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COMMENT

Smith and Lilienfeld's Meta-Analysis of the Response Modulation Hypothesis: Important Theoretical and Quantitative Clarifications

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In the first meta-analytic review of the response modulation hypothesis (RMH), an attention-based model for understanding the etiology of psychopathy, Smith and Lilienfeld (2015) report that the average effect size for response modulation deficits in psychopathic individuals fell in the small to medium range (r = .20; $p < .001$, d = .41). Moreover, support for the RMH extended to both psychopathy dimensions, applied across diverse assessments and settings, and spanned child, adult, female, and male samples. The analysis also revealed good empirical support for a central tenet of the RMH, namely that response modulation deficits are not limited to the processing of threat or other emotion stimuli. Unfortunately, the Smith and Lilienfeld meta-analysis contains several theoretical and quantitative problems, including failing to distinguish adequately between the tasks used to evaluate RMH predictions and the theory itself, confusion regarding the evolution of the RMH and its impact on effect sizes, misinterpretations of RMH predictions and evidence regarding dominant response sets, passive avoidance, and primary task performance, and biased statements promoting the low fear model over the RMH. In this response, we endeavor to reduce misunderstanding by addressing the most salient issues, with the hope that increasing clarity will sharpen the focus of future research and result in more valid assessments of the RMH.

Keywords: effect size, meta-analysis, psychopathy, response modulation hypothesis

After years of investment in the response modulation hypothesis (RMH), it was gratifying to find this work the focus of a thorough review in *Psychological Bulletin*. We are pleased that support for the RMH was found across diverse psychopathy assessments, research settings, and laboratory measures, including emotionally neutral as well as emotion-related measures. Moreover, support for the RMH spanned male and female samples, child and adult samples, and investigators with different degrees of allegiance to the model. According to Smith and Lilienfeld (2015) "the estimated effect size for the relation between response modulation (RM) deficits and psychopathy was not insubstantial, falling in the small to medium range (r = .20; d = .41)" (p. 51). Although the Smith and Lilienfeld study makes a valuable contribution, we are concerned that a number of misrepresentations leave the reader with misimpressions regarding the specifics of the RMH, the quality of the evidence supporting it, its limitations, and its status relative to the low fear model. In this response to their article, we address the most salient theoretical and quantitative issues.

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Response modulation entails interrupting an ongoing response or prepotent focus of attention to accommodate new information and, if necessary, revise behavior. According to the RMH, the cognitive, affective, and behavioral abnormalities associated with psychopathy reflect a deficiency in the instinctive integration of diverse processing streams. The RMH does not identify a particular brain region (e.g., amygdala, dorsolateral prefrontal cortex) or conventional aspect of attention (e.g., cognitive control, attention switching) as responsible for the psychopathic deficit (Blair & Mitchell, 2009). In our view, the attention abnormalities manifested by psychopathic individuals are relatively unique and reflect abnormal brain processes that have yet to be fully characterized (Hamilton, Hiatt Racer, & Newman, 2015). Nevertheless, the theory postulates a dysfunctional psychological mechanism, which in turn, generates specific and testable hypotheses that are distinct from other psychopathy models. In developing the RMH, our focus has been to evaluate these hypotheses, refine the parameters of the model, and explore the limits of its predictive validity rather than develop specific well-validated measures of the response modulation construct, per se. This strategy has important implications for Smith and Lilienfeld's meta-analytic evaluation of the RMH.

The Use of Meta-Analysis to Evaluate the RMH: Problems Distinguishing Task Effects From Theory

For a meta-analysis to provide an accurate and constructive evaluation of the RMH, the data used to generate effect sizes must

accurately address the core predictions and relevant aims of the model. Assuming that the core affective, cognitive, and behavioral deficits associated with psychopathic individuals are attributable to their difficulty processing secondary information (i.e., poor response modulation), it follows that these deficits will be reduced or eliminated under conditions that minimize demands for response modulation. Most research on the RMH has proceeded using experiments that evaluate this hypothesis by manipulating demands for response modulation across conditions. To the extent that the RMH accurately predicts the circumstances under which psychopathy-related deficits are observed, the heuristic value of the model is substantiated. Rather than evaluate these between-condition experimental predictions, Smith and Lilienfeld focus entirely on the size of psychopathy-related deficits (i.e., only one condition). Although this approach appears to address the RMH because the deficit condition is thought to involve response modulation, it is inappropriate for two reasons: (a) the tasks used to evaluate the RMH were generally selected because of their importance to the field, rather than their quality as measures of response modulation, and (b) because the meta-analysis focused on single conditions rather than contrasting performance across conditions. The implications of conflating task performance with theoretical predictions may be more readily understood using concrete examples related to passive avoidance, tasks tapping the low fear deficit, attention tasks assessing response modulation, and tasks designed to test the boundary conditions of the RMH.

First, one of the earliest studies of the RMH focused on the poor passive avoidance of psychopathic offenders (Newman & Kosson, 1986). Using a task adapted from Moses, Ratliff, and Ratliff (1979), participants were given the opportunity to press a button while two-digit numbers appeared on a computer monitor. Participants won money for pressing while some numbers were presented, but lost money for pressing while other numbers were presented. Although participants were instructed to win as much money as they could, they also had to learn to inhibit punished responses (i.e., passive avoidance) to preserve their earnings. Because poor passive avoidance learning was regarded as one of the most important and well-replicated findings in the field and the task appeared to require response modulation (interrupting a dominant set for reward to learn passive avoidance), we predicted that psychopathic participants would perform more poorly on this task. More importantly, we contrasted performance in this reward-punishment task with performance on a well-matched punishment-only task that was designed to reduce demands on response modulation. *A propos* to Smith and Lilienfeld's meta-analysis, we did not regard performance in the reward-punishment task as a discerning test of the RMH because most major theories of psychopathy predict a passive avoidance deficit and it is impossible to know whether their poor performance on the task relates to response modulation or other factors. However, in contrast to other theories, the RMH predicts that the passive avoidance deficit of psychopathic individuals will be moderated by demands for response modulation. Thus, it is the fact that psychopathic participants committed significantly more passive avoidance errors in the reward-punishment task but performed as well as controls in the punishment-only task that provides support for the utility of the RMH. This is a classic way to test theory. To the extent that the results support the situation-specific predictions of the RMH, the findings advance understanding and support the utility of the model.

Second, using this approach, we investigated many of the deficits associated with the low fear model, including passive avoidance learning, skin-conductance response to threat cues, fear-potentiated startle, and amygdala activation (see Newman & Baskin-Sommers, 2012 for review). Contrary to the logic implied by the Smith and Lilienfeld meta-analysis, we do not equate performance in the selected tasks (e.g., fear conditioning) with response modulation. In experiments investigating the situation-specific nature of low fear deficit, we theorize that the low fear reflects a problem of response modulation and evaluate this hypothesis by examining responses to the same stimuli under well-matched experimental conditions that reduce demands for response modulation. Thus, it is the difference between the experimental conditions that addresses the validity and utility of the RMH. Ironically, when Smith and Lilienfeld estimate the magnitude of the psychopathy-related deficit in passive avoidance, fear-potentiated startle, skin conductance response to threat cues, or amygdala activation during instructed fear conditioning, the average effect size pertains to the fear deficit, which the field has identified as the primary deficit of interest in psychopathy. To the extent that the average effect size of the meta-analysis is perceived as disappointingly small, the problem reflects the average effect size of the deficits that are of general interest to the field as much as any shortcoming of the RMH.

Although the deficits examined by Smith and Lilienfeld may reflect psychopathy-related deficits in response modulation, the validity of the RMH is better evaluated using experimental procedures that manipulate response modulation as opposed to fear per se (e.g., CS+ vs. CS− in fear conditioning). To address such predictions, Smith and Lilienfeld would have needed to examine psychopathy by condition interactions or compare performance across conditions in some other way, but they did not. In fairness to Smith and Lilienfeld, we also have reservations about evaluating such effects in a meta-analysis. First, interaction effect sizes are notoriously small even when they are associated with statistically and clinically significant effects (McClelland & Judd, 1993). Second, the magnitude of such interactions and other cross-condition comparisons is still limited by the task selected for study and the effect size of the psychopathy-related deficit being investigated. Thus, even if a response modulation manipulation completely eliminates psychopathy-related deficits in threat sensitivity, such effects will reflect the strength of the original fear deficit as much or more than the strength of theory. When a theory-based experimental manipulation eliminates psychopathy-related deficits in passive avoidance learning (Newman & Kosson, 1986), fear-potentiated startle (Newman, Curtin, Bertsch & Baskin-Sommers, 2010), and amygdala activation (Larson et al., 2013), it attests to the power and clinical significance of the theory. However, these qualities of the theory are not addressed by the meta-analytic approach employed by Smith & Lilienfeld and, thus, the analysis appears ill suited to evaluating the RMH.

Third, the preponderance of research on the RMH has focused on fear and other emotion deficits for the purpose of evaluating clinically significant predictions generated by the model rather than measuring response modulation per se. However, there are at least two major exceptions: the card perseveration task (Newman, Patterson & Kosson, 1987), which was originally developed to assess response modulation and the modified Stroop and Flanker tasks, which were used to assess response modulation in the

absence of emotion stimuli (e.g., Hiatt, Schmitt & Newman, 2004; Newman, Schmitt & Voss, 1997; Zeier, Maxwell, & Newman, 2009; Zeier & Newman, 2013). According to Smith and Lilienfeld, the average effect sizes for the card and reaction time (RT) interference tasks are .35 (d = .747) and .28 (d = .583), respectively. Such findings suggest that psychopathy-related differences in response modulation may be more robust when using tasks that were developed or selected to measure response modulation. Moreover, they raise the possibility that the strategy of testing theoretical predictions using previously established (e.g., low fear) deficits facilitates theory development at the expense of generating large effect sizes in a meta-analysis.

Finally, inspection of the effects reported by Smith and Lilienfeld reveal other ways in which the goal of maximizing effect size in a meta-analysis may be at odds with theory development. Over the years, our lab has conducted a number of high-risk studies to develop the RMH (e.g., characterize its underlying mechanisms) and specify its limitations. Such studies are desirable from a theoretical point of view, but hurt the average effect size reported by Smith and Lilienfeld. For example, investigators had theorized that the poor response modulation of psychopathic individuals was attributable to their exaggerated sensitivity to reward cues (Arnett, Smith & Newman, 1997; Gorenstein & Newman, 1980; O'Brien & Frick, 1996; Quay, 1993). To evaluate this hypothesis, we examined whether psychopathic offenders, like other disinhibited individuals, are hyper-reactive to reward and the degree to which such hyper-reactivity is linked to deficient response suppression (Newman, Patterson, Howland & Nichols, 1990). Our results contradicted the hypothesis that psychopathy is associated with exaggerated sensitivity to reward cues and, thus, the proposal that it was responsible for the response modulation deficits of psychopathic individuals. Although highly informative on a theoretical level, the negative effect size for these comparisons had a strong negative impact on Smith and Lilienfeld's evaluation of the RMH. Given an apparent conflict between theory development and playing it safe to maximize effect size differences, we opted to be governed by theoretical rather than effect size considerations.

In other attempts to refine our theory we employed alternative performance measures to examine the boundary conditions of the RMH and/or bridge to other models of psychopathy. For example, Arnett, Howland, Smith, & Newman (1993) added an 8-s intertrial interval to the basic passive avoidance task to examine whether putative psychophysiological indicators of Gray's behavioral approach and avoidance systems would clarify the response modulation deficit as proposed by Fowles (1988). The task modifications required to measure psychophysiology eliminated the typical passive avoidance deficit, and, though informative, had a substantial negative effect (r = −.25) on Smith and Lilienfeld's analyses. Additionally, we often included auxiliary psychophysiological assessments and evaluated alternative hypotheses while testing more clear-cut experimental hypotheses in order to generate additional information and clarify the limits of the model (e.g., Arnett, Smith & Newman, 1997; Baskin-Sommers, Curtin, & Newman, 2013 LPP measure; Newman et al., 1990 Experiment 3; Newman, Wallace, Schmitt, & Arnett, 1997). Because Smith and Lilienfeld combined these results with those testing our principal hypotheses when reporting effect sizes in Table 2, the effect sizes reported are often much smaller than they would have been if the authors had played it safe and only focused on the most clear-cut conditions and hypothesis.

In addition to employing a variety of exploratory measures to define the RMH, our lab has employed a strategy of conducting separate studies to evaluate the generalizability of its findings with European American samples to African American and female samples. There are multiple reasons why laboratory findings with European American samples may not generalize to these other samples. The reasons are not specific to the RMH, but are likely to be more problematic when evaluating etiological hypotheses than personality correlates (Brinkley, Newman, Widiger & Lynam, 2004; Vachon, Lynam, Loeber, & Stouthamer-Loeber, 2012). This is why Cleckley (1976) and others went to great lengths to distinguish true or primary psychopathy from related syndromes that may reflect different etiologies. To the extent that psychopathy is more strongly related to adverse social conditions in African Americans as we have proposed (Brinkley et al., 2004) and psychopathy in women is associated with lower intelligence and high neurotic anxiety as we have found (see Vitale et al., 2011; Vitale, Smith, Brinkley & Newman, 2002), the etiology-related correlates of psychopathy in these groups may be expected to differ. Consistent with this point of view, we have had difficulty replicating expected psychopathy effects in African American and female inmates. These failures to replicate in African American and female samples include core psychopathy deficits such passive avoidance learning, emotion facilitation on lexical decision tasks, and instructed fear conditioning (Anton et al., 2012; Baskin-Sommers et al., 2011; Kosson, Smith & Newman, 1990; Lorenz & Newman, 2002; Newman & Schmitt, 1998; Vitale et al., 2005, 2011). Although these studies are very important from a theoretical point of view, Smith and Lilienfeld's meta-analysis counts these findings as failures of the RMH.

Summary

The Smith and Lilienfeld meta-analysis provides an interesting and informative review of the various tasks that have been used to assess the importance of response modulation for psychopathy-related deficits. However, these tasks were selected primarily to tap established psychopathy-related deficits to test the between-condition hypotheses generated by the RMH. Unfortunately, Smith and Lilienfeld give short shrift to these theory-based predictions of the RMH.

Assessment of RMH Evolution: The Relationship Between Theory and Effect Sizes

To address the evolution of the RMH, Smith and Lilienfeld propose, "that effect sizes drawn from later variations of the RMH (will) yield larger effect sizes than early variations . . . if later variations better capture the deficits of psychopathy" (p. 20). Here too, the authors appear to equate the validity of theoretical predictions with the mean effect size for the tasks used to test the predictions. From a theoretical perspective, understanding the mechanism responsible for an effect is crucial and relates to a theory's ability to make more precise and far ranging predictions. For example, following the initial development of the RMH, it became possible to predict that the insensitivity of psychopathic individuals applies to nonaffective as well as to affective stimuli

under comparable circumstances (those requiring response modulation). This represents a powerful extension of the theory, but we would not expect the average effect size for the nonaffective stimuli to be larger than the effect size for affective stimuli because both effects are presumed to reflect same mechanism.

Furthermore, even if the updated models improved the specificity of the RMH, it is unlikely that Smith and Lilienfeld's analyses would detect such differences. Updated versions of the RMH have clarified the circumstances under which reward contingencies will and will not engender response modulation deficits. Moreover, they have clarified how the degree of overlap between primary and secondary focus and time constraints affect the quality of response modulation in psychopathic individuals. In light of these developments, updated versions of the RMH would not predict deficits on a number of tasks, such as the pattern-matching task employed by Newman et al. (1990) or the BART task, which rely on reward focus alone to establish a dominant response set, or tasks used by Kosson and colleagues that involve switching attention between explicit goal-relevant stimuli. Although dropping these and other effects that are not predicted by the updated models would increase the average effect size associated with the RMH, to observe the increase it would be necessary to recompute the total effect size using all studies that satisfy the revised criteria, a strategy that was not employed by Smith and Lilienfeld.

Summary

We disagree with Smith and Lilienfeld's assumptions that improved versions of the RMH will necessarily be associated with larger effect sizes and that the incremental contributions of the revised models may be evaluated by focusing only on the new tasks associated with these models.

Confusion Surrounding a Dominant Response and Its Relationship to Passive Avoidance Tasks

Smith and Lilienfeld claim that problems operationalizing the definition of dominant response sets may preclude a priori determination of when this component of the model has been satisfied. A dominant response set refers to the prepotent focus of attention in a particular context. Given their response modulation deficit, this focus largely determines when psychopathic individuals will and will not process information. Thus, the definition and operationalization of the dominant response set concept has fundamental importance for the RMH. Because it is a theoretical construct, the dominant response set lends itself to disagreements regarding the quality of its operationalization, but Smith and Lilienfeld's concerns are exaggerated by misunderstanding and a failure to appreciate the distinction between theory-related speculation and a priori experimental manipulations.

For example, according to the RMH, the dominant response set created by Lykken's (1957) manifest maze task is sufficient to undermine learning of his latent passive avoidance task in psychopathic individuals. This interpretation of Lykken's manifest-latent manipulation is post hoc, but it represents a theoretical, rather than arbitrary, claim. Similarly, it is theoretical speculation to propose that the RMH can account for the psychopathy-related deficit on the lexical-decision task (e.g., Williamson, Harpur & Hare, 1991) by postulating that the word identification aspect of the task creates a dominant response set, which in conjunction with an attention bottleneck undermines emotion facilitation. Nevertheless, it is fair to propose that the RMH can explain these deficits in light of experimental evidence in similar situations. This is how good theory works, and it is reasonable to offer such interpretations as long as the claims are ultimately testable.

In fact, such theory-based proposals are supported by subsequent research involving a priori experimental manipulations of the dominant response set. Using a priori manipulations of the dominant response set under well-matched experimental conditions, we have rigorously evaluated the role of dominant response sets in moderating psychopathy-related deficits in passive-avoidance learning, flanker interference, instructed fear conditioning, amygdala activation, and other dependent variables (e.g., Baskin-Sommers et al., 2011; Larson et al., 2013; Newman & Kosson, 1986; Newman et al., 1985, 2010; Zeier et al., 2009, 2013). In each case, the experimental manipulation is clearly specified so that readers can evaluate the extent to which it reflects a valid manipulation of the dominant response set construct.

Rather than evaluate the numerous experimental manipulations that have been used to operationalize the dominant response set construct, Smith and Lilienfeld elected to evaluate the importance of dominant response sets using a reward pretreatment that our lab had employed to strengthen a reward focus manipulation in early work with our go/no-go passive avoidance task. Based on this analysis, the authors reported that "effect sizes for the go/no-go task did not differ significantly regardless of the pre-treatment condition (Reward pretreatment: $r = .09$; No pretreatment: $r = .15$), although they were in the opposite direction from that predicted by the RMH." (p. 40) and they concluded, "contrary to the RMH, we detected no evidence that research designs that incorporated more pronounced response sets . . . yielded more marked RM deficits" (p. 22).

The authors' decision to evaluate the importance of dominant response sets using the pretreatment procedure is difficult to understand for several reasons. First, the authors ignore our lab's major program of research (described above) that has used carefully constructed experimental manipulations to evaluate the impact of dominant response sets. Instead, the authors focus on a procedure that our lab has never used as an experimental manipulation (i.e., we never manipulated the presence vs. absence of this procedure to evaluate its effect on the performance of psychopathic participants). Second, the pretreatment procedure was designed to strengthen, rather than establish, a dominant response set for reward. Although early studies of passive avoidance learning demonstrated that psychopathic offenders over-focused on reward cues (e.g., Newman & Kosson, 1986), the reward pretreatment, consisting of 4 to 18 trials, was used to enrich the schedule of reward with the goal of strengthening the dominant response set. In other words, the procedure was designed to be incremental and, thus, it would be inaccurate to interpret the effect as reflecting the full impact of dominant response sets on the performance of psychopathic individuals. Third, in our program of research that evaluates the presence versus absence of a dominant response set, we examine the effect of this manipulation within a study using carefully matched experimental conditions. Conversely, Smith and Lilienfeld examined the effects of the pretreatment procedure using data from separate studies.

Even though we strongly disagree with Smith and Lilienfeld's decision to evaluate our dominant response set construct using the pretreatment procedure, we were surprised to learn that the effects of the pretreatment procedure were opposite rather than incremental, as we would expect based on the RMH. Thus, we requested and received from the authors the list of studies used in this analysis. The compiled list included 28 studies, 16 involving a reward pretreatment and 12 with no pretreatment. Attempts to verify the studies showed that at least 7 of the 16 studies in the pretreatment group either did not employ the go/no-go passive avoidance task or did not use a pretreatment. Moreover, the effect sizes reported by Smith and Lilienfeld did not match those reported in some of the articles. Using the nine legitimate pretreatment studies and correcting two erroneous effect sizes,[1] we calculated the effect size for the psychopathy-related difference in passive avoidance in the pretreatment condition to be .224. After eliminating three of the 12 studies from the no-pretreatment list because they did not use the go/no-go passive avoidance task, the average effect size for these studies was .190. Thus, contrary to Smith and Lilienfeld's claim, the effect size for pretreatment studies (.224) is directionally larger than the effect size for no pretreatment studies (.190), rather than in the opposite direction. For all of these reasons, we find the authors' conclusions regarding the importance of dominant response sets in psychopathy to be inaccurate.

Inspection of these data (rs = .224 and .190) also sheds light on the disappointing passive avoidance evidence (r = .09) highlighted by Smith and Lilienfeld. While quoting MacCoon et al. (2004, p. 329) to establish that passive avoidance tasks are especially relevant for the RMH, the authors claim that go/no-go tasks "are deemed to be quintessential measures of passive avoidance learning" (p. 57). This claim is highly misleading. Our research group has never predicted deficits in generic go/no-go performance. In fact, the reward-only and punishment-only conditions commonly used as control conditions in our passive avoidance studies, and for which no differences have ever been expected, are go/no-go tasks (e.g., Newman et al., 1985, 1997). In contrast to the generic go/no-go task, the go/no-go passive avoidance task involves learning. The performance deficit postulated by the RMH is based on the premise that psychopathy involves a problem in this learning process, namely a failure to interrupt goal-directed behavior (i.e., pause), reflect, and learn from punishment feedback (Patterson & Newman, 1993). By contrast, the generic go/no-go task uses clearly defined stimuli, such as up-arrows for go stimuli and down-arrows for no/go stimuli; and thus, there is no reason to pause and reflect after a mistake because participants already know the predefined rule. In light of these circumstances, a response modulation deficit is expected to have little or no effect on generic go/no-go performance. Thus, collapsing across generic and passive avoidance go/no-go tasks to evaluate passive avoidance learning is inappropriate and our reanalysis of Smith and Lilienfeld's data is consistent with this claim. Using data presented in Table 2 for which we were able to determine that the go/no-go passive avoidance task was used, and that the effect size pertained to passive avoidance learning as opposed to some other dependent measure, we found the effect size to be .215. By contrast, the effect size for generic go/no-go tasks averaged −.026. For Table 2 effect sizes that collapsed across diverse dependent measures, making it impossible to evaluate passive avoidance, the average was .058. Including data from the mental maze task (r = .313) provides even stronger support for the predicted passive avoidance deficit.

Summary

Dominant response sets pertain to an individual's processing priorities. Because a dominant response set is an abstract concept, it can be difficult to identify and quantify after the fact. However, dominant response sets may be clearly operationalized, rather than inferred, using explicit manipulations of attention-related focus. In light of clear experimental manipulations of dominant response sets, it strikes us as grossly inappropriate to define and evaluate dominant response sets using the reward pretreatment procedure. Furthermore, the use of generic go/no-go tasks to evaluate RMH predictions regarding passive avoidance learning deficits is inappropriate and distorts the evidence both for the effects of reward pretreatments and the magnitude of passive avoidance learning deficits in psychopathy. When focusing on appropriate experimental manipulations and tasks, the effect sizes supporting the RMH are considerably larger than those reported by Smith and Lilienfeld.

Can the RMH Explain Psychopathy-Related Deficits on Primary Tasks?

In this section, we address the special case of a dominant response set that distinguishes between primary and secondary components of a primary task. According to Smith and Lilienfeld, "one major challenge to the RMH derives from study designs that contain emotionally laden stimuli but do not attempt to establish a clear-cut dominant response set" (p. 22). Although most tests of the RMH use explicit manipulations to operationalize the dominant response set, this is not the only way to establish a dominant response set that curtails the processing of secondary information.

A good example involves the picture-viewing paradigm in which participants are typically instructed to focus attention on the picture the entire time that it is presented. Despite this primary focus on the picture stimuli, Bradley and colleagues (2007) have demonstrated that the processing of affective pictures proceeds in stages. Because picture processing creates a perceptual load, a picture must be processed to a significant degree before a participant's response to its affective content can be measured using startle responses. To the extent that picture perception is prepotent (i.e., primary) as suggested by Bradley and colleagues, this would constitute a dominant response set (i.e., prepotent focus of attention). Furthermore, owing to their attention bottleneck, the RMH predicts that this dominant response will, differentially (though temporarily), reduce emotion processing and emotion-modulated startle in psychopathic individuals. This is not a statement of fact, but a post hoc theoretical prediction that requires experimental substantiation. To this end, Baskin-Sommers et al. (2013) employed the picture-viewing paradigm while manipulating attention-related demands associated with picture processing, by making some pictures familiar and some novel (Ferrari et al., 2011). As predicted, psychopathic individuals displayed the expected deficit in emotion-modulated startle while viewing novel pictures, and the

[1] Most of the invalid studies included in this list involved go/no-go tasks rather than the go/no-go passive avoidance task, but there were also more basic inaccuracies in the identification of relevant studies. The two mistakes involved (a) using an effect size from an irrelevant study by Vitale, Newman, Serin, & Bolt (2005) study listed in Table 2 as opposed to a pertinent study by Vitale et al. (2005) and (b) an incorrect combination of effect sizes from Newman et al., 1990, in which only one of the experiments involved the reward pretreatment.

deficit was significantly reduced in the familiar picture condition, which required less perceptual processing (see also Sadeh & Verona, 2012 for a manipulation using picture complexity).[2]

The above findings are consistent with the proposal that an attention bottleneck results in slower, more sequential, processing of information in psychopathic offenders (see also, Hamilton, Hiatt Racer, & Newman, 2015; Hamilton & Newman, 2015; Sadeh & Verona, 2008) and that this bottleneck temporarily inhibits the processing of affective information (Newman & Baskin-Sommers, 2012). Contrary to Smith and Lilienfeld's claim, then, the evidence that psychopathic individuals show deficient processing of primary information in some contexts is not damning. Rather, the attention bottleneck version of the model provides a testable explanation for the difficulty that psychopathic individuals experience when their primary task involves complex, multicomponent (e.g., picture) stimuli.

Using essentially the same logic, the attention bottleneck model can account for the continuous flash suppression (CFS) results that Smith and Lilienfeld regard as particularly problematic for the RMH. In this paradigm, "one eye receives a dynamic, continually changing (every 20 milliseconds) flow of stimuli, usually Mondrian images, and . . . the other eye receives faces displaying emotional expressions, such as fear or happiness. For a few seconds, the facial expression is suppressed from visual awareness by the dynamically changing stimuli and then briefly 'breaks through' this suppression into awareness" (p. 62). In comparison with the target emotion faces, which are initially presented at 0% intensity (i.e., delayed), the Mondrian images are four times larger and initially shown at 100% intensity. These circumstances establish the Mondrian images as a prepotent focus of attention (i.e., early dominant response set) and the continuously changing images increase the likelihood that attention will continue to be actively engaged. The fact that it took an average of 3.45 seconds for the faces to "break through" (Sylvers et al., 2011) supports our view that the Mondrian images created strong demands on perception and attention during the so-called "preattentive" period that limited face processing. As in the picture-viewing task, the Mondrian images create a prepotent perceptual load that inhibits the processing of secondary emotion cues. Although Smith and Lilienfeld may consider face processing to be the primary task owing to task instructions, research by Baskin-Sommers et al. (2011) demonstrates that the first information presented will receive attention regardless of task instructions. We do not agree with Smith and Lilienfeld that the CFS findings cannot be explained by the RMH because "the facial stimuli emerged from suppression too rapidly to engage attention" (p. 62), especially because it took more than 3 seconds for the faces to break through. In our view, attention was engaged by the Mondrian images and these findings support rather than refute the proposal that an attention bottleneck precludes processing of secondary information in psychopathic individuals.

Summary

A dominant response set need not involve experimental instructions. Even while directing attention to a complex, multidimensional stimulus, psychopathic individuals will have difficulty processing the nondominant dimensions (e.g., emotion cues in the lexical-decision task), unless the information overlaps with their dominant response set (e.g., standard color-word Stroop task; see Hiatt et al., 2004; MacCoon et al., 2004). The claim that psychopathy-related deficits on primary tasks cannot be explained by the RMH reflects Smith and Lilienfeld's incomplete understanding of the RMH and the data supporting it. Though our explanation for the Sylvers et al. (2011) findings is post hoc, a good theory of psychopathy should not be limited to a priori hypothesis testing; it should attempt to account for all important findings in the field. Moreover, as shown by Baskin-Sommers et al. (2011), when such post hoc explanations are amenable to rigorous experimental investigation, they are capable of advancing theoretical understanding.

The Role of Stimulus Valence in Evaluating the RMH

The RMH predicts psychopathy-related deficits in response to secondary stimuli that elicit a reliable response in nonpsychopathic individuals regardless of affective valence. As noted by Smith and Lilienfeld, this prediction is unique to the RMH and distinguishes it from the low fear model. In support of the prediction that response modulation deficits are not constrained by stimulus valence, Smith and Lilienfeld found that psychopathy-related effects for motivationally neutral tasks were directionally larger than those for emotion deficit tasks (p. 18).

Although these findings provide robust differential support for the RMH versus low fear model, the authors later minimize this important finding, stating, "other findings in the psychopathy literature are less consistent with the RMH" (p. 23). To support their claim, they cite a study by Lorber (2004), which found psychopathy-related differences in skin conductance were greater in response to aversive stimuli than to neutral stimuli. Smith and Lilienfeld suggest that this finding is inconsistent with the RMH prediction because "deficits among psychopathic individuals should emerge even in the presence of motivationally neutral stimuli" (p. 23), but their interpretation of these data is confusing and incorrect.

The RMH predicts a situation-specific deficit in processing secondary information, but it does not predict that psychopathic individuals are less adept at processing neutral information regardless of the experimental context. For the deficit to be measured, it follows from the theory that the information must be secondary and it stands to reason that the information must elicit a meaningful response for differences in the magnitude of the response to be measured. In light of the fact that nonpsychopathic participants did not display a reliable skin-conductance response to the neutral stimuli that were used as an experimental control in the Lorber study (i.e., a floor effect), it would be difficult or near impossible for psychopathic participants to be less responsive. To conduct a proper test of this prediction of the RMH, it would be necessary to use secondary stimuli that are roughly equivalent in their ability to elicit a significant response in nonpsychopathic individuals. This point is also important when testing RMH predictions using positive affect stimuli, which typically elicit weaker reactions than fear and other negative affect stimuli. Although the goal of eliciting meaningful reactions appears to be most readily achieved using salient emotion cues, it can also be achieved using neutral

[2] Although Smith and Lilienfeld's Table 2 lists the effect size for this study to be .08, Sadeh and Verona (2012) found a much larger effect size (r = .26) for the predicted association between PCL:SV affective-interpersonal traits and emotion modulated startle in the high complexity condition.

stimuli made salient by their incongruity with primary stimuli (e.g., Zeier et al., 2009).

Summary

The RMH predicts psychopathy-related deficits in response to motivationally neutral, as well as emotionally significant, secondary cues, and the evidence supports this prediction. However, a reasonable test of this prediction must use secondary emotional and neutral stimuli that are salient enough to elicit measurable responses in nonpsychopathic individuals[3]; otherwise, the test of the model is undermined by a floor effect (i.e., absence of an effect in either group). Overall then, the Smith and Lilienfeld analysis provides solid support for this key prediction of the RMH and, as far as we can tell, the proposed counterevidence does nothing to diminish this support.

Comparisons With the Low Fear Model

A recurrent theme in the Smith and Lilienfeld review concerns comparison of the RMH with the low fear model. The authors imply that the small to moderate effect size found for the RMH suggests that other models may provide more effective means of understanding psychopathy. However, it is impossible to draw inferences regarding the relative merits of alternative models without also considering the mean effect size of competing models. In light of Smith and Lilienfeld's frequent, but selective, comparison of the response modulation and low fear models, we examined previously published results and meta-analyses for the low fear model.

Reflecting concerns about support for the overall low fear model, Hoppenbrouwers, Bulten, and Brazil (2016) parsed results for automatic versus conscious threat processing in an attempt to boost support for the low fear model. Even so, the average effect sizes were .21 (k = 18) and .097 (k = 16) for automatic and conscious threat processing, respectively (weighted average = .157). Another recent meta-analysis by Dawel et al. (2012) yielded effect sizes of .153 (k = 21) and .333 (k = 5) for processing fear faces and vocal stimuli, respectively (weighted average = .191). A meta-analysis by Wilson et al. (2011) yielded an average effect size of .10 for fear faces. Moreover, although Smith and Lilienfeld's own analyses included both fear and other emotion stimuli, their results showed that, "tasks with emotional content (r = .17, k = 99) yielded similar effect sizes to those of neutral tasks (r = .20, k = 38)" (p. 42). Although the RMH and low fear models differ in many respects, average effect size does not appear to be one of them. In light of such findings, Smith and Lilienfeld's tendency to focus on weaknesses of the RMH to explain modest effect sizes appears to be misplaced.

At a theoretical level, both the low fear and response modulation models recognize the importance of threat processing deficits for explaining psychopathic behavior. However, they differ on two key dimensions: First, the low fear model posits a pan-situational deficit in threat processing, whereas the RMH posits a situation-specific deficit in threat processing. Second, low fear model predictions are necessarily limited to fear (threat)-related stimuli, whereas the RMH predicts performance anomalies involving a wider range of secondary stimuli. Smith and Lilienfeld note these key differences and imply that they are the most relevant criteria for contrasting the models, but they do not follow through on the point. Despite considerable evidence supporting the differential predictions of the RMH, their discussion focuses on idiosyncratic concerns and counterexamples of questionable relevance as illustrated by the following examples.

Evaluating the situation-specific nature of their deficit, Newman and Kosson (1986) found that psychopathic offenders displayed a significant passive avoidance deficit when correct go responses were rewarded and incorrect no-go responses were punished, but they performed as well as controls when both incorrect go and no/go responses resulted in punishment (i.e., their avoidance deficit was not pan-situational). Smith and Lilienfeld dismiss the significance of this result because Newman and Kosson used monetary punishments. This criticism is misleading because they ignore multiple conceptual replications that used electric shocks (e.g., psychopathic offenders were relatively unresponsive to shock-related stimuli if they were secondary to goal-relevant stimuli, but their deficit was significantly reduced or eliminated completely when the same threat cues were primary; Baskin-Sommers et al., 2011; Larson et al., 2013; Newman et al., 2010). Notably and ironically, Smith and Lilienfeld also overlook concerns about the use of monetary punishments when discussing our card perseveration task. Defending the low fear model, they propose that: "Lykken (1995) and other proponents of the low fear hypothesis would presumably suggest that psychopathic individuals were unresponsive to the shifting reinforcement contingencies because of an absence of fear of punishment." (p. 5). These and other statements belie the authors' systematic and dispassionate comparison of the models.

The other "key differential prediction" (p. 26) noted by Smith and Lilienfeld is that the RMH model predicts response modulation deficits for neutral as well as emotion stimuli, whereas the low fear model does not. According to the authors, the prediction that psychopathic individuals should display "similar effect sizes for tasks that are both motivationally neutral and those that are motivationally laden" (p. 8) was well supported by their meta-analysis (p. 21). Moreover, consistent with the proposal that these effects reflect the same processes, psychopathy-related deficits in processing motivationally neutral secondary cues, like those involving secondary emotion cues, are reduced to nonsignificance when attention is directed to the secondary stimuli (e.g., Zeier et al., 2009).

[3] The importance of this point is clearly illustrated in another study cited by Smith and Lilienfeld. Using electrodermal responses to conditioned and unconditioned stimuli as the dependent variable, Hare and Quinn (1971) reported a significant psychopathy-related difference in a condition involving electric shocks and a nonsignificant difference in a second condition using pictures of nude females rather than electric shocks as the unconditioned stimulus. Although this finding also seems to support the importance of affective valence in revealing psychopathy-related differences, inspection of the data suggests a different interpretation. Whereas psychopathic participants were equally unresponsive to the conditioned stimuli across the two conditions, controls displayed a larger response to the shock-related stimuli than the nude-related stimuli. Such data suggest that the likelihood of finding psychopathy-related deficits is closely tied to the ability of experimental stimuli to elicit a strong response in controls (as opposed to changes in the responsiveness of psychopathic participants).

Summary

According to Lilienfeld (2004), an "etiological model of psychopathy . . . should account not merely for findings that are generated by its advocates, but also for the larger corpus of well-replicated findings in the literature" (p. 25). In light of the fact that the RMH has addressed diverse emotion, inhibitory, language, memory, and attention deficits in psychopathy and the low fear model's difficulty accounting for such deficits, including nonfear emotions, the RMH appears to provide a more parsimonious theoretical explanation for the psychopathy syndrome. Moreover, meta-analytic studies suggest that effect sizes for the RMH are as large or larger than those found for the low fear model, even while generating more specific and comprehensive predictions than the low fear model.

Assessment of Publication Bias

Smith and Lilienfeld evaluated publication bias by comparing 77 published studies with 13 unpublished studies and found a significant difference in effect size ($r = .23$ vs. $r = -.01$). Including studies with multiple samples, we could find only 11 unpublished studies in Table 2. Of these 11, three used the generic go/no-go tasks and two used the BART tasks, which we do not regard as valid response modulation tasks for reasons noted in previous sections. Of the remaining six studies, two other effect sizes pertained to African American samples that were selectively extracted from Schmitt's (2000) dissertation, presumably because his data for European Americans participants were published elsewhere. Moreover, with regard to estimating adjustments for "missing" findings (p. 36), Smith and Lilienfeld acknowledge that the "Egger's test of the regression intercept became nonsignificant" when one unusually large study was omitted from the analysis.

Summary

In Smith and Lilienfeld's analysis of publication bias, the unpublished studies do not constitute a representative sample, the number of legitimate studies used to evaluate publication bias is very small, eliminating one study was sufficient to eliminate the evidence for publication bias, and eliminating inappropriate studies would reduce the evidence for bias even further, suggesting that this finding is not a very reliable or robust one. Additionally, we are disappointed that the authors chose not to report the fail-safe statistics reported in earlier drafts of their article, which demonstrated the substantial reliability of psychopathy-related differences in response modulation.

Conclusions

Understanding the emotion, inhibitory, and self-regulation dysfunctions of psychopathic individuals has been a long-time research priority in the field of psychopathy. For this reason, proponents of the RMH have used the model to clarify these dysfunctions, rather than develop and evaluate specific measures of the response modulation process. More specifically, instead of quantifying response modulation deficits in psychopathy, research on the RMH has evaluated theory-driven predictions regarding the factors that *moderate* known psychopathy-related deficits. Although theoretically and clinically important, Smith and Lilienfeld do not evaluate these predictions of the RMH. Though overlooked in the current meta-analysis, there is abundant evidence concerning the ability of attention-related manipulations to alter the expression of psychopathic deficits across multiple domains (Newman & Baskin-Sommers, 2012). Moreover, the power of these and other attention manipulations to moderate core deficits in psychopathy is increasingly recognized by other psychopathy researchers (e.g., Decety, Chen, Harenski & Kiehl, 2013; Dadds, Perry, Hawes et al., 2006; Meffert, Gazzola, den Boer, et al., 2013; Moul, Killcross & Dadds, 2012). Theories are useful when they help to organize existing facts about a phenomenon, generate novel and valid predictions, and provide a more parsimonious explanation for the facts than competing explanations. Research on the RMH has been conducted with these fundamental standards in mind and fares well by these criteria. By specifying the contextual variables that moderate psychopathy-related deficits, the literature on response modulation allows for the development of new cognitive-affective interventions and prevention strategies to address these deficits (see Baskin-Sommers, Curtin & Newman, 2015; Dadds et al., 2012).

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