposure to marital conflict. These hypotheses are tested in a series of Marital Conflict \times RSA \times EDR (three-way) interactions.

An important consideration concerns power for detecting interaction effects, which is always considerably lower than power for detecting main effects in a regression equation (see Whisman & McClelland, 2005). One reason for this is that the power to detect any effect in statistics depends on the reliability of the measures used. The more measurement error, the lower the statistical power. This is compounded when testing interactions because the reliability of the interaction term ($\alpha \times \beta$ for a two-way interaction) equals the product of the reliabilities of the main effects. Thus, even with very high reliabilities for two IVs (e.g., .85 each), the reliability of the interaction term is reduced considerably (.85 \times .85 = .72). As a result, achieving a conventionally acceptable power level of .80 ($\alpha = .05$) to detect a medium-sized interaction effect (partial $r = .13$; see Cohen, 1988) requires 55 participants compared with 37 participants to detect a medium-sized main effect ($r^2 = .24$). This effect is of course compounded with each additional IV in the interaction term. For this reason, higher-order interactions are both difficult to detect and difficult to replicate.

In the present series of studies, 32 terms were entered into each of the hierarchical regressions. Using Study 1 as an example, with an overall R^2 of .13 for mother-reported delinquency, power to detect a three-way interaction effect was about .4. I do not point this out to criticize the authors' work. Recruiting these many children and their parents to participate in psychological research is not an easy task. Moreover, not only did they detect the predicted interactions, they did so in three separate studies. Had only one study yielded a three-way interaction, their findings would have been met with frank skepticism. With consistent replication across studies, raters, and tasks, however, we can be much more confident in their results.

DEVELOPMENTAL VERSUS CLINICAL SAMPLES

Of course the key finding in the El-Sheikh and colleagues' monograph is the consistency with which SNS \times PNS interactions emerged across measures and studies. By definition, these interactions imply that high RSA (or RSA augmentation) is protective in some situations but a risk factor in others. This deserves comment because many developmentalists have

found positive externalizing-RSA relations (e.g., Dietrich et al., 2007), whereas many developmental psychopathologists have found negative externalizing-RSA relations (see Beauchaine et al., 2007). In fact, the direction of effects has been so consistent across developmental and psychopathological samples that such findings (a) are surely not anomalous and (b) suggest a different mechanism for RSA-behavior relations at the extreme end of the externalizing spectrum. They may also be due to differences in tasks used by developmentalists and developmental psychopathologists. Again, this is not a criticism of the El-Sheikh and colleagues' studies. Rather, it is an empirical instantiation of a core tenet of developmental psychopathology: Mechanisms of behavior are not always the same at the extremes of a distribution as they are near the mode. Thus, findings from this monograph should be interpreted with the understanding that very few participants met criteria for clinical levels of externalizing behavior (i.e., $T \geq 70$). To confuse matters further, a considerable body of research indicates that moderate reductions in RSA during attention demanding tasks are adaptive, whereas large reductions in RSA mark emotion dysregulation (Beauchaine, 2001).

CENTRAL NERVOUS SYSTEM (CNS) SUBSTRATES OF PERIPHERAL PHYSIOLOGY

One reason that we use autonomic measures with children is because it is much more difficult to perform neuroimaging experiments with them than it is with adults. Furthermore, complex motor sequences such as those required of the star-tracing task are difficult to execute in a scanner. Nevertheless, autonomic responding does provide for some inferences regarding the CNS substrates of behavior.

Most that has been written about the central mechanisms of RSA concerns the medullary nuclei that innervate the heart via the vagus nerve (see Porges, 2007). These nuclei include both the dorsal motor nucleus and the nucleus ambiguus. Although Porges acknowledges bidirectional feedback between the frontal cortex and the medullary nuclei that constitute the ventral vagal complex, he does not describe which frontal regions likely affect RSA. However, given the consistency with which RSA has been linked to emotion regulation and dysregulation (see Beauchaine, 2001), we might infer which structures modulate vagal control of the heart. Two likely candidates include the amygdala and the ventromedial prefrontal cortex (VMPFC), structures that have been linked consistently with emotion regulation capabilities (Goldsmith, Pollak, & Davidson, 2008). The VMPFC appears to inhibit amygdala activation when individuals actively down-regulate negative emotion. Furthermore, lesions to the VMPFC impair

autonomic responses to emotionally valenced stimuli (Verbane & Owens, 1998). In general, this set of observations may imply that children with higher RSA regulate their responses through effective modulation of amygdala activity by the VMPFC.

EDR also has source nuclei in the medulla (Roy, Sequeira, & Delerm, 1993), with cortical control mechanisms that are variegated and complex. Nevertheless, evidence suggests excitatory influences from the amygdala, and inhibitory influences from the hippocampus (Sequeira & Roy, 1993). Interestingly, these structures comprise much of the septohippocampal system (Gray's behavioral inhibition system), activity of which is often presumed to be indexed by EDR (Fowles, 1988). According to this formulation, high EDR reflects trait anxiety. Thus, children with high EDR might be more sensitive to adverse environmental events. In contrast, children with very little EDR are often insensitive to environmental contingencies, placing them at risk for externalizing behaviors.

AUTONOMIC PSYCHOPHYSIOLOGY AND BEHAVIOR

One assumption of those who engage in autonomic psychophysiology work is that peripheral measures reflect tendencies toward certain classes of functional (or dysfunctional) behavior. Here I consider the behavioral correlates of EDR and RSA and how those might interact with one other and with marital conflict. Note that when I refer to EDR, I mean both baseline and reactivity, and when I refer to RSA, I mean both RSA and RSA reactivity. This is because all reported interactions were of the same form, whether baseline or reactivity measures were used.

In the context of marital conflict, low EDR was protective when coupled with high RSA, and high EDR was protective when coupled with low RSA. Considering the former relation first, low EDR presumably marks low septohippocampal system activity, expressed psychologically as low trait anxiety (Gray & McNaughton, 2000). Ordinarily, this would be a risk factor for externalizing behaviors because children low in trait anxiety are less concerned about the consequences of their actions than their peers. However, low trait anxiety is also likely to render a child less sensitive to adverse environmental events, including marital conflict. In addition, high RSA, particularly in emotionally evocative situations, likely marks strong top-down emotion regulation capabilities (Beauchaine, 2001), effected through a CNS network including the amygdala and the VMPFC. Thus, these children may be doubly protected from the consequences of marital conflict because they are less sensitive to it as a result of low trait anxiety and because they are well regulated.

Following the same line of reasoning, the second low-risk “group” of children (high EDR and low RSA) is especially interesting because their ANS pattern implies both high trait anxiety and deficient emotion regulation. Such a child would be especially sensitive to marital conflict yet have little capacity for self-regulation. This combination should place the child at risk for *internalizing* disorders, not externalizing disorders. Indeed, given exposure to family adversity (paternal antisocial personality symptoms, maternal depression), we found that high cardiac preejection period reactivity (a marker of SNS responding) and low RSA predicted child depression (Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007). El-Sheikh and colleagues did not analyze internalizing outcomes, but it may be that children with high EDR and low RSA are only protected from externalizing forms of psychopathology. If the authors collected data on internalizing symptoms, it would be interesting to see those data analyzed.

Considering the high-risk “groups,” children with low EDR and low RSA would seem to be at greatest risk for externalizing psychopathology from a brain-behavior perspective. These children are low in trait anxiety and poorly regulated. As the authors note, low EDR has been found in many studies of externalizing children and adults (Lorber, 2004). Without strong emotion regulation capabilities, as indexed by RSA, these children may be especially likely to learn and use aversive behaviors, including the externalizing outcomes measured by the authors. Thus, the fact that these children are at high risk in the context of marital conflict comes as little surprise.

From the perspective taken thus far, the link between externalizing outcomes, marital conflict, high EDR, and high RSA is the most difficult to explain. Although these children should be high in trait anxiety, placing them at risk for internalizing disorders, they should also be well regulated. Yet in the studies reported in this monograph they exhibit higher externalizing behaviors than their peers. It will be interesting to see whether future studies yield similar results. One possibility is that this group of children, with both high EDR and externalizing conduct, is predominantly female.

SEX DIFFERENCES IN ANS-BEHAVIOR RELATIONS

Although the authors correctly note that they did not have enough statistical power to examine sex effects, ANS-behavior relations often differ for boys versus girls. In our own research, we have found that, in contrast to findings derived from male samples, both cardiac PEP and RSA fail to differentiate aggressive girls from controls, and that aggressive girls score *higher* than controls on baseline EDR (Beauchaine, Hong, & Marsh, 2008).

This is consistent with El-Sheikh, Keller, and Erath's (2007) work indicating that marital conflict predicted externalizing behaviors among girls—especially among those with high EDR reactivity. In contrast, marital conflict predicted externalizing behaviors among boys with low EDR reactivity. Given such sex effects, and given that the authors' three samples were comprised of roughly equal numbers of boys and girls, it is all the more impressive that they were able to detect the predicted interactions. In future studies it will be interesting to see whether the same interactions apply to both boys and girls or whether one sex was driving some of the observed results more than the other.

CONCLUDING REMARKS

At the beginning of this commentary I noted that the authors' research likely marks the beginning of a new generation of studies evaluating vulnerabilities and resiliencies to marital conflict. The three studies presented provide a consistent set of findings linking ANS functioning to marital conflict and externalizing behaviors. As with all quality research, the findings described in this monograph also pose questions for future investigation. Perhaps most notably, how do SNS \times PNS interactions moderate links between marital conflict and internalizing outcomes, which may be more likely to affect girls than boys? Second, what are the implications of this work for the prevention of emerging externalizing (and internalizing) behaviors among children exposed to marital conflict? Clearly, the most optimal outcome is to successfully intervene with parents to reduce marital conflict directly. But what about children who have already been exposed and developed well-canalized behavior problems? Are current interventions sufficient to reduce problem behaviors among such children, or do new interventions need to be developed? Should interventions differ based on whether ANS coinhibition or coactivation is observed? It is increasingly recognized that biological vulnerabilities should be taken into account when devising prevention and intervention programs (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). For example, one might postulate that children who display a pattern of coinhibition should be assigned to interventions that target emotion regulation skills. This is because low trait anxiety, indexed by SNS functioning, appears to be more stable than emotion regulation capabilities, indexed by PNS functioning. Indeed, PNS activity and emotion regulation skills may be especially amenable to change in response to environmental events (Beauchaine et al., 2007). Given the impressive productivity of the El-Sheikh research group, and given the increasing attention being paid to Biology \times Environment interactions among developmental psychopathologists, there is little doubt that these questions and others will be addressed in the upcoming years.

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