Psychological Bulletin
2015, Vol. 141, No. 6, 1145–1177

0033-2909/15/$12.00 http://dx.doi.org/10.1037/bul0000024

The Response Modulation Hypothesis of Psychopathy: A Meta-Analytic and Narrative Analysis

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The causes of psychopathy, a condition characterized by interpersonal (e.g., superficial charm), affective (e.g., lack of empathy), and behavioral (e.g., impulsive actions) features, remain contested. The present review examines 1 of the most influential etiological models of psychopathy, the *response modulation hypothesis* (RMH), which proposes that psychopathic individuals exhibit difficulties in adjusting their behavior in the presence of a dominant response set. We conduct a meta-analysis and narrative literature review to examine the RMH quantitatively and qualitatively, estimate the statistical effects of response modulation (RM) deficits in psychopathic individuals, and ascertain the boundary conditions of the RMH. Ninety-four samples from published and unpublished studies involving 7,340 participants were identified for inclusion. Overall results provided some support for the RMH, revealing a small to medium relationship between psychopathy and RM deficits ($r = .20$, $p < .001$, $d = .41$) that extended to both psychopathy dimensions. Moreover, as predicted by the RMH, RM deficits were observed for both affectively neutral and affectively laden tasks. A number of moderators, such as anxiety, laboratory task, dependent measure, psychopathy measure, and race, contributed to significant variability in effect sizes; we also found evidence for potential publication bias using 2 methods, raising questions concerning the robustness of RM findings. An ancillary narrative review revealed that the RMH is inconsistent with a number of replicated findings in the psychopathy literature, suggesting that the RMH, at least in its present form, is unlikely to provide a comprehensive etiological account of psychopathy. Nevertheless, more recent attentional versions of the RMH may hold promise with respect to intervention. Further fruitful directions for research on the RMH, including the use of multiple dependent measures of RM and latent variable approaches, are delineated.

Keywords: psychopathy, response modulation hypothesis, etiology

Psychopathic personality, or psychopathy, is a condition characterized by interpersonal features such as superficial charm and grandiosity, affective features such as lack of empathy and callousness, and behavioral features such as impulsivity and antisocial behavior (Hare, 1991/2003). In a classic book, *The Mask of Sanity*, Cleckley (1941/1988) described the psychopath as a hybrid creature. He or she, usually he, displays a façade of likability, a "mask of sanity" that conceals marked deficits in empathy, guilt, and interpersonal attachment. Cleckley delineated 16 features he believed to be central to psychopathy, including superficial charm, lack of anxiety, absence of psychotic/neurotic symptoms, egocentricity, lack of remorse or empathy, incapacity for love or close relationships, poor impulse control, irresponsibility, and unmotivated antisocial deviance. Others have described psychopathy in more menacing ways, positing such features as "lovelessness" and "guiltlessness" as the crux of the disorder (McCord & McCord, 1964). Despite longstanding historical efforts to delineate the key features of psychopathy (Hare & Neumann, 2008), the etiology of this condition remains a subject of intense controversy (Skeem, Polaschek, Patrick, & Lilienfeld, 2011).

In the present manuscript, we examine the scientific status of one of the most widely researched and influential etiological models of psychopathy, namely, the *response modulation hypothesis* (RMH). According to the RMH, the core deficit of psychopathy is cognitive in nature. Although the RMH has passed through a number of incarnations since its introduction in the early 1980s, it has been consistent in its central presuppositions. Specifically, the RMH proposes that once engaged in a dominant response set, psychopaths' attentional focus becomes unduly narrowed, precluding adequate processing of extraneous stimuli, including—but not limited to—punishment (Patterson & Newman, 1993). Before examining the RMH in detail, however, we discuss key conceptual challenges bearing on the operationalization of psychopathy.

Conceptual Issues in Psychopathy

Potentially hindering progress on the etiology of psychopathy has been conceptual disagreement concerning the nature and boundaries of this condition (Lewis, 1974). Contributing to this confusion is the relation of psychopathy to its presumed counterpart in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*), Antisocial Personality Disorder (ASPD; Arrigo & Shipley,

This article was published Online First August 24, 2015.

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We would like to acknowledge Dylan Jordan, Wenting Kang, and Katie Delgado for their invaluable work with identifying and coding studies included in this review and Irwin Waldman, PhD, Patricia Brennan, PhD, and Harold Gouzoules, PhD, for their valued consultations on the review.

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2001). The two disorders are sometimes considered to be synonymous, an erroneous belief perpetuated by the Section II text of the *DSM–5*, which states that "this pattern [ASPD] has also been referred to as psychopathy" (American Psychiatric Association, 2013, p. 659). Nevertheless, although ASPD and psychopathy are moderately correlated (Hare, 1991/2003), the two conditions are conceptually and empirically separable. Conceptually, ASPD is characterized primarily by a severe and chronic pattern of behavior that violates social norms (e.g., lying, stealing, cheating, and abusing animals; American Psychiatric Association, 2013), whereas psychopathy is operationalized largely in terms of personality dispositions (Berg et al., 2013; Lilienfeld, 1994). Like individuals with ASPD, those with psychopathy sometimes engage in antisocial behavior (Hare, 1991/2003). Empirically, psychopathy differs from ASPD in being characterized by more pronounced interpersonal and affective features, especially physical and social boldness (the latter of which encompasses superficial charm and poise) and perhaps callousness (Lilienfeld, 1994; Venables, Hall, & Patrick, 2014). Nevertheless, at least some of the behavioral deficits associated with ASPD, such as recklessness and criminal behavior, may stem in part from the same callousness and impulse control deficits found in psychopathy (Anderson, Sellbom, Wygant, Salekin, & Krueger, 2014).

Psychopathy: A Two-Factor Model

In an effort to address the long history of conceptual disagreements regarding the clinical criteria for psychopathy, Hare (1980) developed a now well-validated assessment procedure, the Psychopathy Checklist (PCL), for measuring the personality traits and antisocial behaviors associated with the disorder. Subsequent investigations of the PCL and several other measures of psychopathy led to the more recent conceptualization of the disorder within the context of an oblique two-factor model (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Harpur, Hakstian, & Hare, 1988; Harpur, Hare, & Hakstian, 1989). Prior to these studies, psychopathy was viewed primarily as a global trait, often assessed by a total score on the PCL or other measures. Nevertheless, early factor analyses of the PCL and its revision, the PCL-R, often revealed a replicable two factor structure, with each factor comprising two facets (Hare, 1991/2003; Harpur et al., 1988).

The first factor, termed Factor I, consists primarily of the affective and interpersonal traits outlined by Cleckley (1941/1988; see Harpur et al., 1989). These traits include glibness, egocentricity, lying, manipulativeness, lack of remorse and empathy, and a failure to accept responsibility for one's actions. Factor I is associated with related traits such as narcissism, interpersonal dominance, and low levels of anxiety (Harpur et al., 1989; Neumann, Johansson, & Hare, 2013). As mentioned previously, two underlying facets load on this factor, namely, the interpersonal and affective facets of the PCL. In contrast, Factor II is associated primarily with irresponsibility, impulsivity, a lack of behavioral controls, and persistent antisocial and criminal behaviors (Hare, 1991/2003). This factor is associated with a "chronically unstable and antisocial lifestyle" (Harpur et al., 1989, p. 6). In addition, this factor is more behaviorally based than is Factor I and strongly resembles criteria used to assess ASPD in the *DSM–5* (American Psychiatric Association, 2013). Indeed, empirically, Factor II psychopathy is closely related to ASPD (Hare, 1991/2003; Lilienfeld, 1998).

A two-factor model often emerges when using both the PCL and alternative psychopathy measures, such as the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996). The PPI is a widely used self-report measure designed to assess the traits of the disorder as described by Cleckley (1941) and other early theorists. Unlike the PCL, the PPI does not overtly assess antisocial behavior, but instead focuses on internal dispositions associated with psychopathy. Nevertheless, a two factor structure also emerges from factor analyses of the PPI (Benning et al., 2003; but see Neumann, Malterer, & Newman, 2008 for an alternative factor structure). Like the PCL factors, the dimensions of the PPI display differential correlates. The first factor of the PPI, termed *Fearless Dominance* (PPI-I), is positively associated with physical boldness, emotional stability and adjustment, social dominance, and immunity to stress. Although these adaptive correlates help to distinguish PPI-I from PCL Factor I, some view psychopathy largely or exclusively as a maladaptive condition, and have called into question the conceptual relevance of fearless dominance to the construct of psychopathy (Miller & Lynam, 2012; but see Lilienfeld et al., 2012, for a rebuttal). In contrast, the second factor of the PPI, *Self-Centered Impulsivity* (PPI-II), is negatively associated with stability, adjustment, and stress immunity, and positively associated with externalizing behavior (Benning et al., 2003).

This differential pattern of associations seen across dimensions of psychopathy raises questions regarding the global assessment of the condition by means of total scores and bears implications for its multidimensionality (Lilienfeld, Watts, Francis Smith, Berg, & Latzman, 2014). Indeed, some authors have conjectured that psychopathy is a configuration of largely independent personality traits, a conjecture supported by at least some psychometric data (Lilienfeld & Fowler, 2006). If psychopathy is not unitary, the efforts to identify a single etiology for this condition may be misguided (Lilienfeld et al., 2014), a possibility to which we return.

Primary Versus Secondary Psychopathy

In addition to growing support for the conceptualization of psychopathy as a nonunitary construct, a large body of research points to the existence of separable subtypes of psychopathy. Karpman (1941) was one of the first to suggest the existence of variants, or at least phenotypic mimics, of the disorder. He delineated two major variants, which he termed primary and secondary psychopathy (see also Blackburn, 1975; Lykken, 1995). Karpman posited that primary psychopaths are marked by a core affective deficit that is largely genetic in nature. In contrast, secondary psychopaths are characterized by an emotional disturbance that is the consequence of environmental factors, such as child abuse, neglect, or deviant peer influence. Central to Karpman's primary/secondary psychopathy distinction is trait anxiety. According to Karpman, the primary psychopath, much like the prototypical psychopath described by Cleckley (1941), is characterized by a pronounced deficit in trait anxiety. In contrast, Karpman described secondary psychopaths as neurotic individuals who exhibit elevated trait anxiety. More recently, other theorists have hypothesized that primary and secondary psychopathy comprise different constellations of traits. For example, Levenson, Kiehl, and Fitz-

patrick (1995), following Karpman's lead, proposed that primary psychopaths are marked by emotional detachment, whereas secondary psychopaths are marked by impulsivity.

Etiological Models of Psychopathy

The crucial question of the etiology of psychopathy remains heavily contested. Although several major causal models have been proposed, the field appears no closer to a consensus on the etiology of this condition than it was when the laboratory study of psychopathy was initiated nearly 60 years ago (Lykken, 1957). A better understanding of the causes of psychopathy may help researchers parse this heterogeneous construct and shed light on the sources of its covariation with other features of psychopathology. Moreover, elucidating the causes of psychopathy may ultimately aid in the development of greatly needed treatment and prevention efforts.

Over the years, several prominent models of psychopathy have emerged. Relatively early models such as those of Quay (1965) and Zuckerman (1978) explained psychopathy largely as a disorder of excessive sensation seeking. According to these models, psychopathic individuals are characterized by cortical hypoarousal, in turn leading to "stimulus hunger" and a desire to take risks, including antisocial activities. Nevertheless, these models have largely fallen out of favor in light of suggestions that the chronic underarousal observed in psychopathy may be a consequence rather than a cause of emotional deficits, such as fearlessness or broader affective detachment (e.g., Fowles, 1980). Over the past two decades, two broad models have emerged as forerunners of explanation of the origins of psychopathy, the low fear and response modulation (RM) models.

Low Fear Model

The *low fear model*, proposed by Lykken (1957, 1995), points to a relative absence of fear as the core developmental precursor of the disorder. This model proposes that psychopathic individuals are marked by inadequate fear, which in turn gives rise to the other major features of the condition, such as superficial charm, lack of guilt, risk-taking, and failure to learn from punishment. A number of studies have offered at least some support for this hypothesis (see Lykken, 1995) by indicating that psychopaths do not exhibit marked conditioned fear responses (e.g., skin conductance responses), lack anticipatory responses to aversive stimuli, display deficits in passive avoidance learning (learning to avoid behaviors that often result in punishment), and exhibit diminished fear-potentiated startle responses (Hare, 1965a, 1965b; Hare & Quinn, 1971; Hetherington & Klinger, 1964; Patrick, Bradley, & Lang, 1993; Schachter & Latane, 1964; Schmauk, 1970). Blair's (2001) more recent and closely allied *violence inhibition* model proposes that because of amygdala deficits, psychopathic individuals do not experience adequate levels of fear and allied negative emotions in response to others' distress. Because these emotions typically inhibit aggression in normal individuals, psychopathic individuals are chronically prone to engaging in violence toward others.

At the same time, not all data are consistent with the low fear model (e.g., Newman & Brinkley, 1997). For example, low levels of self-reported fear and harm avoidance are largely unassociated with global psychopathy (Schmitt & Newman, 1999), raising questions regarding Lykken's (1995) conjecture that fearlessness is a key source trait (Cattell, 1957) underpinning psychopathy. Moreover, even when self-reported fear and the broader dimension of Constraint (Tellegen, in press) are significantly correlated with certain psychopathy dimensions, they tend to be selective to the unique variance in PCL-R Factor II (the antisocial lifestyle features of psychopathy) rather than to PCL-R Factor I (the core interpersonal and affective deficits of psychopathy; Benning et al., 2003), suggesting that fear deficits may not account adequately for many of the key features of psychopathy outlined by Cleckley (1941/1988).

Response Modulation Hypothesis (RMH)

Gorenstein and Newman (1980) proposed a now influential alternative to Lykken's low fear explanation of psychopathy. The authors looked to animals with septal and hippocampal lesions to aid in the interpretation of human conditions, such as psychopathy and attention-deficit/hyperactivity disorder, that are characterized by disinhibition. Drawing comparisons between the behavioral consequences of septally lesioned animals, on the one hand, and psychopaths and other disinhibited individuals, on the other, Gorenstein and Newman underscored the utility of examining the cognitive and psychological abnormalities that serve as a link between humans and nonhuman animals exhibiting inhibitory deficits. Like psychopaths, septally lesioned animals display impairments on tasks that are presumably related to an ability to control impulses, particularly passive avoidance tasks. In one study, septally lesioned cats that were trained to approach a food dispenser began receiving electric shocks from the same dispenser. The lesioned cats did not learn to inhibit their behavior and continued to approach the food despite the shocks. In contrast, nonlesioned cats quickly learned to avoid the shock-rigged food bowl (McCleary, 1966).

Gorenstein and Newman (1980) highlighted important features of the aforementioned task and those such as Lykken's (1957) mental maze task, which requires participants to learn the correct path through a sequence of lever presses while receiving electric shocks in response to certain incorrect choices. These tasks require individuals to avoid punishment when engaging in a competing goal (e.g., problem solving through a maze, trying to obtain a reward such as food). In these scenarios, punishment becomes peripheral information, secondary to goal-directed behavior. Interestingly, septally lesioned animals demonstrate intact avoidance of punishment when the avoidance response is the only requirement (Morgan & Mitchell, 1969). Gorenstein and Newman interpreted this phenomenon as a "heightened sensitivity to reward" (p. 312) rather than as a deficit in passive avoidance. The authors suspected that psychopathic individuals, like septally lesioned animals, exhibit avoidance deficits only in the presence of a competing reward.

Newman, Widom, and Nathan (1985) were among the first to address the possibility of hypersensitivity to reward in psychopathic individuals. They proposed that psychopathic individuals, like septally lesioned rodents (Donovick, Burright, & Bengelloun, 1979), are particularly inattentive to other cues in the environment, including punishment, once they are focused on a goal, such as obtaining a reward. In later years, the model was expanded to encompass motivationally neutral cues from the environment, as

well as punishment. Thus, according to Newman et al., deficits in passive avoidance learning should emerge only when cues for an approach response predominate over other environmental cues that might indicate that a change in behavior is called for. The authors examined this hypothesis using a go/no-go discrimination task in which participants were presented with competing approach contingencies (i.e., reward) and avoidance contingencies (i.e., punishment). Two versions of the task were used. In both versions, participants were exposed to a series of cards bearing numbers. Participants were instructed to learn by trial and error when to respond (by touching the card) or not respond (by refraining from touching the card). In the first task, some numbers yielded monetary reward when tapped whereas other numbers yielded monetary loss when tapped. In this paradigm, passive avoidance errors occurred when participants' responded to a number that resulted in punishment (i.e., loss of monetary reward). In the second task, participants received rewards for tapping correct numbers and for *not t*apping incorrect numbers. No punishment was administered and participants were rewarded for appropriate response inhibition. This task was used to assess general learning and to determine if psychopathic individuals can inhibit a response when receiving an equivalent reward for response inhibition.

Participants were assessed for psychopathy using both the Psychopathic deviate and Welsh Anxiety scales of the Minnesota Multiphasic Personality Inventory (MMPI); the latter measure was used to group participants in accord with the primary/secondary psychopathy distinction (Karpman, 1941). Specifically, participants were subdivided into low anxiety psychopaths (presumably primary psychopaths), low anxiety comparison participants, high anxiety psychopaths (presumably secondary psychopaths), and high anxiety comparison participants. To test the hypothesis that psychopathic individuals, specifically primary psychopaths, demonstrate a hypersensitivity to reward, or are deficient in their ability to suspend goal-directed behavior in anticipation of punishment, Newman et al. conducted planned comparisons of the mean number of passive avoidance errors committed by low anxiety psychopaths (primary psychopaths), high anxiety psychopaths (secondary psychopaths), and low anxiety controls. Consistent with hypotheses, the primary psychopathy group committed significantly more passive avoidance errors than did both comparison participants and secondary psychopaths on the first task, which comprised competing reward and punishment contingencies. Secondary psychopaths did not differ from comparison participants in the number of passive avoidance errors. In the second task, no significant difference in task performance emerged across groups. The authors took these findings as evidence that primary psychopathic individuals are deficient in their ability to inhibit goal-directed behavior in anticipation of reward. According to their account, the drive for reward becomes so dominant that psychopaths ignore other cues, including cues of punishment, which might suggest that an alteration of behavior is warranted. In contrast, when an inhibition of goal-directed behavior does not compete with the motivation to obtain a reward (i.e., inhibition is rewarded), an approach response does not become dominant and psychopathic individuals show normal response inhibition.

Newman and colleagues further developed these findings into a more comprehensive etiological theory of psychopathy termed the RMH. According to Newman (1998), RM involves a shift of attention from goal-directed behavior to the evaluation of environmental cues or feedback. In normal individuals, attention is shifted periodically to attend to environmental cues. If behavior is deemed appropriate (e.g., because of a reward) it is continued or if deemed inappropriate (e.g., because of a punishment), it may be suspended. According to this model, psychopathic individuals demonstrate difficulties in adjusting their behavior in the presence of a dominant "response set," that is, an overriding way of reacting to the environment. This response set is often, but not necessarily, cued by rewarding stimuli.

This model departs from traditional models of psychopathy, which point to motivational or emotional deficits, such as a heightened threshold for experiencing fear, as the root causes of the disorder. In particular, the RMH contrasts with that of Lykken's (1957, 1995) low fear model in several of its predictions. We focus on two primary points of divergence between these models here. First, the low fear hypothesis predicts that psychopathic individuals will fail to respond to cues of punishment or anxiety-provoking stimuli across a wide range of scenarios. In contrast, the RMH posits that psychopathic individuals will show deficits in punishment learning *only* when faced with competing reward contingencies or other dominant response sets. In this respect, the RMH is more specific in its postulation of deficits compared with the low fear model. Second, in contrast to the low fear model, the RMH proposes that in the presence of a dominant response set, psychopathic individuals will exhibit deficits in responding to extraneous stimuli that are neutral as well as those that are potentially fear-inducing or punishing. In this respect, the RMH is more general in its postulation of deficits compared with the low fear model. The writings of Gorenstein and Newman (1980) and Newman et al. (1985) spawned an early body of research aimed at identifying deficient RM in psychopathic individuals.

Newman, Patterson, and Kosson (1987) took on this task, proposing that psychopaths' failure to respond to punishment was not due to their lack of fear, as proposed by Lykken (1957). Instead, the authors hypothesized that this behavior stems from response perseveration. Response perseveration can be defined as a tendency to continue engaging in a response set despite environmental cues, such as punishment, that would indicate that this response is no longer adaptive. Seventy-two participants were divided into groups of psychopaths (score of 32 or above) and nonpsychopaths (score of 20 or below) using cut-offs on the PCL (Hare, 1980). Participants engaged in a card-playing task. In the beginning of the task, when a participant played a card there was initially a high rate of reward (i.e., 5 cents). As the task continued, the probability of reward progressively decreased while the probability of punishment (i.e., losing 5 cents) increased. The authors predicted that because of the initial high rate of reward, a dominant approach response set (i.e., desire to play a card) would be established. According to the RMH, normals should adjust their response set throughout the task, responding to the increased rate of punishment. In contrast, psychopathic individuals should perseverate on the dominant response, not accounting for the changing punishment contingencies as the task went on. This trend would be evidenced by the number of cards played, with psychopathic individuals playing more cards than normals, thereby earning less money.

Results were consistent with hypotheses; psychopathic individuals played significantly more cards and earned significantly less money than did normals. Of interest to the authors, in an additional

experimental condition in which participants were forced to pause after receiving punishment feedback, the response perseveration of psychopathic individuals diminished. Newman et al. (1987) took these findings as further evidence of deficient RM in psychopathic individuals. Furthermore, findings that a forced pause after feedback diminished response perseveration in psychopaths suggested that forced reflection was an effective way to interrupt psychopaths' dominant response sets and increase their attention to extraneous cues, such as punishment. Although these results support the RMH, they do not provide evidence against the low fear hypothesis. Instead, 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.

Other early studies on RM targeted deficient passive avoidance and sought to determine the boundary conditions under which psychopathic individuals display such deficits. As mentioned previously, some researchers have invoked lack of sensitivity to punishment cues and the accompanying failure to learn from punishment as hallmark features of the disorder (Lykken, 1995). Newman and Kosson (1986) sought to test an alternative hypothesis for passive avoidance deficits observed in psychopaths. They proposed that psychopaths are indeed capable of learning from punishment, but only when avoidance of punishment is established as a dominant response and does not compete with a reward contingency. Participants were 60 White male inmates divided into groups of psychopaths and nonpsychopaths based on a cut-off score on the PCL. The experimental task was almost identical to the go/no-go task used in the Newman et al. (1985) study. In the first trial, participants were rewarded with money for correct responses and punished with loss of money for incorrect responses. Nevertheless, unlike the Newman et al. (1985) study, in a second trial of the task, participants started with a certain amount of money and were merely punished for incorrect responses. No rewards were given for correct responses.

Like the Newman et al. (1985) study, in the first trial of the task, psychopathic participants were expected to establish a dominant response set toward goal-directed behavior aimed at achieving a reward. In turn, the psychopathic participants would ostensibly be so distracted by this drive for reward that they would ignore other environmental information, primarily the fact that approach responses were sometimes punished. Thus, in the condition combining reward and punishment contingencies, psychopaths were expected to display slower learning, as indicated by a higher rate of passive avoidance errors compared with that of comparison participants. Newman and Kosson (1986) hypothesized that in the absence of a competing reward contingency, psychopathic participants would not establish a dominant response set for approach behavior, and thus would perform as well or better than controls on the punishment-only version of the go/no-go task. As predicted, psychopathic individuals made significantly more passive avoidance errors than did nonpsychopaths in the first trial of the go/no-go task, which involved both punishment and reward contingencies. In contrast, psychopaths did not differ from nonpsychopaths in the punishment-only condition. The researchers interpreted the results of this and the Newman et al. (1985) study as indicative of the specificity rather than generality of psychopaths' decreased responsiveness to punishment. According to them, deficits in passive avoidance in psychopaths are specific to situations in which competing goals of approach and avoidance behavior are present.

The absence of group differences in the punishment-only condition, therefore, seemingly runs counter to Lykken's (1995) low fear model. Nevertheless, the extent to which Newman and Kosson's study affords an adequate test of the low fear model is unclear, as the loss of relatively modest amounts of money may not trigger much fear in *nonpsychopaths*. Indeed, for such a task to afford an adequate test of the low fear model, it must be fear-inducing for the comparison group. Moreover, previous work had found that psychopaths can learn well, and perhaps at least as well, as nonpsychopaths, from monetary punishment (Schmauk, 1970), probably because they are motivated to avoid losing money, an outcome that is in their self-interest. In contrast, psychopaths' fear deficits may not motivate them strongly to avoid electric shock, social disapproval, or other aversive stimuli.

Other studies sought to examine the mechanisms rather than the consequences of RM deficits in psychopaths. Newman, Patterson, Howland, and Nichols (1990) suspected that dominant response sets for reward were resistant to interruption in psychopaths because of a decreased amount of time spent reflecting on negative feedback, such as punishment. By failing to pause and absorb environmental information, psychopaths do not learn to adjust or modulate their responses. This provocative hypothesis was tested using a go/no-go task with combined reward and punishment contingencies described earlier (Newman et al., 1985). When participants responded to stimuli, they received feedback indicating whether their response was correct. Participants were required to press a button to terminate the feedback and continue with the task. The amount of time elapsed before terminating feedback was used as an indication of reflection. Consistent with the results of previous studies, low anxious psychopaths (as determined by cut-off scores on the PCL and Welsh Anxiety Scale) committed significantly more passive avoidance errors than did low anxious nonpsychopaths. Furthermore, as predicted, low anxious psychopaths paused for significantly less time following punishment than did low anxious controls. The authors interpreted these findings as evidence that low anxious psychopaths are unmotivated to suspend a dominant response set for reward in the presence of punishment cues. According to Newman et al., this decreased reflection on punishment, or processing of environmental cues by reallocating attention, might help to explain psychopaths' failure to learn from punishment.

In a review article on syndromes of disinhibition, Patterson and Newman (1993) defined RM in more general terms as a shift of attention from the implementation of goal-directed behavior to the evaluation of that behavior. Until the late 1990s, much of the research on RM centered on passive avoidance deficits and the reaction to punishment cues in psychopaths. To better distinguish their model from the low fear hypothesis, Proponents of the RMH sought to examine the generalizability of the model to motivationally neutral stimuli, particularly stimuli that do not involve punishment. As they noted, the low fear model does not predict deficits in the presence of motivationally neutral stimuli, whereas the RMH does.

In one study, Newman, Schmitt, and Voss (1997) evaluated the sensitivity of psychopathic individuals to motivationally neutral contextual cues that are secondary to a primary task, or dominant response set. Participants were 124 minimum security inmates

divided into groups of low and high anxious psychopaths as well as nonpsychopaths using cut-off scores on the PCL-R and the Welsh Anxiety Scale. RM was assessed using a picture-word task created for the assessment of the processing of contextual cues. The task required participants to determine if specific words or pictures were related. First, a contextual display consisting of a drawing and a superimposed word was presented. In this display, the drawing and the word were never related. Second, a test display was presented consisting of either a picture or a word (depending on the trial). Participants were asked to determine if the word or the picture in the contextual display was related to the word or picture in the test display. Thus, participants needed to focus their attention on either the word or the picture in the contextual display, and ignore the other component of the display. On some trials, the component of the contextual display to be ignored was conceptually related to the test display whereas the component to attend to was not. On such "test" trials, normal individuals would be expected to display interference, as indicated by a slower reaction time (RT). According to the RMH, if psychopaths fail to accommodate contextual information, they would not demonstrate such interference. Indeed, as predicted by the RMH, but not the low fear model, low anxious psychopaths displayed significantly less interference than low anxious nonpsychopaths on such trials. Because this study drew on a laboratory task that is affectively neutral, Newman et al. (1997) interpreted these findings as evidence for the generalizability of the RMH, accounting for deficits in information processing and attention that are not predicted by competing etiological explanations of psychopathy, such as the low fear model. Furthermore, this finding effectively rules out alternative explanations such as a lack of motivation to perform well in psychopathic individuals. For the picture word task, the RMH predicts—counterintuitively—that psychopathic individuals will actually perform better than nonpsychopaths.

Over the years, the RMH has evolved from a relatively specific explanation of passive avoidance deficits to an account of psychopaths' cognitive abnormalities. Currently, the poor RM observed in psychopaths is often conceptualized by proponents of the RMH as a cognitive or information processing deficit, whereby psychopaths fail to attend to and accommodate contextual or environmental cues when engaged in a dominant response set (e.g., goal-directed behavior; Newman, 1998). Thus, appropriate RM would entail the interruption of a dominant response set and a shift of attention to contextual cues, an evaluation of behavior, and often a modification of behavior in line with environmental feedback. The dominant response set is typically conceptualized as the primary task or primary focus of attention, whereas contextual or environmental cues involve any stimuli secondary to that task or focus of attention (Newman et al., 1997; Zeier, Maxwell, & Newman, 2009). The stimuli may be punishment-related, but they may also be motivationally neutral. Some dominant response sets are largely automatic, such as an approach response for a reward. In contrast, other dominant response sets are established by encouraging participants to focus their attention on a certain task or aspect of the environment. As noted by MacCoon, Wallace, and Newman (2004), the primary and secondary aspects of a task are best conceptualized on a dimension from very clear operationalizations of RM to ones that are less clear. We return to the issue of the dimensionality of dominant response sets later in the manuscript.

Summary: The Evolution of the RMH

In summary, the RMH has evolved over time since its initial appearance in the literature (see Newman, 2014). In particular, four major stages of the RMH can be noted (see Table 1). The first stage of the model highlighted reward-driven perseveration as the core of psychopathic RM deficits (Gorenstein & Newman, 1980). This early phase of research on the RMH emphasized psychopathic individuals' hypersensitivity to reward, which interfered with their ability to attend and respond to punishment in the face of competing contingencies in paradigms like the go/no-go or gambling tasks (e.g., Newman & Kosson, 1986). Building on the theory of perseveration and reward sensitivity, the second stage of the model was aimed at separating the mechanisms contributing to RM deficits in psychopathy versus other disorders marked by disinhibited behavior. In particular, this iteration of the model suggested that psychopathic individuals showed decreased reflection time after punishment, contributing to RM deficits (Patterson & Newman, 1993).

The first major alteration came in the third stage with the advent of the context-appropriate balance of attention (CABA) model

Table 1
Evolution of the Response Modulation Hypothesis Overtime

Model stage	Description	Major associated publications
Perseveration	Response modulation deficits are due to a hypersensitivity to reward that interferes with the ability to attend and respond to punishment in the face of competing contingencies	Gorenstein and Newman (1980); Newman and Kosson (1986); Newman, Patterson, and Kosson (1987)
Disinhibition vs. reflection	Response modulation deficits are due to a lack of reflection time after punishment	Newman, Patterson, Howland, and Nichols (1990); Patterson and Newman (1993)
Context-appropriate balance of attention (CABA)[a]	Response modulation deficits are due to an inappropriate balance of attention between goal-relevant behavior and secondary information	MacCoon, Wallace, and Newman (2004); Newman, Schmitt, and Voss (1997)
Attentional bottleneck[a]	Response modulation deficits are due to early selective attention, creating a bottleneck that interferes with the processing of subsequent information	Baskin-Sommers, Curtin, and Newman (2011); Newman and Baskin-Sommers (2011)

[a] The CABA and the attentional bottleneck models represent the most recent incarnations of the response modulation hypothesis that encompass attentional abnormalities in response to affectively neutral and affectively laden stimuli; see also Newman (2014).

(e.g., MacCoon et al., 2004), which posits that psychopathic deficits derive from an imbalance between bottom-up and top-down attentional processes. According to this model, once a dominant response set is established (regardless of its affective relevance), psychopathic individuals exhibit difficulty shifting attention to extraneous cues in the environment. The name "context-appropriate balance of attention" emphasizes the fact that in some cases, this imbalance of attention may be adaptive (e.g., studying for a test) whereas in others it may be maladaptive (e.g., a lack of responsiveness to punishment signals; MacCoon et al., 2004). The current version of the model is often referred to by proponents of the RMH as the attentional bottleneck hypothesis (e.g., Baskin-Sommers, Curtin, & Newman, 2013). This hypothesis proposes that psychopathic individuals are marked by early selective attention abnormalities that, once established, preclude the processing of nongoal-relevant information. This hypothesis encompasses and ostensibly explains early findings from the RMH literature while generating new hypotheses. In particular, the model also posits that psychopathic individuals will show deficits in cognitively complex tasks that create an attentional overload (i.e., bottleneck) whereby they fail to process secondary information.

In summary, the RMH has evolved considerably since its inception in the 1980s. Along with the model, the methodological techniques, particularly the modal experimental tasks used to assess RMH, have changed correspondingly. Hence, in our meta-analytic review, we examine whether effect sizes drawn from later variations of the RMH yield larger effect sizes than early variations, as would be expected if later variations better capture the deficits of psychopathy.

Early Challenges to the RMH

The RMH has been periodically criticized, particularly by proponents of the low fear model. For example, Lykken (1995) provided a detailed critique of research on the RMH in psychopaths, highlighting what he regarded as anomalous and at times inconsistent findings. For example, Newman et al. (1985) found that psychopaths committed more passive avoidance errors than did nonpsychopaths on a go/no-go task combining reward and punishment, but performed no better than controls on a go/no-go task involving only rewards. Nevertheless, one might expect psychopaths to display differential performance across the two tasks, given Newman's initial perseveration version of the RMH (see Table 1), although this was not the case. Thus, Lykken interpreted this study as providing only partial support for the RMH.

Lykken (1995) outlined several other criticisms of the evidentiary basis for the RMH, especially the tendency for RMH proponents to inconsistently use trait anxiety to subdivide psychopaths and controls into low anxiety and high anxiety subgroups. Specifically, in some studies (e.g., Howland, Kosson, Patterson, & Newman, 1993; Kosson & Newman, 1986; Newman & Kosson, 1986), researchers used the PCL total score alone to classify participants into psychopathic versus nonpsychopathic groups, whereas in other studies (e.g., Arnett, Howland, Smith, & Newman, 1993; Newman et al., 1990) they used the Welsh Anxiety Scale to subdivide participants into primary (low anxiety) and secondary (high anxiety) subgroups, seemingly when initial group differences were nonsignificant. Newman and Brinkley (1997) responded to these criticisms by contending that although they did not consistently use the Welsh Anxiety Scale to subdivide high PCL scorers into primary and secondary psychopathy subgroups, they have done so in their subsequent work with positive results (Newman and Brinkley, 1997, p. 241). Nevertheless, their team has relied on psychopathy scores alone (i.e., without subdividing participants by trait anxiety) in a number of later studies (e.g., Newman, Curtin, Bertsch, & Baskin-Sommers, 2010). In other studies, investigators have measured trait anxiety but have not reported findings for anxiety subgroups (e.g., Howland et al., 1993). Hence, the extent to which the RMH findings are robust across differing operationalizations of psychopathy requires clarification.

In addition, some of the original RMH findings of Newman and his colleagues have not been replicated by other investigative teams, although the extent to which these replication failures reflect methodological or sampling differences that are extraneous to the RMH itself remains unclear. For example, Howard, Payamal, and Neo (1997) found no evidence for passive avoidance deficits in psychopaths when using a mixed incentive go/no-go task. Furthermore, many of the early studies on RM drew on samples of incarcerated White males. In more recent years, researchers have attempted to extend the RMH to other samples, such as females or Blacks. Nevertheless, these efforts have met with mixed success (e.g., Newman & Schmitt, 1998; Vitale & Newman, 2001). These findings may reflect limitations in the generalizability of the RMH; alternatively, they may suggest that the psychopathy construct itself manifests differently in Whites as opposed to Blacks (Sullivan & Kosson, 2006). For example, the negative environmental experiences (e.g., poverty, prejudice, and exposure to inner city violence) faced by many Blacks may sometimes result in high psychopathy scores in the absence of an underlying psychopathy disposition (e.g., Lykken, 1995). Indeed, race differences in the expressions of psychopathy have emerged in several studies. For example, Kosson, Smith, and Newman (1990) found that measures of impulsivity were less correlated with psychopathy in Blacks than in Whites, raising the possibility that psychopathy scores possess a different meaning across races (see also Thornquist & Zuckerman, 1995). Nevertheless, the robustness of the RMH across races has not yet been subjected to meta-analytic investigation.

As mentioned previously, growing research on psychopathy suggests that the disorder may be multidimensional rather than unidimensional. If so, the substantial reliance on psychopathy total scores to examine RM deficits may be problematic. This could be the case if the RMH were applicable to only certain dimensions of psychopathy, such as disinhibition (see Patrick, Fowles, & Krueger, 2009) or affective deficits. Hence, one goal of the present meta-analysis is to examine whether RMH deficits are more selective to certain features of psychopathy than others. As a reasonably comprehensive model of psychopathy, the RMH predicts that RM deficits should extend to most and ideally all features of this condition.

Furthermore, much of the research on RM (as well as on other psychopathy models, e.g., Blair, Jones, Clark, & Smith, 1997), which subdivides participants into psychopathic versus nonpsychopathic subgroups, implicitly treats psychopathy as a taxonic rather than a dimensional trait. Nevertheless, growing data suggest that psychopathy is underpinned by several dimensions rather than a taxon (e.g., Edens, Marcus, Lilienfeld, & Poythress, 2006). Furthermore, the often variable cut-off scores used to subdivide

participants into groups of psychopaths and nonpsychopaths could either obscure or exaggerate effects, depending on the nature of these cut-offs. Most commonly, the dichotomization of dimensional scales reduces statistical power, making the detection of effects more difficult (Cohen, 1983; MacCallum, Zhang, Preacher, & Rucker, 2002). Alternatively, extreme-groups designs comparing participants from the bottom and top of a distribution, the most frequent design used in the RMH literature (e.g., Vitale, MacCoon, & Newman, 2011), can exaggerate group differences (Preacher, Rucker, MacCallum, & Nicewander, 2005). They can also introduce false positives and difficulties with replicability given that extreme scores tend to be less reliable and more susceptible to regression to the mean than less extreme scores (Preacher, 2015). Furthermore, extreme-groups designs preclude investigators from examining curvilinear effects, especially those in which intermediate scorers on the PCL-R and other psychopathy measures differ qualitatively in their RM performance from low or high scorers. Nevertheless, the extent to which the use of extreme-group designs impacts effect sizes in the RMH literature is presently unknown.

Present Review

The RMH has emerged as one of the most influential etiological explanations of psychopathy. Indeed, according to the *Google Scholar* database, several of the original articles on the RMH, such as Newman et al. (1985), has been cited over 300 times, Newman and Kosson (1986) over 400 times, and Patterson and Newman (1993) over 500 times. Until relatively recently, a dearth of studies on the RMH has derived from independent researchers. Nevertheless, this state of affairs has begun to change over the past decade. For example, a number of researchers unaffiliated with the initial developers of the RMH, including those both within and outside of North America, have recently examined this model in diverse samples (e.g., Brazil et al., 2012; Heritage & Benning, 2013).

The current state of the RMH literature calls for a relatively comprehensive review and evaluation. The aims of the present review are to (a) synthesize the RMH literature in both quantitative and narrative form, (b) estimate the overall magnitude of the relation between RM deficits and psychopathy, (c) evaluate the specificity of the RMH to psychopathy as opposed to other disorders, such ADHD (e.g., Farmer & Rucklidge, 2006), and (d) examine both the strengths and weaknesses of the RMH when evaluated within the context of the broader psychopathy literature. As noted earlier, we explicitly accounted for the evolution of the RMH over time by examining tasks that are ostensibly relatively selective indicators of different versions of this model.

By examining moderators, we also aimed to ascertain the boundary conditions under which the RMH does and does not hold. A robust etiological model of psychopathy should ideally hold across different measures of the condition, demographic characteristics of participants, and experimental tasks.

With respect to point (d) above, we examined the crucial question of the extent to which the RMH accords with other well-established findings in the psychopathy literature, especially results not generated from the RMH framework. An etiological model of psychopathy, like that of all other psychological conditions, 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 (Lilienfeld, 2004).

Particularly controversial in any meta-analytic review of psychopathy are issues of gender and race, as some researchers suggest that psychopathy manifests differently across these categories. For example, some research suggests that psychopathic females show a differential pattern of behavioral correlates (e.g., recidivism) than males (Miller, Watts, & Jones, 2011; Salekin, Rogers, Ustad, & Sewell, 1998; Verona & Vitale, 2006). Indeed, studies from the RMH literature have raised questions about the replicability of RM deficits in females (Vitale & Newman, 2001). As observed earlier, the manifestation of psychopathy across racial groups is similarly a source of controversy (Kosson et al., 1990). Hence, we quantitatively examined the extent to which the RMH holds across gender and race, bearing in mind the caveat that any detected moderator effects might reflect genuine differences in the construct of psychopathy rather than inherent limitations of the RMH per se.

In addition to examining the boundary conditions of the RMH, we attempted to partially disentangle the RMH from its primary competitor, namely, the low fear model (Lykken, 1995). Proponents of the RMH have proposed the hypothesis as a direct alternative to the low fear model. Specifically, they have argued that the RM model is "both more specific and more general than traditional accounts emphasizing low fear or insensitivity to punishment cues" (Newman, Schmitt, & Voss, 1997, p. 564). On the one hand, the RMH is more specific than the low fear model because it does not predict broad abnormalities in fear-related processing, but only fear-related deficits in highly specific situations. According to the RMH, psychopathic individuals should display insensitivity to punishment cues, but only in the context of a competing dominant response set. On the other hand, proponents of the RMH propose that the model is broader than competing hypotheses in that it predicts abnormalities beyond those predicted by the low fear model. Specifically, when a dominant response set has been established, the RMH predicts that psychopathic individuals will show general attentional abnormalities not only in the presence of punishment, but in emotionally neutral contexts as well.

Although the RMH and low fear models cannot be compared directly in the context of this review, an additional aim is to examine differential predictions of these two models. We focus on one key differential prediction in particular. According to the RMH, one would expect similar effect sizes for tasks that are both motivationally neutral and those that are motivationally laden. In contrast, the low fear model predicts higher effect sizes for those tasks rich in emotionally laden, namely fear-inducing, content.

We also attempted to examine the potential impact of study quality and other study characteristics on effect sizes. Proponents of the RMH have acknowledged that different laboratory measures of RMH probably fall along a dimension, with some ostensibly assessing RM deficits better than do others (see MacCoon, Wallace, & Newman, 2004). Nevertheless, these authors have not explicitly stated which measures are more "pure" indicators of RMH than others, rendering the coding of RMH measure quality challenging. At the same time, certain RMH tasks are designed to be better measures of RM compared with others. For example, in the case of the go/no-go task (see "Data Collection and Coding of Moderators"), a number of investigators have introduced a "pretreatment" phase in which "go" stimuli are repeatedly introduced before the primary task (e.g., Newman et al., 1990). This phase is

intended to induce a potent dominant response set in participants, and is therefore presumed to be more sensitive to RM deficits compared with standard go/no-go tasks. Hence, the RMH predicts that tasks that use a pretreatment phase should yield larger effect sizes than do comparable tasks that do not. In addition, the use of self-report measures in the detection of psychopathy has been criticized by some authors given that psychopathic individuals are prone to dishonesty and exaggeration of their positive attributes (Edens, Hart, Johnson, Johnson, & Olver, 2000; but see Lilienfeld & Fowler, 2006, for a different view). In light of this concern, we examined the method of assessment, including the use of interview-based versus self-report measures, as a potential moderator of effect sizes.

Method

Eligibility Criteria

Inclusion criteria for studies in the analyses were prespecified. To be considered for inclusion, studies needed to include measures of psychopathy. Additionally, participants were required to engage in a task assessing RM. It was not required that RM be the primary focus of the study, only that a task frequently used to measure RM deficits (e.g., go/no-go, picture word task, spatially separated Stroop) be administered. A RM task was defined as any procedure requiring participants to engage in a primary task (e.g., dominant response) while processing extraneous information that requires a shift of attention from the primary task. Studies examining participants from any setting (e.g., community, prison, and psychiatric inpatient) and of any age or racial background were included. No restrictions were placed on the type of outcome measures used in the study. The outcome measures used in a study typically depended on the experimental task used and varied across studies. Identical eligibility criteria were used for the identification of unpublished studies. Only master's, dissertation theses, and white papers were included as unpublished studies.

Information Sources and Search Criteria

Studies were identified by searching electronic databases, scanning the reference lists of articles, and by contacting selected experts in the field. No restrictions on publication date were applied to the search. The electronic databases used for the search were PsycInfo and Google Scholar. The final search was conducted on February 1, 2015. The following search terms were used to identify potential studies of interest: psychopathy, sociopathy, response modulation, selective attention, go/no-go, visual search, spatially separated Stroop, attentional focus, passive avoidance, picture word, card perseveration, and attention bottleneck. All variants of the terms psychopathy and sociopathy (e.g., psychopath, sociopath, psychopathic personality, and sociopathic personality) were included in the search. Unpublished studies were identified using the same search terms, which were entered into ProQuest's electronic database specifically for the identification of master's and dissertation theses.

Study Selection

Eligibility assessment of each potential study was conducted by the first author in an unblinded standardized manner. Initial manuscripts retrieved from the search were screened using the title and abstract of the report. After the initial screening process, the remaining manuscripts were reviewed in their entirety for the eligibility criteria.

Data Collection and Coding of Moderators

Extraction of data from the selected manuscripts was completed by the authors and two independent reviewers. Five studies lacking appropriate information for the coding of effect sizes were excluded from the analyses and selected findings from an additional four studies were excluded for the lack of appropriate information to code effect sizes for subsets of the samples (e.g., Black participants). In addition to effect sizes, basic information, used to inform moderator variables in meta-regression analyses, was extracted from each study. This information comprised the characteristics of participants, such as the age of the sample coded categorically (e.g., adult, adolescent, and child) and quantitatively when available (e.g., mean age of the sample), the racial composition of the sample (e.g., percent White, Black, Hispanic, and Asian), and the gender composition of the sample (e.g., percent female and male). Because of scattered reports of psychopathy by-laterality interactions for some laboratory tasks (Lorenz & Newman, 2002), some RMH researchers have examined the handedness of participants. Thus, the composition of handedness of the sample was coded when available (e.g., percent right-handed and left-handed). Included studies were also coded based on the setting from which the sample was recruited. Sample settings were inpatient psychiatric units, prison systems, undergraduate or preparatory school settings, and general community settings.

In addition, each manuscript was coded for the type of psychopathy assessment used. Although the substantial majority of studies used variants of the PCL-R, a number used alternative modes of psychopathy assessment, such as the Psychopathic deviate scale of the MMPI, Multidimensional Personality Questionnaire, Psychopathic Personality Inventory, Self-Report Psychopathy Scale, Levenson's Self-Report Psychopathy Scale, and the Antisocial Process Screening Device (see Lilienfeld & Fowler, 2006, for a review). Modes of assessment were coded as self-report or interview, and when categorical analyses were used in a study, cut-off scores used to divide individuals into psychopathic and nonpsychopathic groups were recorded. Most typically, a cut-off score of 30 or above is used to operationalize psychopathy, although there is little empirical justification for these cut-off scores and researchers sometimes use variable cut-off criteria across studies. Of those studies conducting categorical analyses, the nature of the comparison groups differed across studies. Thus, the type of comparison group used (e.g., nonpsychopathic inmates, healthy community members) was recorded.

Finally, information regarding the nature of the experimental procedure and data analyses within a study was extracted. Because a variety of tasks have been used in the RM literature, the type of task used was coded in addition to the outcome measure used by the researchers. Common tasks used to assess RM included, but were not limited to, go/no-go tasks, picture word tasks, spatially separated Stroop tasks, card perseveration tasks, and lexical decision-making tasks. Again, these tasks were coded as moderators of the relation between psychopathy and RM deficits.

To account for the theoretical evolution of the model over time, experimental tasks were categorized into one of four versions of the RMH: perseveration (e.g., go/no-go, gambling tasks), reflection versus disinhibition (e.g., RT on perseveration tasks), CABA (e.g., picture word task, spatially separated Stroop), and attentional bottleneck (e.g., instructed fear tasks). For studies using the widely used go/no-go task, the presence or absence of a reward pretreatment to establish a dominant response set was coded and examined as a moderator. As noted earlier, the RMH predicts that the use of a reward pretreatment should engender a more potent dominant response set, hence yielding larger deficits (e.g., Newman et al., 1990).

Additionally, a variety of dependent measures were used to assess RM, such as passive avoidance errors, reflection or response time after punishment, response time interference, and psychophysiological indicators. As discussed previously, a number of studies in the RMH literature have subdivided participants into groups of low and high anxiety psychopaths and controls (e.g., Arnett et al., 1993; Newman et al., 1990). Thus, for each effect size, we coded whether that effect size corresponded to participants of high anxiety, low anxiety, or combined anxiety levels. Finally, a number of studies report analyses conducted using covariates, such as IQ or baseline on laboratory task; as such, the type of covariate used, if any, was coded for each study. Of the studies included in the meta-analysis, only four effect sizes were based on the use of covariates.

Coding of Allegiance Effects

Although the aim of the present review was to provide a systematic review of the RMH literature, the validity of meta-analytic results inevitably hinges on the validity and methodological quality of studies included in the review. To account for potential biases across studies, each study was coded for the degree of researcher allegiance to the RMH. This method was adapted from an allegiance coding scheme devised by Gaffan, Tsaousis, and Kemper-Wheeler (1995) to meta-analytically examine the potential influence of researcher allegiance on treatment effect sizes in psychotherapy outcome studies. Two independent raters coded the studies for RMH allegiance using a 0, 1, 2, or 3 scale. The studies were rated by means of an examination of the introductory section of the manuscript. Studies were given a score of 3 if the introduction referenced the superiority of the RMH to other hypotheses or if RMH was the only hypothesis examined *and* if the lead proponent of RMH (e.g., Joseph Newman) was an author on the article. On the opposite extreme, a study was given a score of zero if a RM task was included in the study without specific mention of the validity of the RMH. The mean allegiance score from the two raters was computed and these mean allegiance scores were examined as potential moderators of effect size. Interrater reliability of allegiance scores was high ($ICC = .88$).

Summary Measures and Planned Method of Analysis

Given that we could not assume that the variability in effect sizes stemmed solely from sampling error (i.e., we also predicted that some of this variability would be due to the coded moderators, such as the psychopathy measure or RM task used), the meta-analysis was performed using a random effects model. For studies drawing on multiple measures of psychopathy, the mean of the effect sizes across the study was taken to account for the lack of independence among multiple effects sizes stemming from the same sample. Because psychopathy is becoming increasingly accepted as a dimensional rather than categorical construct (e.g., Guay, Ruscio, Knight, & Hare, 2007; Marcus, John, & Edens, 2004), correlational values were used to summarize the relations between psychopathy and RM deficits. Thus, the primary outcome measure was the correlation between psychopathy scores and RM. The data were coded such that positive correlations indicate increasing deficits in RM with increasing levels of psychopathic traits, that is, in the direction of support for the RMH. Nevertheless, to supplement the correlational analyses, we also reported Cohen's *d* for the overall effect sizes.

To calculate the mean effect size, we used the Comprehensive Meta-Analysis Program (version 2.0). The random effects model used in the analyses assumes that studies included in the review differ from each other systematically. Thus, this model accounts for variation in effect sizes from study to study because of random error within the individual studies (as in the fixed effects model), but also accounts for true variation that exists from study to study. To estimate the heterogeneity of effect sizes, we calculated the I^2 statistic, which is an indicator of the percentage of heterogeneity in effect sizes that is due to true variability versus random error. A *Q*-statistic was also calculated to determine the statistical significance of the observed heterogeneity across moderators.

Subgroup analyses were used to examine categorical moderators of the summary effect size. These analyses were conducted using a mixed effects model. In this model, studies within certain subgroups (e.g., low anxiety, high anxiety, and combined) are pooled using a random effects model. The difference in effect sizes across subgroups is tested for significance using a fixed effects model. For continuous moderators, meta-regression techniques were used to examine the relation between the variable of interest and the mean effect size using a random effects model. Finally, publication bias was tested using several metrics. First, we compared the effect sizes derived from published versus unpublished studies with the assumption that smaller effect sizes for the latter might reflect publication bias. Second, funnel plots were examined as a graphical means of detecting publication bias. A funnel plot is a scatterplot of effect size against a measure of study size (typically, *SE*). The assumption is that studies large in size will fall close to the mean. In contrast, studies smaller in size are expected to be equally dispersed on either side of the mean effect size. Evidence of potential publication bias occurs when smaller studies are asymmetrically dispersed around the mean effect size, particularly when the plot shows a concentration of small studies with large effect sizes and an absence of small studies with small effect sizes. Results such as these point to a potential tendency to publish small studies with large effect sizes that are likely to reach statistical significance and not to publish small studies with small effect sizes, perhaps because investigators attribute such null results to low power and place them in the proverbial "file drawer" (Egger, Smith, Schneider, & Minder, 1997; Light & Pillemer, 1984). Egger's test of intercept bias was further used to provide a statistical estimate of the degree and significance of asymmetry in the funnel plot (Egger et al., 1997) and Duval and Tweedie's (2000) trim and fill method was used to provide an adjusted estimate of effect size after publication bias is accounted for.

Results

In total, 94 independent samples were identified for inclusion in the review. The included studies involved a total of 7,340 participants. See Table 2 for an overview of the characteristics of the studies. Using a random effects model, the overall results indicated a small to medium relation between total psychopathy scores and RM deficits ($r = .16$; 95% confidence interval [CI] [.11, .20], $d = .32$, $p < .001$). Significant heterogeneity among the effect sizes was detected ($I^2 = 70.0\%$, $df = 116$, $p < .001$). A review of the relative weight of each study (ranging from .42 to 1.3) indicated that no one study dominated the estimated mean effect size. One study (i.e., Vitale, Brinkley, Hiatt, & Newman, 2007) was identified as an outlier (Residual >3.0), but sequential sensitivity analyses removing each study yielded only trivial changes to the summary effect size (*r*s = .15–.16).

Because of the significant heterogeneity among the effect sizes and a priori hypotheses regarding moderators of the relations between psychopathy and RM, several follow-up analyses were performed. Because researchers often separate psychopaths and controls based on levels of trait anxiety (e.g., Newman & Kosson, 1986), subgroup analyses were performed to examine potential differences in effect sizes across samples of low anxiety, high anxiety, and combined anxiety psychopaths and controls. A random effects analysis revealed significant heterogeneity in effect sizes across anxiety subgroups, $Q(2) = 14.5$, $p < .05$. Planned comparisons revealed no difference between the effect sizes of combined and low anxiety subgroups, $Q(1) = .13$ $p = .78$. Samples of high anxiety individuals were associated with significantly smaller effect sizes ($r = .00$, $k = 27$) than both combined ($r = .20$, $k = 52$) and low anxiety ($r = .18$, $k = 33$) subgroups, $Q(1) = 14.4$, $p < .001$, $Q(1) = 6.78$, $p < .05$, respectively). Thus, as predicted by the RMH, deficits in RM do not clearly emerge among psychopathic individuals with elevated levels of anxiety, or presumed secondary psychopaths. The absence of a difference between effect sizes for combined and low anxiety subgroups may be due to small sample sizes. Nevertheless, due to these findings, the remaining analyses were performed after excluding subgroups of high anxiety individuals.

A summary effect size excluding high anxiety individuals revealed a slightly higher correlation between total psychopathy scores and RM deficits in the small to medium range ($r = .20$; 95% CI [.15, .25], $d = .41$). In this reduced sample of studies, significant heterogeneity among the effect sizes was still detected ($I^2 = 70.2\%$, $df = 89$, $p < .001$). A review of the relative weight of each study (ranging from .52 to 1.7) again indicated that no one study dominated the estimated mean effect size. One study (i.e., Vitale, Brinkley, Hiatt, & Newman, 2007) was identified as an outlier (Residual >3.0), but sequential sensitivity analyses removing each study yielded only trivial changes to summary effect sizes (*r*s = .19–.20). Finally, one study included in the meta-analysis (i.e., Poythress et al., 2010) was characterized by a particularly large sample size ($n = 1{,}381$) and a low overall effect size ($r = -.01$). To provide an estimate of the strength of the RMH that is not unduly influenced by one investigation, all major analyses were conducted both including and excluding the Poythress et al. study.

Allegiance and Publication Bias

Meta-regression analyses revealed that the allegiance score did not contribute significantly to the correlation between psychopathy and RM deficits in published studies ($Q_{model} = 1.57$, $df = 1$, $p = .20$). Allegiance scores were also not significantly associated with RM deficits for the total sample of combined published and unpublished studies. These results remained nonsignificant after removing the Poythress et al. (2010) study.

A series of analyses were conducted to estimate the potential impact of publication bias on the RMH literature. Published ($k = 77$) studies exhibited significantly higher effect sizes ($r = .23$) than unpublished ($k = 13$) studies ($r = -.01$, $Q = 11.5$, $p < .01$), with the analyses remaining exactly the same after removing the Poythress et al. (2010) study. In addition, an examination of Figure 1 further points to the presence of potential publication bias in the published RMH literature. The funnel plot shows a clear concentration of studies toward the right side of the funnel. This suggests that as sample sizes become smaller (i.e., as *SE* increases) effects are more likely to be published if the effect size is larger than average and if statistical significance is more likely to be achieved. Egger's test of the regression intercept indicates that this bias is statistically significant ($\beta_0 = 1.77$, $df = 75$, $p < .001$). In addition to Egger's test, Duval and Tweedie's (2000) trim and fill method was used to impute the presumed "missing" findings and re-estimate the effect size of the relation between psychopathy and RM without ostensible publication bias. Using this method, the estimated correlation between psychopathy and RM was reduced to $r = .11$ based on an estimated 18 "missing" findings. These missing findings can be viewed graphically in Figure 1 as filled black circles. This figure illustrates the conspicuous number of findings missing in the left portion of the funnel plot, raising the possibility that the published literature may not provide an accurate representation of the true RM effect size. After removing the Poythress et al. (2010) study, Egger's test of the regression intercept became nonsignificant and the Duval and Tweedie (2000) corrected effect size increased ($r = .14$). Nevertheless, these results overall raise questions regarding the robustness of the RMH arising from possible publication bias.

Follow-up analyses were conducted examining the continued existence of publication bias after the inclusion of unpublished dissertations and master's theses. Even with the inclusion of unpublished studies, the results of the analysis point to the possibility of publication bias. Egger's test of regression intercept indicates that this potential bias is statistically significant ($\beta_0 = 1.31$, $df = 88$, $p < .01$). Duval and Tweedie's (2000) trim and fill method re-estimated the effect size to a reduced correlation of $r = .10$ based on an estimated 17 missing studies. Again, after removing the Poythress et al. (2010) study, Egger's test of the regression intercept became nonsignificant and the Duval and Tweedie (2000) corrected effect size increased ($r = .14$).

Confounding of Moderators

Before examining the impact of moderators on the RMH, a series of analyses was performed to examine the degree to which moderators in this review were confounded. The issue of confounded moderators is common in meta-analyses; however, be-

Table 2
Characteristics of Studies in Meta-Analysis

Study	*N*	Sample	Sample demographics (%)	Psychopathy measure	Experimental paradigm	Outcome measure	Model version	Average effect size (*r*)
Anderson (2011)[a]	40	Adult community	Female–52 Male–47 White–80 Asian–3 Hispanic–17	PPI	Instructed fear	ERP	AB	.14
Arnett, Howland, Smith, and Newman (1993)	63	Adult prison	Male–100 White–100	PCL variant	Go/no-go	PAE; Heart rate	Perseveration	−.25
Arnett, Smith, and Newman (1997)	63;71	Adult prison	Male–100 White–100	PCL variant	Go/no-go	PAE; Reflection time	Perseveration; Disinhibition vs. reflection	−.11
Baskin-Sommers, Curtin, and Newman (2011)	87	Adult prison	Male–100 White–100	PCL variant	Instructed fear	FPS	AB	.13
Baskin-Sommers, Curtin, and Newman (2013)	136	Adult prison	Male–100	PCL variant	Instructed fear	FPS; ERP	AB	.18
Baskin-Sommers and Newman (2014)	106	Adult prison	Male–100 White–66 Black–31 Asian–1 Native American–2	PCL variant	Gaze task	Task performance	AB	.57
Bauer (1999)[a]	80	Adolescent prison	Female–100	PCL variant	Go/no-go	PAE	Perseveration	.05
Berstein, Newman, Wallace, and Luh (2000)	42	Adult prison	Male–100 White–100	PCL variant	Lexical decision	Task Performance	CABA	.42
Blair, Colledge, and Mitchell (2001)	51	Primary school	Male–100 White–98 Black–2	PSD	Gambling task; passive avoidance	Task performance; PAE	Perseveration	.07
Blair et al. (2004)	40	Adult prison	Male–100 White–88 Black–12	PCL variant	Go/no-go	PAE	Perseveration	.41
Brazil et al. (2012)	59	Adult prison		PCL variant	Visual oddball	PAE	CABA	.18
Brinkley, Schmitt, and Newman (2005)[b]	58;124	Adult prison	Male–100 White–100	PCL variant	Semantic stroop	Response Facilitation	CABA	.19
Cale and Lilienfeld (2002)	75	Actors	Female–48 Male–52 White–91 Black–7 Asian–1	PPI	Go/no-go Picture word	PAE; Interference	Perseveration; CABA	.11
Carolan, Jaspers-Fayer, Asmaro, and Douglas (2014)	32	Undergraduate	Female–62 Male–38	PPI	Emotional Stroop	Interference	CABA	.40
Chesno and Killmann (1975)	27	Adult prison	Male–100	Cleckley criteria	Mental maze	PAE	Perseveration	.04
Christianson et al. (1996)	62	Adult prison	Male–100 White–84	PCL variant	Emotional memory	Task performance	CABA	.36
†Dadds et al. (2006)	33;65	Primary school	Male–100	CU traits/ APSD	Fear recognition task	Fear recognition	CABA	.42
Derefinko (2009)[a]	91	Undergraduate	Male–100	PPI; SRP; FFM prototype	BART; go/no-go	Task performance; PAE	Perseveration	−.01
Dvorak-Bertsch, Curtin, Rubenstein, and Newman (2009)	55	Undergraduate	Female–37 Male–63	MPQ prototype	Instructed fear	FPS	AB	.15

Table 2 (*continued*)

Study	*N*	Sample	Sample demographics (%)	Psychopathy measure	Experimental paradigm	Outcome measure	Model version	Average effect size (*r*)
Epstein, Poythress, and Brandon (2006)	169	Misdemeanants	Female–59 Male–41 White–42 Black–40 Asian–1 Hispanic–10	SRP	Go/no-go	PAE	Perseveration	.19
Glass and Newman (2009)	239	Adult prison	Male–100 White–100	PCL variant	Emotional memory	Word recall	CABA	.19
Goldstein (1998)[a]	148	Adult prison	Male–100	PCL variant	Go/no-go	PAE	Perseveration	−.05
Hamilton, Baskin-Sommers, and Newman (2014)	117	Adult prison	Male–100 White–100	PCL variant	Spatially separated Stroop	Interference	CABA	.24
Heritage and Benning (2013)	89	Adult community	Female–56 Male–44 White–70 Black–27	MPQ prototype	Lexical decision	Interference; task performance; ERP	CABA	.10
†Hiatt, Schmitt, and Newman (2004)	75;69	Adult prison	Male–100 White–100	PCL variant	Picture word; spatially separated Stroop	Interference	CABA	.29
Howard, Payamal, and Neo (1997)	50	Adult prison	Male–100 Asian–100	PCL variant	Go/no-go	PAE	Perseveration	−.05
Howland, Kosson, Patterson, and Newman (1993)	49	Adult prison	Male–100 White–100	PCL variant	Cued reaction time	Error rate	Perseveration	.18
Hunt, Hopko, Bare, Lejuez, and Robinson (2005)	80	Undergraduate	Male–38 Female–62 White–79 Black–13 Asian–5 Hispanic–5	SRP	BART	Task performance	Perseveration	.25
Jutai and Hare (1983)	39	Adult prison	Male–100 White–100	PCL variant	Dichotic listening/ selective attention	ERP	CABA	.51
Kiehl, Smith, Hare, and Liddle (2000)	36	Adult prison	Male–100	PCL variant	Go/No-Go	ERP; PAE	Perseveration	.31
Kosson (1996)	60	Adult prison	Male–100 White–100	PCL variant	Dichotic listening/ selective attention	Reaction time; task performance	CABA	−.03
Kosson, Miller, Byrnes, and Leveroni (2007)	172	Adult prison	Male–100 White–50 Black–50	PCL variant	Global/local processing	Reaction time	CABA	.14
Kosson and Newman (1986)	72	Adult prison	Male–100 White–100	PCL variant	Visual Search	Task Performance	CABA	.10
Kosson, Smith, and Newman (1990)	59	Adult prison	Male–100 Black–100	PCL variant	Go/no-go	PAE	Perseveration	.27
Larson et al. (2013)	71	Adult prison	Male–100 White–100	PCL variant	Instructed Fear	Amygdala Activation	AB	.11
Loney (2000)[a]	52	Primary school	Male–100 White–25 Black–75	PSD	Picture Word	Interference	CABA	−.24
Lorenz and Newman (2002)	100	Adult prison	Male–100 White–100	PCL variant	Lexical decision	Response Facilitation	CABA	.31
Lykken (1957)	69	Adult prison	Female–34 Male–66	Cleckley criteria	Mental maze	PAE	Perseveration	.43
Lynam, Whiteside, and Jones (1999)	70	Adult community	Male–100 White–100	LSRP	*Q* search; Go/no-go	Reaction time; PAE	Perseveration	.23
MacKenzie (2012)[a]	60	Undergraduate	Female–72 Male–28 White–100	SRP	Lexical decision	Interference	CABA	.33
Mayer, Kosson, and Bedrick (2006)	91	Adult prison	Male–100	PCL variant	Spatially separated Stroop	Interference	CABA	.18
Mitchell, Colledge, Leonard, and Blair (2002)	51	Adult prison	Male–100 White–96 Black–4	PCL variant	Gambling task; passive avoidance	Task performance	Perseveration	.20

(*table continues*)

Table 2 (*continued*)

Study	*N*	Sample	Sample demographics (%)	Psychopathy measure	Experimental paradigm	Outcome measure	Model version	Average effect size (*r*)
Mitchell, Richell, Leonard, and Blair (2006)	35	Adult prison	Male–100 White–80 Black–17 Asian–3	PCL variant	Emotional interrupt	Task performance; interference	CABA	.22
Moltó, Poy, Segarra, Pastor, and Montañés (2007)	47	Adult prison	Male–100	PCL variant	Passive avoidance	Cards played; PAE	Perseveration	.60
Moulton (1999)[a]	26	Adult inpatient	White–8 Black–80 Hispanic–2	PCL variant	Go/no-go	Task performance; cards played	Perseveration	.29
Munro (2009)[a]	15	Adult prison	Male–100	PCL variant	Go/no-go	PAE	Perseveration	−.46
Newman, Curtin, Bertsch, and Baskin-Sommers (2010)	125	Adult prison	Male–100 White–100	PCL variant	Instructed fear	FPS; ERP	AB	.26
Newman and Kosson (1986)	60	Adult prison	Male–100 White–100	PCL variant	Go/no-go	PAE	Perseveration	.31
Newman, Kosson, and Patterson (1992)	158	Adult prison	Male–100 White–100	PCL variant	Delayed gratification	Task performance	Perseveration	.20
Newman, Patterson, Howland, and Nichols (1990)[b]	59;122	Adult prison	Male–100 White–100	PCL variant	Go/no-go	PAE; Reflection time	Reflection vs. disinhibition	.11
Newman, Patterson, and Kosson (1987)	72	Adult prison	Male–100 White–100	PCL variant	Passive avoidance	Task performance	Perseveration	.49
Newman and Schmitt (1998)	97;110	Adult prison	Male–100 White–100; Black–100 (second sample)	PCL variant	Go/no-go	PAE	Perseveration	.25
Newman, Schmitt, and Voss (1997)	68;56	Adult prison	Male–100; White–100; Black–100 (second sample)	PCL variant	Picture word	Interference	CABA	.44
Newman, Wallace, Schmitt, and Arnett (1997)	48	Adult prison	Male–100 White–100	PCL variant	*Q* search	Reaction time	Perseveration	.12
Newman, Widom, and Nathan (1985)	90;40	Primary school	Male–100 White–100	PCL variant	Go/no-go	PAE	Perseveration	.35
Pham, Vanderstukken, Philippot, and Vanderlinden (2003)	36	Adult prison	Male–100	PCL variant	Passive avoidance	PAE	Perseveration	.20
Poythress et al. (2010)	1380	Adult prison/ psychiatric	Male–82 Female–18 White–65 Black–35 Hispanic–7	LSRP; PPI; PCL variant	Go/no-go	PAE	Perseveration	−.01
Roose et al. (2013)	79	Adolescents with behavior problems		YPI	PSRTT	Reaction time	CABA	.20
Roussy and Toupin (2000)	54	Adolescent prison	Men–100	PCL variant	Go/no-go	PAE	Perseveration	.31
Sadeh & Verona (2008)	107	Adult community	Male–100 White–69 Black–2 Asian–16 Hispanic–6	PPI	Perceptual load	Reaction time	AB	.08
Sadeh and Verona (2012)	63	Mixed prison/ community	Female–18 Male–82 White–40 Black–49 Hispanic–3	PCL variant	Instructed fear	FPS; ERP	AB	.08

Table 2 (*continued*)

Study	*N*	Sample	Sample demographics (%)	Psychopathy measure	Experimental paradigm	Outcome measure	Model version	Average effect size (*r*)
Scerbo et al. (1990)	40	Adolescent prison	Male–100 White–23 Black–50 Hispanic–24	SRP	Go/no-go	PAE	Perseveration	.06
Schachter and Latane (1964)	30	Adult prison	Male–100	Cleckley criteria	Mental maze	PAE	Perseveration	.24
Schmauk (1970)	90	Adult prison	Male–100	Pd	Mental maze	PAE	Perseveration	.30
Schmitt (2000)[a,b]	26;37	Adult prison	Male–100 Black–100	PCL variant	Picture word; spatially separated Stroop	Interference	CABA	−.32
Siegel (1978)	74	Adult prison	Male–100	Cleckley Criteria	Gambling task	PAE	Perseveration	.35
Singh (2003)[a]	52	Undergraduate	Female–48 Male–52 White–81 Black–8 Hispanic–10	LSRP	Passive avoidance	Cards played	Perseveration	.25
Suchy and Kosson (2005)	58	Adult prison	Male–100 White–62 Black–38	PCL variant	Dichotic listening/ selective attention	PAE	CABA	.19
Swogger (2006)[a]	119	Adult prison	Male–100 White–47 Black–53	PCL variant	BART; Go/no-go	Task performance; PAE	Perseveration	.14
Thornquist and Zuckerman (1995)	79	Adult prison	Male–100 White–100	PCL variant	Go/no-go	PAE	Perseveration	.37
Varlamov et al. (2011)	68	Adult inpatient	Male–100	PCL variant	Go/no-go	PAE; ERP	Perseveration	.26
Venables and Patrick (2014)	152	Adult prison	Male–100	PCL variant	Visual oddball	ERP	CABA	.14
Vitale, Brinkley, Hiatt, and Newman (2007)	285	Adult prison	Female–100 White–100	PCL variant	Picture word	Interference	CABA	.57
Vitale and Newman (2001)	112	Adult prison	Female–100 White–100	PCL variant	Passive avoidance	Cards played; Task performance	Perseveration	−.05
Vitale et al. (2005)	304	Adolescent community	Male–53 Female–47 White–100	APSD	Picture word Go/no-go	Interference; PAE	CABA; perseveration	.17
Vitale, MacCoon, and Newman (2011)	117	Adult prison	Female–100 White–100	PCL variant	Lexical decision; Go/no-go	Response facilitation; PAE	CABA; perseveration	.02
Wolf et al. (2012)	53	Adult prison	Male–100 White–100	PCL variant	Attentional blink	Task performance	AB	.32
Zeier, Maxwell, and Newman (2009)	110	Adult prison	Male–100 White–100	PCL variant	Flanker task	Interference	CABA	.30
Zeier and Newman (2013a)	127	Adult prison	Male–100 White–100	PCL variant; PPI	Picture word	Interference	AB	.30
Zeier and Newman (2013b)	120	Adult prison	Male–100 White–78 Black–20 Hispanic–2	PCL variant	Flanker task	Interference	CABA	.42

Note. PPI = Psychopathic Personality Inventory; ERP = event-related potential; AB = attention bottleneck; PCL = Psychopathy Checklist; PAE = passive avoidance errors; FPS = fear potentiated startle; CABA = context-appropriate balance of attention; PSD = Psychopathy Screening Device; CU = callous/unemotional; APSD = Antisocial Process Screening Device; SRP = Self-Report Psychopathy Scale; FFM = five-factor model; MPQ = Multidimensional Personality Questionnaire; FPS = Fear Potentiated Startle; BART = Balloon Analog Risk Task; LSRP = Levenson Self-Report Psychopathy Scale; YPI = Youth Psychopathy Inventory.

[a] Unpublished. [b] A number of publications present multi-part studies with independent samples; for these publications multiple sample sizes are listed.

cause of a small number of studies across moderators, a lack of adequate statistical power (i.e., a sufficient number of studies comparing confounded with unconfounded moderators) prevented us from parsing the unique influences of each moderator (see Lipsey, 2003). To give readers a sense of the degree of the confounding among moderators, Pearson's ϕ coefficients and point-biserial correlations were calculated for key study characteristics. The moderator analyses to follow in later sections should be interpreted with these findings in mind, as they suggest that certain moderators in our analysis may actually be proxies for other moderators.

Point-biserial correlational analyses were conducted to examine the associations among the continuous variables (i.e., sample gender and racial composition) and binary variables (i.e., PCL based measure vs. non-PCL based measure, interview vs. self-report

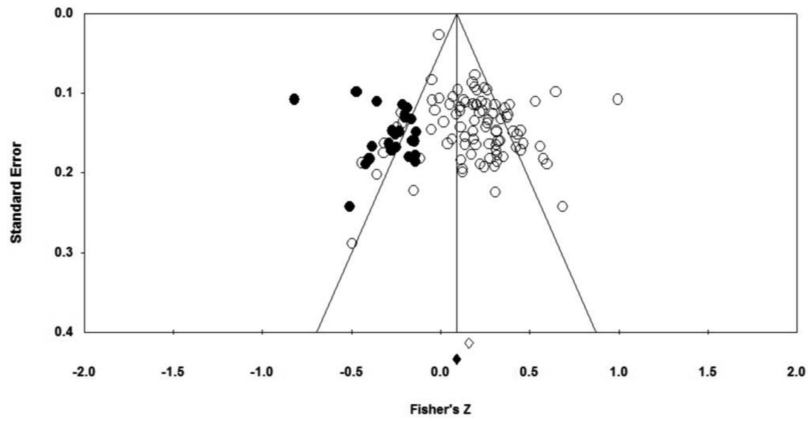

Figure 1. Publication bias in the response modulation literature. The figure above plots the effect size (Fisher's *Z*) of each finding by the standard error of that finding. The white circles represent each individual finding included in the review. In contrast, the black circles represent findings presumed "missing" from the literature using the Duval and Tweedie's (2000) trim and fill method. Similarly, the white diamond represents that overall estimated effect size from the meta-analysis whereas the black diamond represents the adjusted estimated effect size based on the imputed studies.

measure, and prison vs. nonprison setting). The gender composition of the sample was significantly associated with the sample setting and measure type (i.e., self-report vs. interview). In the present review, as the percentage of females in the sample increased, so did the use of self-report measures and samples from community settings ($r = .20$, $r = .19$, $p < .05$, respectively). In addition, as the percentage of females in the sample increased, the percentage of Hispanic individuals increased, $r = .30$, $p < .05$. Finally, as the percentage of Hispanic individuals in the samples increased, so did the use of non PCL-based measures, self-report measures, and samples from community settings ($r = .20$; $r = .42$; $r = .29$, $p < .01$, respectively). The percentage of Whites, Blacks, and Asians was not significantly associated with the aforementioned moderators.

Pearson's ϕ coefficients were also calculated to examine the degree of association among categorical moderators (e.g., PCL vs. non-PCL based measures, interview vs. self-report, and prison vs. nonprison setting). PCL-based measures were significantly more likely to be used in prison settings (Pearson's $\phi = .53$). Not surprisingly, studies using samples from prison settings were also significantly more likely to use interview measures, especially the PCL and its progeny (Pearson's $\phi = .83$).

Evaluating the Boundary Conditions of the RMH

A series of analyses were performed to determine the robustness of the RMH across sample type, psychopathy measure, outcome measure, and control group, among other moderators.

Study characteristics. The effect size was associated with significant variation based on the psychopathy measure used, suggesting that the evidence for the RMH differs as a function of the method of psychopathy assessment, $Q(12) = 25.1$, $p < .01$. The correlations across psychopathy measures ranged from $r = -.06$–$.42$ with the five-factor model (FFM) psychopathy prototype showing the smallest effect size and callous/unemotional (CU) traits derived from the APSD showing the largest effect size (see Table 3). The two most frequently used measures in this review (i.e., PCL variants and PPI) yielded comparable effect sizes in the small to medium range ($r = .19$, $r = .15$, respectively). After removing the Poythress et al. (2010) study, psychopathy measure remained a significant moderator, but the effect sizes for the PPI, LSRP, and PCL variants increased slightly ($r = .17$, $r = .24$, $r = .20$, respectively).

The effect size did not differ based on the nature of the psychopathy assessment (e.g., interview, self-report, observer report), $Q(2) = 1.63$, $p = .44$. Interview measures (consisting of the PCL variants and Cleckley criteria) showed the largest effect sizes ($r = .20$, $k = 93$), whereas observer and self-report measures showed similarly sized correlations ($r = .15$, $k = 5$; $r = .14$, $k = 39$,

Table 3
Correlation Between Various Psychopathy Measures and Response Modulation Deficits

Psychopathy measure	Correlation	Number of findings included
APSD	.17**	4
Cleckley criteria	.20	6
CU traits	.42***	2
FFM prototype	−.06	3
LSRP	.17**	6
PCL and variants	.19***	86
Pd	.33**	2
PPI	.15**	15
PSD	−.01	3
RDC	.30°	1
SRP	.15**	8
YPI	.20°	1

Note. APSD = Antisocial Process Screening Device; CU = callous/unemotional; FFM = five-factor model; Levenson Self-Report Psychopathy Scale; PCL = Psychopathy Checklist; Pd = Psychopathic deviate; PPI = Psychopathic Personality Inventory; PSD = Psychopathy Screening Device; RDC = research diagnostic criteria; SRP = Self-Report Psychopathy Scale; YPI = Youth Psychopathy Inventory.
° $p < .1$. * $p < .05$. ** $p < .01$. *** $p < .001$.

respectively). After removing the Poythress et al. (2010) study, the effect sizes for self-report and observer-report measures increased only trivially ($r = .16$, $r = .15$, respectively).

The studies in the meta-analysis drew on samples from a variety of settings (e.g., prison, inpatient, undergraduate/student, community). The effect size varied based on the setting, $Q(8) = 37.8$, $p < .001$. Effect sizes across the sample settings ranged from −.01 to .32 with a primary school sample showing small to medium effect sizes and a combined prison/court mandated treatment sample showing a negligible effect size (see Table 4). Follow-up analyses were conducted removing the Poythress et al. (2010) study (e.g., prison/court mandated treatment sample). After removing this sample, the effect sizes did not vary significantly by sample setting. Additionally, the effect size varied based on age (e.g., adult, adolescent, and youth) of the sample, $Q(3) = 9.15$, $p < .05$, with child samples showing the largest effect sizes ($r = .42$, $k = 2$) and a combined youth/adolescent sample the smallest ($r = .07$, $k = 1$). Results remained exactly the same for this analysis after removing the Poythress et al. (2010) study.

Fifty-six of the studies were based on categorical determinations of psychopathy. The effect sizes derived from categorical analyses did not differ significantly from the effect sizes derived from studies analyzing data dimensionally, $Q(1) = .37$, $p = .54$, even after removing the Poythress et al. (2010) study. Studies using categorical determinations of psychopathy also used a variety of comparison groups, such as nonpsychopathic inmates and healthy community controls. The relation between psychopathy and RM deficits did not differ significantly as a function of the comparison group, $Q(2) = 1.2$, $p = .54$. Similarly, for studies analyzing data categorically using the PCL-R, meta-regression analyses revealed no significant difference in effect size based on PCL-R cutoff scores.

RMH laboratory tasks. Results revealed significant heterogeneity in effect sizes for different RM tasks, $Q(24) = 55.8$, $p < .001$. The correlations between psychopathy and RM deficits ranged from .02 (p = ns) for semantic Stroop tasks and .57 ($p < .001$) for a gaze direction recognition task. Unexpectedly, for one of the most widely examined RM tasks, the go/no-go task, the effect size was small, $r = .09$, $p < .01$. These effects changed only trivially after removing the large Poythress et al. (2010) study ($r = .10$ for go/no-go). See Table 5 for a full list of RM tasks used by studies included in this review and their respective effect sizes.

Table 4
Correlation Between Psychopathy and Response Modulation Deficits Based on Sample Setting

Sample setting	Correlation	Number of findings included
Adolescents with behavior problems	.20°	1
Actors	.11	1
Community	.13***	6
Inpatient	.27°	2
Misdemeanants	.19*	1
Prison	.19***	69
Combined prison/psychiatric	−.01	1
Primary school	.32**	4
Undergraduate	.23*	5

° $p < .1$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 5
Correlation Between Psychopathy and Response Modulation Deficits Based on Experimental Task Used

Task	Correlation	Number of findings included
Attentional blink	.32*	1
BART	.10**	9
Cued reaction time	.18°	2
Delay of gratification	.20°	1
Dichotic listening/selective attention	.16	4
Emotional interrupt	.22*	2
Emotional memory	.26**	2
Emotional Stroop	.41***	4
Fear recognition	.42***	2
Flanker task	.36***	2
Gambling task	.18*	3
Gaze task	.57***	1
Global-local processing	.14	2
Go/no-go	.09**	43
Instructed fear task	.18***	11
Lexical decision making	.27**	5
Mental maze	.19	6
Oddball task	.19°	2
Other passive avoidance tasks	.26***	11
PSRTT	.20°	1
Picture word task	.25*	11
Q search	.19°	2
Spatially separated Stroop	.20**	11
Semantic Stroop	.02	3
Visual search	.09	1

Note. BART = Balloon Analog Risk Task; PSRTT = Point Scoring Reaction Time Test.
° $p < .1$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Overall, these results suggest that the relation between RM and psychopathy varies across tasks and differs as a function of the laboratory paradigm used.

Subanalyses were performed to examine the commonly used go/no-go task. Many proponents of the RMH administer the go/no-go task with a practice round that is saturated with reward stimuli. As noted earlier, this "reward pretreatment" is intended to establish a dominant response set for reward, and should therefore be associated with larger effect sizes. Nevertheless, moderator analyses revealed effect sizes for the go/no-go task that did not differ significantly regardless of the pretreatment condition (Reward pretreatment: $r = .09$; No pretreatment: $r = .15$), although they were in the opposite direction from that predicted by the RMH. These effects remained largely the same, with only trivial changes in effect sizes (Reward pretreatment: $r = .10$; No pretreatment: $r = .14$), after removing the Poythress et al. (2010) study.

Because passive avoidance (e.g., go/no-go, card perseveration) tasks are among the more commonly used tasks used to assess RM deficits, additional analyses were conducted to examine moderators among these tasks. Among passive avoidance tasks, effect sizes differed significantly based on psychopathy measure used, $Q(9) = 41.5$, $p < .001$. Effect sizes ranged from −.19 and −.09 for the FFM prototype and PPI-R, respectively, to .39 for the Pd scale of the MMPI. The effect size for the PCL-R was similar to that of the main analyses ($r = .17$). Sample setting was also a significant moderator, $Q(7) = 18.0$, $p < .001$, with inpatient

samples showing the largest effect sizes ($r = .27$) and a combined prison/psychiatric sample showing the smallest effect size ($r = -.01$). Other variables such as sample age, analytic strategy, gender, and race were not significant moderators.

RMH outcome measure. Outcome measures included amygdala activation, a variety of event-related potential responses (ERP), heart rate, passive avoidance errors, and measures of performance specific to each task. The relation between psychopathy and effect size varied substantially based on the outcome measure of choice, $Q(13) = 27.4$, $p < .05$. One outcome measure, namely heart rate, yielded findings that ran in the opposite direction from that predicted by the RMH, $r = -.22$, $p = .07$. Nevertheless, this estimate is based on one study in which this outcome index was used. The strongest effect size emerged for numbers of cards played in card perseveration tasks and fear recognition in an emotion recognition task ($r = .35$, $p < .001$; $r = .42$, $p < .001$). Consistent with the results for the go/no-go tasks, the overall effect size for studies using passive avoidance errors as an index of RM was small, $r = .13$, $p < .001$. The effect size for passive avoidance errors was altered minimally ($r = .15$) after removing the Poythress et al. (2010) study. See Table 6 for a full depiction of effect sizes based on outcome measures.

Approximately 13% of the effect sizes in this review used physiological outcome measures, such as heart rate, FPS, and ERP data. Post hoc moderator analyses revealed no differences in effect sizes between behavioral and physiological outcome measures. Furthermore, sensitivity analyses removing physiological measures from the analyses resulted in exactly the same overall effect size.

RMH task type and model version. Because of the wide variety of tasks used in the RM literature, follow-up analyses were conducted to examine overarching trends in the effect sizes across these tasks. Post hoc coding was used to categorize the tasks as emotionally laden (e.g., fear-potentiated startle paradigms, emotional memory facilitation, and go/no-go task) or emotionally neutral (e.g., picture word task, spatially separated Stroop). Results of the analyses did not reveal significant heterogeneity in effect sizes for the different categories of tasks, $Q(1) = .97$, $p = .33$. As predicted by the RMH, tasks with emotional content ($r = .17$, $k = 99$) yielded similar effect sizes to those of neutral tasks ($r = .20$, $k = 38$). After removing the Poythress et al. (2010) study, the moderator remained nonsignificant and the effect sizes for emotional and neutral tasks changed only trivially ($r = .18$, $r = .20$, respectively).

Table 6
Correlation Between Psychopathy and Response Modulation Deficits Based on Outcome Measure Used

Outcome measure	Correlation	Number of findings included
Amygdala activation	.11	1
Cards played	.35**	7
ERP data	.20*	11
Response facilitation	.11	6
Fear recognition	.42***	2
Fear potentiated startle response	.24**	3
Heart rate	−.22	1
Reaction time interference	.28*	20
Passive avoidance errors	.13***	50
Reaction time	.19***	7
Reflection time after punishment	.14	3
Task performance	.15***	22
Word recall	.19*	1

Note. ERP = event-related potential.
* $p < .05$. ** $p < .01$. *** $p < .01$.

As mentioned previously, the RMH has evolved considerably from its early conceptualization of response perseveration, to a model accounting for motivationally neutral attentional abnormalities, to the contemporary conceptualization of RM deficits as the result of an early attentional bottleneck. As also noted previously, each effect size was categorized according to its incarnation of the RMH model. Moderator analyses did not reveal significant variation in effect sizes across the iterations of the RMH model being tested, $Q(3) = 6.10$, $p = .11$. Thus, effect sizes have remained largely consistent across the evolution of the model. As a more indirect way of examining whether effect sizes have increased in magnitude in conjunction with the evolution of the RMH, publication year was examined as a moderator; however, no significant changes in effect size were found based on year of publication.

Demographic characteristics. The majority of studies on RM and psychopathy have utilized male-only samples. Nevertheless, a number of studies have examined the relation between RM deficits and psychopathy in female-only and mixed gender samples. Prior to this review, results for the RMH literature offered mixed support for the generalization of the model to females (e.g., Vitale, MacCoon, & Newman, 2011). Nevertheless, our results suggest that the effects of psychopathy on RM do generalize to females. When examined as a moderator in a random effects meta-regression model, the percentage of males in the sample did not alter the overall effect size ($Q_{model} = 1.2$, $p = .27$). These analyses may have been underpowered because of the relatively small portion of studies that included female participants (22%). When the Poythress et al. (2010) study was removed, these results remained nonsignificant.

Most studies on RM and psychopathy draw on White samples. Meta-regression analyses were used to examine the change in effect size as the racial composition of the study sample changed. Results indicated that as the percentage of White participants increased, the effect size also increased ($Q_{model} = 6.96$, $p < .01$). Similarly, as the percentage of Blacks in the samples increased, the effect size significantly decreased ($Q_{model} = 5.15$, $p < .05$). As the percentage of Asian and Hispanic individuals in the samples increased, the effect size did not change significantly ($Q_{model} = 2.03$, $p = .15$; $Q_{model} = .86$, $p = .35$, respectively). After removing the Poythress et al. (2010) study, the percentage of Whites remained a significant moderator whereas the percentage of Blacks, Asians, and Hispanics were not significant moderators.

Categorical moderator analyses were conducted to estimate overall effect sizes for exclusively White, Black, and Asian samples. Because none of the studies drew on purely Hispanic samples, a summary estimate of the RM effect size for Hispanic individuals could not be estimated. These analyses revealed marginally significant heterogeneity in effect sizes across race, $Q(3) = 6.24$, $p = .05$. Pure Black and Asian samples showed small effect sizes that were in the opposite direction of the RMH ($r = -.04$, $k = 6$; $r = -.05$, $k = 1$, respectively). White samples showed the highest effect sizes in the medium range ($r = .21$, $k = 39$). These

effects sizes remained exactly the same after removing the Poythress et al. (2010) study.

Psychopathy dimensions. Because of the differential pattern of correlates often exhibited by the subdimensions of psychopathy (Hare, 1991/2003), the examination of psychopathy solely at the global level may be problematic. Only a small number of studies, however, have examined the relation between psychopathy and RM at the factor level. The examination of these dimensions is important to the extent that it may shed light on the features of psychopathy that may be responsible for RM deficits; it also provides a stringent test of the RMH, which posits that RM deficits extend to all features of psychopathy. Our analyses did not reveal significant differences in effect sizes as a function of psychopathy subdimensions, $Q(2) = 2.18$, $p = .14$. Although the difference was not significant, the estimated effect size for Factor I, namely, the core affective and interpersonal traits of psychopathy, $r = .11$, $k = 35$, $p < .001$ was slightly larger than that for Factor II, namely, the antisocial and impulsive lifestyle features of psychopathy, $r = .06$, $k = 37$, $p < .01$. These results suggest that both dimensions of psychopathy are relevant to RM deficits, although the effect sizes were small at best. After removing the Poythress et al. (2010) study, the moderation remained nonsignificant, although the effect sizes for Factor I and Factor II increased slightly ($r = .13$, $r = .07$, respectively).

Discriminant validity. The credibility of a theory of specific etiology (Meehl, 1977) hinges on demonstrating that the ostensible causal variables are specific to the construct of interest (e.g., psychopathy) rather than to other constructs (Cook, 1990; Shadish, 1995). For example, several other conditions marked by disinhibition and poor impulse control, such as borderline personality disorder (BPD), antisocial personality disorder (ASPD), and attention deficit hyperactivity disorder (ADHD), have been found to be associated with similar patterns of RM deficits, especially on go/no-go tasks (Dolan & Park, 2002; Farmer & Rucklidge, 2006; Hochhausen, Lorenz, & Newman, 2002; Yong-Liang et al., 2000). Similarly, studies examining the relation between personality traits, such as extraversion, point to the possibility of comparable deficits in RM (Newman, 1987). Hence, following our meta-analytic review of the RMH as applied to psychopathy, we now examine the important question of discriminant validity, in particular the role of RM deficits in other conditions presumably marked by disinhibition (e.g., Gorenstein & Newman, 1980). Because of the small number of studies for these other psychological conditions, our review is narrative rather than meta-analytic in nature.

Attention-deficit/hyperactivity disorder (ADHD). One research team attempted to apply the RMH to the etiology of ADHD. With a sample of 41 adolescent youth, Farmer and Rucklidge (2006) examined the association among passive avoidance errors and ADHD on a mixed incentive go/no-go task identical to those used by RMH researchers (i.e., Patterson, Kosson, & Newman, 1987). Results indicated that youth with ADHD exhibited more passive avoidance errors than did normals, even after controlling for IQ and comorbid Oppositional Defiant/Conduct Disorder symptoms. Furthermore, consistent with Patterson and Newman's (1993) disinhibition versus reflection version of the RMH, reflection time after punishment was negatively associated with passive avoidance errors. Although the researchers did not directly examine the association between ADHD symptoms and reflection time, this finding potentially suggests that individuals with ADHD and psychopathic individuals may show similar pathways to RM deficits. This proposition is strengthened by the fact that ADHD individuals showed no fewer omission errors than controls, suggesting that their high levels of passive avoidance errors are not merely the result of disinhibited responding in general.

Antisocial personality disorder. A handful of studies has attempted to distinguish psychopathy from antisocial personality disorder (ASPD). Given that individuals with ASPD tend to show executive control deficits (Morgan & Lilienfeld, 2000; Ogilvie, Stewart, Chan, & Shum, 2011), whereas psychopathic individuals may not (e.g., Brinkley, Schmitt, & Newman, 2005), Zeier, Baskin-Sommers, Hiatt Racer, and Newman (2012) sought to separate the cognitive deficits associated with ASPD from those associated with psychopathy (e.g., early attentional bottleneck). Using a modified flanker task to assess cognitive control, the authors predicted that ASPD would be positively associated with interference on the task whereas psychopathy would show no association. As predicted, increasing levels of ASPD were associated with greater interference on the task. However, contrary to predictions, psychopathy was also positively associated with interference on the task. Furthermore, this interference was accounted for by shared variance in psychopathy scores and ASPD symptoms. The authors hypothesized that these unexpected findings may have resulted from a lack of temporal or spatial separation of the distractors from the target stimuli, thereby precluding psychopathic individuals from establishing an early attentional filter facilitating the ability to screen out irrelevant stimuli. Nevertheless, these findings raise questions regarding the specificity of RMH deficits to psychopathy as opposed to ASPD.

Despite the anomalies in the aforementioned study, others have been more successful in establishing the specificity of RM deficits to psychopathy. Drawing on a lexical decision-making task previously used to assess RM deficits, Kosson, Lorenz, and Newman (2006) sought to examine the potential differences in etiology underlying ASPD and psychopathy. This task asks participants to indicate whether a string of letters is an English word. Previous research indicates that most participants show response time facilitation when the string contains an affectively tinged word, such as LOVE, compared with an affectively neutral word, such as LEAF (Williamson, Harpur, & Hare, 1991). According to the RMH, psychopathic individuals become hyperfocused on the dominant task (i.e., identifying the letter string) and ignore extraneous information, such as the affective valence of the word. Thus, such individuals should display less affective facilitation than do controls. Furthermore, if such deficits are distinctive to psychopathy, individuals with ASPD should not show such deficits. Consistent with the authors' hypotheses, the psychopathic group displayed less affective facilitation than did controls and the ASPD group. Additionally, the ASPD group did not differ from controls in affective facilitation. These results lend support to the idea that RM deficits are distinctive to psychopathy. However, the task used in this study relied on emotional versus nonemotional words as the secondary or extraneous stimuli. Thus, the results of this study cannot rule out the possibility that deficits in emotional processing are largely responsible for the difference between psychopathic and antisocial inmates (but see Lorenz & Newman, 2002, for an alternative view).

Borderline personality disorder (BPD). Some researchers have argued that BPD overlaps with psychopathy (e.g., Miller et

al., 2010). This overlap may be particularly related to impulsivity, which is a core feature of BPD. Indeed, psychopathic and borderline individuals show similar performance on go/no-go tasks; specifically, both groups exhibit more passive avoidance errors compared with healthy controls (Hochhausen, Lorenz, & Newman, 2002). RMH proponents (e.g., Baskin-Sommers, Vitale, MacCoon, & Newman, 2012) have proposed that BPD is also characterized by attentional abnormalities, with a superficial similarity to the RM deficits associated with psychopathy. In the case of passive avoidance errors, RMH advocates have highlighted the diverse pathways to RM deficits. For example, in addition to increased passive avoidance errors, individuals with BPD commit fewer omission errors on the go/no-go task, a pattern not typically observed with psychopathic individuals.

Proponents of the RMH have also examined BPD using instructed fear paradigms adapted from the attentional bottleneck literature. RMH advocates propose that, like psychopathic individuals, individuals with BPD show deficits in regulating attention (Baskin-Sommers et al., 2012). Specifically, BPD individuals readily develop dominant response sets to personally relevant information or when given instructions to attend toward or away from selected stimuli. Thus, exaggerated emotional responses seen in BPD individuals may be due to the activation of a dominant, emotion-focused response set. Using an instructed fear paradigm, Baskin-Sommers et al. (2012) corroborated this hypothesis by showing that, compared with other offenders, offenders with elevated BPD features exhibited greater fear potentiated startle (FPS) in threat focus conditions and normal FPS in conditions in which they were instructed to focus on threat-irrelevant information. These findings differ from those in psychopathic individuals, who show diminished FPS in alternative focus conditions and normal FPS when instructed to focus on threat relevant information (e.g., Baskin-Sommers, Curtin, & Newman, 2013). For individuals with BPD, once a dominant response set toward affective information is established, it becomes difficult for them to shift their attention away from such information, resulting in heightened emotional responses.

Externalizing psychopathology. Much of the research attempting to establish the specificity of RM deficits to psychopathy centers on the separation of psychopathy from externalizing traits more broadly (e.g., Baskin-Sommers & Newman, 2014; Baskin-Sommers et al., 2012; Zeier & Newman, 2013b). Baskin-Sommers et al. (2012) proposed an attentional model of externalizing whereby individuals become overfocused on motivationally significant cues (e.g., cues of threat or reward) and subsequently show difficulty reallocating attention and regulating responses. According to them, like individuals with BPD, externalizers overreact to emotional information. Baskin-Sommers et al. (2012) found support for this hypothesis by showing that externalizers exhibit increased FPS in a threat focus condition. Thus, like psychopathic individuals, externalizers appear to show an overallocation of attention to a dominant response set. In contrast to psychopaths, externalizers display this response specifically to motivationally relevant stimuli.

Other studies have attempted to disentangle the relationship among RM deficits, psychopathy, and externalizing traits. Zeier and Newman (2013b) found that RM deficits on a flanker task are exhibited only in a subset of psychopathic individuals with low levels of externalizing behavior. The authors suggested that these findings may be indicative of the specificity of RM deficits to primary rather than secondary psychopathy. Externalizers also show distinctive responses on attentional blink tasks, which have been used to study RM deficits in psychopathy. Whereas psychopathic individuals exhibit diminished attentional blink responses, externalizers show heightened responses because of an overallocation of attention to goal-relevant information (Baskin-Sommers et al., 2012).

Summary of discriminant validity findings. Proponents of the RMH have made significant efforts to establish the specificity of RM deficits to psychopathy and disentangle such deficits from related disorders, particularly those relevant to disinhibition or impulsivity. In particular, RMH advocates have acknowledged the similarities across such disorders on go/no-go tasks and proposed that several distinct etiological pathways result in RM deficits (e.g., Newman & Wallace, 1993). In summary, a number of disorders such as psychopathy, BPD, ASPD, and externalizing behavior more broadly, are marked by RM deficits, namely, an overfocusing on attention and subsequent inability to incorporate extraneous information. Nevertheless, the precise nature of these deficits appears to distinguish psychopathy from broader disorders of disinhibition, with the latter being more selective to affectively charged stimuli.

Discussion

The RMH has emerged as one of the leading explanations of psychopathy. This model posits that psychopathy is associated with deficits in the relatively automatic shift of attention from goal-directed behavior to extraneous stimuli that point to the need for an alteration in one's behavior (Patterson & Newman, 1993). The theory has acquired sufficient traction that laboratory paradigms designed to detect RM deficits in psychopaths are now sometimes used as criteria for the construct validation of psychopathy assessment instruments (e.g., Epstein, Poythress, & Brandon, 2006; Lynam, Whiteside, & Jones, 1999) and RM deficits have been identified as promising target areas for the study of psychopathy treatment (Wallace, Schmitt, Vitale, & Newman, 2000; Wallace, Vitale, & Newman, 1999). Nevertheless, because research on the relation between psychopathy and RM deficits has not been the subject of a systematic review, conclusions regarding the validity of the RMH have required further scrutiny. Our review yielded a number of novel and important findings concerning the RMH, many of which suggest fruitful directions for research on the RMH and other etiological models of psychopathy.

Successful Corroborations of the RMH

The results of the present review lend some support to the RMH. The estimated effect size for the relation between RM deficits and psychopathy was not insubstantial, falling in the small to medium range ($r = .20$, $d = .41$). As such, RM deficits assessed at the level of individual RM tasks account for ~4% of the variance in psychopathy scores.

Although the RMH effect sizes show significant variation across tasks, we found that a number of RMH tasks exhibited particularly robust correlations with global psychopathy. For several tasks, the associations among RM deficits and psychopathy were in the medium to large range ($r > .30$). These tasks include fear recog-

nition, flanker, attentional blink, gaze, and the emotional Stroop. Although these tasks were associated with impressive effect sizes, these results should be interpreted with caution given the small number of studies associated with tasks yielding larger effect sizes. Nevertheless, other tasks frequently used by proponents of the RMH, such as the picture word task and passive avoidance tasks other than the go/no-go, yielded effect sizes close to the medium range ($r = .25$, $r = .26$, respectively).

Several results of this meta-analysis are consistent with theoretical predictions derived from the RMH. RMH proponents have offered the model as an alternative to Lykken's (1995) low fear hypothesis, arguing that the RMH is "both more specific and more general than traditional accounts emphasizing low fear or insensitivity to punishment cues" (Newman, Schmitt, & Voss, 1997, p. 564). According to the RMH, psychopathic individuals show fear insensitivity, but only in the context of a competing dominant response set. Furthermore, the RMH is broader than competing hypotheses in that it predicts abnormalities beyond those predicted by fear insensitivity theories, namely, to affectively neutral stimuli. Our results lend indirect support for this position, as evidenced by the relative similarity in effect sizes across affectively laden versus affectively neutral tasks. Moreover, this finding appears to run counter to the low fear model.

Furthermore, although only a limited number of studies have examined psychopathy subdimensions, the results suggest that Factor I, which assesses the core affective and interpersonal deficits of psychopathy, and Factor II, which assesses a chronic antisocial lifestyle, display similar associations with RM deficits. These results suggest that RM deficits are associated with psychopathy globally, and are not limited to only one feature of psychopathy, such as impulsivity. Nevertheless, because few studies examined these psychopathy subdimensions, this conclusion must remain tentative.

Finally, proponents of the RMH have made efforts to establish the uniqueness of RM deficits to psychopathy and disentangle such deficits from related disorders of disinhibition. As the literature currently stands, psychopathy appears to be marked by RM deficits that are somewhat different in nature from those of most other disorders.

Questions Raised by Our Review

Although the present review reveals provisional support for certain aspects of the RMH, it raises a number of questions and concerns.

Publication bias. Our meta-analytic review yielded evidence for potential publication bias using two different methods: (a) comparison of effect sizes from published versus unpublished studies and (b) examination of the funnel plot. With respect to (a), the effect size from unpublished studies was close to zero, raising questions concerning the robustness of the RMH. Moreover, virtually all these studies drew on RMH tasks (e.g., go/no-go task, picture word task, spatially separated Stroop task) that are widely used in the psychopathy literature, as well as validated measures of psychopathy, such as the PCL-R. At the same time, this conclusion was based on only 13 unpublished studies, so conclusions regarding potential publication bias must remain tentative. With respect to (b), our funnel plot analyses pointed to a marked paucity of small sample size studies yielding small effect sizes. Although these findings are also suggestive of publication bias, funnel plot analyses are sometimes open to alternative explanations (Sterne et al., 2011). In the case of the present meta-analysis, many of the studies yielding larger effects derived from prison samples, which were themselves generally smaller in size than studies from college and community samples. If the RMH is a more valid model for "clinical" as opposed to "subclinical" psychopathy (Widom, 1977), the funnel plot asymmetry we observed may reflect on the boundary conditions of the RMH rather than on publication bias. At the same time, it is clear that further research will be needed to rule out the possibility that the overall effect size for the RMH in our meta-analysis was overestimated by publication bias.

Multiple corroboration and constructive replication. When appraising a theory, one should ideally expect it to hold across multiple corroborations and constructive replication (Lykken, 1968), the latter often known as conceptual replication. Multiple corroboration refers to the ability of findings to support a hypothesis across substantially different paradigms or sources of evidence (e.g., laboratory tasks, self-report measures, and behavioral observations). Similar to multiple corroboration, constructive (conceptual) replication entails the corroboration of a finding using a variety of experimental methods (e.g., sampling frame, experimental paradigms) that differ from those in the original study. In essence, the validity of an etiological theory depends in part on its ability to hold across different operationalizations of the same construct using diverse experimental methods. In some cases, the RMH appears to have achieved the criterion of multiple corroboration; however, in others, the hypothesis received less support.

On the one hand, we found encouraging support for the theory using certain RM tasks. As mentioned previously, the relation between RM deficits and psychopathy was moderately pronounced for passive avoidance (excluding go/no-go), gaze, flanker, fear recognition, and picture word tasks, and was medium to large in magnitude for prison, inpatient, and primary school samples. Lending further support for the RMH and perhaps most impressively, moderator analyses yielded roughly comparable effect sizes across emotionally neutral and laden tasks. These results are consistent with the position that psychopathic individuals display cognitive or attentional abnormalities that are independent of emotional processes (Newman et al., 1997).

On the other hand, we found marked inconsistency in the relation between RM deficits and psychopathy across attempts at multiple corroboration. RM effect sizes varied, in many cases substantially, based on psychopathy measures, experimental paradigms, and dependent measures used, as well as the samples examined. Our results suggest significant variability in the RM effect ranging from a nontrivial proportion of effect sizes in the opposite direction from prediction (19%) to medium to large positive effect sizes. Nevertheless, further investigation on task moderators is needed because of the small number of studies examining some of the laboratory tasks.

When evaluating the heterogeneity of effect sizes across RMH tasks, it is important to consider that a small effect size for a given task does not necessarily reflect on the validity of the RMH, but could instead reflect the limited validity of the task itself. Hence, examination of results for a single RMH task may underestimate the true predictive power of the model.

Heterogeneity of irrelevancies. Cook (1990) highlighted the importance of "heterogeneity of irrelevancies" for theory generalization. To be confident in a theory, it should hold across variations in sample and experimental design presumed to be conceptually irrelevant to the central hypotheses. Thus, in testing a theory,

researchers should ideally identify these irrelevancies and include them wherever possible to establish theory generalization.

Efforts to establish the generalization of the RMH across irrelevancies are still provisional. Although RMH advocates have made efforts to examine the RMH in diverse populations (e.g., Vitale & Newman, 2001), most effects sizes included in this review (78%) draw on purely male samples, 35% on purely White male samples, and 51% on prison samples. Furthermore, the results of the present meta-analysis raise questions regarding the generalization of the RMH across race. Indeed, the relationship between psychopathy and RM deficits became more pronounced as the concentration of White individuals in the sample increased. Nevertheless, these results may be due partially to biases in psychopathy assessment tools and potential tendencies to overidentify individuals in certain ethnicities (e.g., Blacks) as psychopathic. In particular, some authors have argued that psychopathy, at least as assessed by the PCL-R and its derivatives, possesses a somewhat different meaning in Blacks than in Whites (see Sullivan & Kosson, 2006, for a review). This conclusion could reflect slope bias in commonly used psychopathy assessment instruments, differences in the construct of psychopathy across race, or both. As a consequence, our findings for race differences in the generalizability of the RMH may not reflect negatively on the RMH itself.

Other "irrelevancies" that yielded moderator effects in our review included sample setting, psychopathy measure, and sample age. Despite differences in effect sizes across demographics, the RMH generalized across several other irrelevancies, such as gender, analytic strategy, comparison group, and PCL-R cutoff score. Indeed, one of the few unique predictions of the RMH, namely, that RM deficits will emerge using affectively neutral stimuli, appears to generalize to females on the picture word task. Nonetheless, because few studies included in the review examined these irrelevancies, the analyses may have been underpowered to detect differences across some moderators.

Although these failures to generalize across irrelevancies raise questions regarding the RMH, the extent to which such shortcomings apply to the RMH specifically as opposed to other models of psychopathy is unclear and warrants further investigation. Some inconsistencies of the RMH, such as variability in effect sizes across race, laboratory task, and dependent measure, may apply equally to competing models, such as the low fear model.

Additionally, some heterogeneities in effect size may be indicative of heterogeneity in the broad concept of psychopathy itself. For example, Gray's (1987) model of psychopathy posits that primary psychopathy is associated with a hypoactive behavioral inhibition system (BIS), whereas secondary psychopathy is associated with a hyperactive behavioral activation system (BAS; Fowles, 1988; Gray, 1987; Lykken, 1995). Thus, some heterogeneity in effect sizes for different samples may reflect the existence of differing causal mechanisms of psychopathy or different psychopathy subtypes (see Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003, for a review).

Anomalies confronting the RMH. The successes of the RMH must also be viewed in the context of a number of anomalies. As Kuhn (1970) observed, an accumulation of anomalies—findings that are inconsistent with a paradigm—may point to systemic problems within the paradigm, and ultimately call for its modification or even outright abandonment. A thorough inventory of anomalies confronting the RMH is, therefore, essential to an adequate evaluation of its scientific status. Here, we review six sets of anomalies that pose challenges to the validity of the RMH as a comprehensive etiological account of psychopathy. The first bears on issues emanating directly from our review of the RMH literature, whereas the second through sixth derive from ancillary literature, much of which was not spawned by the RMH but nonetheless bears on its validity.

Anomalies raised by our meta-analysis. Although our meta-analysis yielded several findings supportive of the RMH, such as the presence of learning deficits even in motivationally neutral paradigms, it raised several puzzling questions that have, heretofore, received little or no explicit attention in the psychopathy literature. In particular, we found that the mean effect size for go/no-go tasks, which are traditionally deemed to be quintessential measures of passive avoidance learning, was small in magnitude ($r = .09$). This finding, which was based on 43 effect sizes and can, therefore, assumed to be reasonably stable, poses problems for the robustness of the RMH, especially given that RMH proponents have argued that "passive avoidance tasks are ideal for testing" (MacCoon et al., 2004, p. 329) the attentional deficits posited by the RMH.

As MacCoon et al. (2004) observed, it seems likely that the concept of a dominant response set lies on a continuum, with certain dominant response sets being more pronounced than others. Nevertheless, contrary to the RMH, we detected no evidence that research designs that incorporated more pronounced response sets—by virtue of a pretreatment manipulation—yielded more marked RM deficits than designs that incorporated less pronounced response sets. To the contrary, studies lacking a pretreatment manipulation yielded nonsignificantly higher effect sizes than studies containing this manipulation, rendering low statistical power an unlikely explanation for these negative findings.

We also found no clear evidence that tasks derived from more recent incarnations of the RMH, such as the attentional bottleneck model, were associated with larger effect sizes than tasks derived from earlier incarnations of the RMH, including motivational versions. These results do not necessarily challenge the validity of the RMH per se (especially as the effect sizes for attentional bottleneck tasks were consistently in the predicted direction), although they provide inconclusive support for the position that revisions to this model over time have enhanced its predictive power.

The presence of deficits in the absence of a clear-cut dominant response set. 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. The RMH posits that in the absence of a well-defined focus of initial attention, psychopathic and nonpsychopathic participants should not differ in their laboratory performance. Nevertheless, psychopaths have long been demonstrated to display deficits in classical conditioning and quasi-conditioning paradigms involving aversive stimuli, such as electric shock or loud blasts of white noise, even in the absence of an instructional set to attend to a primary stimulus. For example, psychopaths display less efficient electrodermal classical conditioning in response to neutral sounds that have been paired repeatedly with aversive stimuli, and lower electrodermal activity in anticipation of aversive stimuli (see Hare, 1978; Lorber, 2004; Lykken, 1995, for reviews). For example, Dindo and Fowles (2011) found that scores on the PPI Fearless

Dominance higher-order dimension predicted lower skin conductance responses in anticipation of a loud (102 decibel) white noise blast during a count-down period even though participants were not asked to focus on the numbers appearing on the computer screen during the count-down. More important, deficits in count-down (or count-up) paradigms have been found not only in early psychopathy studies (e.g., Hare, 1965a), but in more recent studies using well-validated psychopathy measures, such as the PCL (Ogloff, Wong, & Greenwood, 1990) and PPI (Dindo & Fowles, 2011).

Moderation of findings by aversive valence. The RMH predicts that deficits among psychopathic individuals should emerge even in the presence of motivationally neutral stimuli. As noted earlier, our meta-analysis offered some support for this hypothesis, as laboratory deficits among psychopathic participants emerged even on motivationally neutral measures, such as the picture word task. Nevertheless, other findings in the psychopathy literature are less consistent with the RMH. In a meta-analytic review of psychophysiological studies, Lorber (2004) reported that the levels of task-related skin conductance activity (but not heart rate) were significantly moderated by valence, with lower skin conductance levels emerging for psychopathic participants only when stimuli were negative/aversive.

The classic passive avoidance learning study by Schmauk (1970) poses a further challenge to the RMH. Schmauk used Lykken's (1957) mental maze paradigm, in which participants must learn a predetermined sequence of lever presses, some of which have been surreptitiously "baited" with punishment. Schmauk supplemented Lykken's (a) electric shock punishment condition with two additional conditions: (b) social punishment, in which the experimental disapprovingly said "Wrong" following each incorrect baited response and (c) monetary punishment, in which the experimenter removed a quarter from a pile of 40 stacked quarters after each incorrect baited response. The RMH posits that in the presence of a dominant response set; in this case the experimental demand to master the sequence of lever presses, psychopathic participants should be equally oblivious to extraneous stimuli across all three conditions. Schmauk replicated Lykken's finding that, compared with normal comparison participants, psychopaths commit more passive avoidance errors in the physical punishment (electric shock condition) and he extended this result by demonstrating comparable deficits in the social punishment condition. In contrast, psychopaths performed nonsignificantly *better* than normals in the monetary punishment condition. This pattern of findings is inconsistent with the RMH, which posits that learning deficits should not be moderated by the content of the extraneous stimuli. Schmauk's results are instead more consonant with the low fear model, as only the physical and social punishment conditions would be expected to induce fear in nonpsychopathic participants and thereby engender between-groups differences. One limitation of Schmauk's study is that he operationalized psychopathy in terms of scores on the MMPI, which are not optimal for identifying primary psychopathy (Lilienfeld, 1994). Nevertheless, if Schmauk's findings can be replicated using better validated measures of psychopathy, such as the PCL-R, they would be challenging to reconcile with the RMH.

Lexical decision-making findings. A well-replicated finding in both the adult and child literatures is that psychopathic individuals, especially those with elevated Factor I features, tend to display a failure to show facilitation of word recognition (e.g., Loney, Frick, Clements, Ellis, & Kerlin, 2003; Williamson et al., 1991; see also Reidy, Zeichner, Hunnicutt-Ferguson, & Lilienfeld, 2008). As noted earlier, when asked to identify whether a string of letters is a word, most individuals display RT facilitation when the word is emotional compared with when it is nonemotional. In contrast, individuals with elevated levels of the affective features of psychopathy tend not to display such facilitation, suggesting that affective deficits render them less responsive to the emotional valence of words (Williamson et al., 1991). These results appear difficult to square with the RMH, which posits that the affective valence of stimuli is irrelevant to psychopathic individuals' laboratory deficits.

Lorenz and Newman (2002, p. 99) attempted to reconcile these lexical decision-making findings with the RMH by positing that the requirement to identify a letter string as a word or nonword generates a dominant response set, whereas the emotional quality of the word is extraneous to participants' primary goals and is, therefore, processed less efficiently by psychopathic participants. Nevertheless, this line of reasoning conflicts with explanations offered previously by RMH proponents. Specifically, these proponents have elsewhere argued that psychopathic individuals exhibit deficits on a spatially separated Stroop task but not a standard Stroop task because the dominant and peripheral stimuli (color of ink and the content of words, respectively) are in the same spatial location in the latter task (Hiatt et al., 2004), and therefore, do not engender an attentional bottleneck. This logic implies that psychopathic individuals should not exhibit deficits on lexical decision-making tasks because the dominant and extraneous stimuli (letter strings' status as words or nonwords and their emotional valence, respectively) are in the same spatial location. Hence, psychopaths' deficits on lexical-decision making tasks are not readily explained by extant versions of the RMH.

Preattentive emotional processing differences. The RMH, at least its attentional bottleneck version, proposes that the serial processing of information generates a backup of information processing in psychopathic individuals, precluding their adequate processing of subsequently presented information. Hence, the RMH predicts that psychopathic individuals should not display deficits when cognitive processing is preattentive, that is, when it occurs too rapidly for attention to be directed to stimuli. In contrast, the low fear model (Lykken, 1995) allows for the possibility that psychopathic individuals display fear recognition deficits preattentively. This hypothesis follows from work demonstrating that most individuals are exquisitely sensitive to threat cues and respond to them "in the blink of an eye" (Ohman, 2008, p. 165), that is, before conscious attention can be engaged.

This hypothesis was tested by Sylvers, Brennan, and Lilienfeld (2011) in a sample of children with varying levels of CU traits. CU traits, which include the absence of guilt, empathy, and deep emotions, may be precursors to the core affective deficits of psychopathy in adulthood (Frick, Ray, Thornton, & Kahn, 2014). Sylvers et al. administered a continuous flash suppression (CFS) task, in which one eye receives a dynamic, continually changing (every 20 ms) flow of stimuli, usually Mondrian images, and in which 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. Previous work using the CFS paradigm revealed that most individuals become aware of fearful faces more quickly than faces displaying other emotions (Yang, Zald, & Blake, 2007). As predicted by the low fear model, Sylvers et al. found that, compared with other children, children with elevated levels of CU traits did not display more rapid detection of fearful (and to a lesser extent, disgusted) faces even when they were presented preattentively. This finding is not readily explained by the RMH given that the facial stimuli emerged from suppression too rapidly to engage attention. Nevertheless, the results of Sylvers et al. will require replication in an independent sample and extension to adults with differing levels of psychopathic traits.

Relevance to both psychopathy dimensions. Our meta-analysis revealed no significant difference in effect sizes for the two major psychopathy dimensions, although the effect sizes were slightly larger for Factor I. Nevertheless, a study by Baskin-Sommers, Zeier, and Newman (2009) calls into question the relevance of recent attentional versions of the RMH to both psychopathy dimensions. In a large prison sample, the authors administered a self-report measure of attentional control (Derryberry & Reed, 2002) in conjunction with three well-validated measures of psychopathy: the PCL-R, the PPI-R, and PPI factor estimates derived from the Multidimensional Personality Questionnaire (Tellegen, in press). The attentional control measure was designed to assess individual differences in the capacity to focus attention on a primary response set (e.g., "When concentrating, I can focus my attention so that I become unaware of what's going on in the room around me") and to shift attention toward important stimuli and away from extraneous stimuli when needed (e.g., "When a distracting thought comes to mind, it is easy for me to shift my attention away from it"). Contrary to predictions, attentional control scores were positively correlated with Factor I but negatively correlated with Factor II across all three psychopathy measures. These results suggest that the capacity of psychopathic individuals to focus attention on a dominant response set, as implied by the RMH, may be specific to Factor I and may not extend to both major dimensions of psychopathy. Nevertheless, the extent to which individuals can report accurately on their attentional capacities is unclear. It is possible, for example, that individuals with elevated Factor I scores overestimate or overstate their cognitive capacities (see Ray et al., 2013, for evidence that Factor I scores on psychopathy self-report measures are sometimes associated with socially desirable responding).

Summary. Carnap's (1947) principle of total evidence requires that a theory account for all relevant evidence, not merely evidence generated deductively from this theory. As we have seen, a number of findings in the psychopathy literature, some of them replicated across independent laboratories using well-validated psychopathy measures, are difficult to square with the RMH. Most notably, consistent deficits in psychopathic individuals, such as deficits in aversive conditioning paradigms, have been reported even in the absence of a clear-cut dominant response set. Another reasonably consistent finding, not readily explained by the RMH, is that the affective valence of secondary stimuli sometimes moderates the existence of laboratory deficits among psychopathic individuals. In particular, stimuli capable of inducing fear tend to be associated with larger differences between psychopathic and nonpsychopathic participants than are other stimuli (e.g., Lorber, 2004). These results appear to be better explained by the low fear model (Lykken, 1995), although this model is characterized by its share of anomalies as well (e.g., Newman & Brinkley, 1997), including the finding that fear deficits are largely unrelated to the externalizing and disinhibitory features of psychopathy (Patrick et al., 2009).

Although none of the anomalies we have identified may be definitive falsifiers of the RMH (see Meehl, 1978, for a discussion of the challenges of falsifying theories in clinical psychology research), collectively they raise questions regarding the capacity of this model to provide a comprehensive account of the deficits of psychopathy. Moreover, the results of Baskin-Sommers et al. (2009), if generalizable beyond self-report indices of attentional control, suggest that the RMH may not extend to Factor II psychopathy features, namely, a chronic pattern of antisocial and impulsive behaviors. In our view, the onus of proof now falls on RMH advocates to either (a) explain how these anomalies can be explained by the RMH or (b) modify the RMH to accommodate these anomalies.

Challenges to falsifiability. One frequently invoked criterion, among numerous others, for theory appraisal in science is the extent to which a theory can in principle be falsified (Meehl, 2002). In this regard, a challenge to evaluating the RMH is the apparent difficulty in falsifying some of its predictions. In particular, the definition of a dominant response set may lack sufficient clarity to allow rigorous empirical testing. Often identified as the primary task, dominant responses are sometimes established through the attentional manipulation of the researcher, whereas at other times they are not. It is frequently unclear how one can know whether a dominant response set has been established in participants without examining data after the fact, nor is it evident what would constitute a valid manipulation check to ascertain the presence of a dominant response set. In some cases, it may be difficult to know whether a dominant response set has been established without determining whether psychopathic individuals fail to show a shift in that response in reaction to environmental cues. Hence, without clear a priori specification, the operationalization of a dominant response set can be potentially tautological, in some cases raising questions regarding the falsifiability of the RMH. If a study does not yield the RM deficits predicted by the RMH, one could readily invoke the ad hoc hypothesis that a dominant response was not present. Conversely, if a study yields behavioral deficits in the absence of any apparent dominant response set, one could readily invoke the ad hoc hypothesis that a dominant response set was present.

Limitations of Our Review

The RMH is a complex etiological model of psychopathy. The frequent evolutions of the model and the use of a multitude of experimental tasks of varying validity render the systematic evaluation of such a theory challenging. As such, the present review is marked by several limitations that are worth noting.

One limitation is the relatively small number of studies examining nonmal and noncorrectional populations. As a consequence, a number of the subgroup and meta-regression analyses may have been underpowered to detect differences across moderators. Furthermore, the lack of sufficient study diversity in the literature, resulting in small cell sizes and a correspond-

ing lack of statistical power, precluded us from parsing the differential contributions of moderators (e.g., race, gender, and sample setting) to the RM effect size. Some of the moderators we examined were highly associated and thereby potentially confounded. This statistical confounding of moderators makes it difficult to interpret the true meaning of any one moderator. Because of the limited number of studies examining certain moderators (e.g., gender), limitations in statistical power precluded us from performing analyses aimed at disentangling confounded moderation (see Lipsey, 2003). Thus, future research should focus on the generalization of the RMH across a heterogeneous pool of irrelevancies (Cook, 1990) and continue to examine the RMH across diverse moderators such as gender, race, sample setting, and nature of control group.

To maximize the inclusion of studies, no methodological restrictions were imposed for study selection. Thus, studies presumably ranging in methodological quality were included. This point may be particularly relevant to variation in effect sizes across laboratory tasks. The liberal inclusion criteria allowed for a maximization of our study sample size (*k*), but also resulted in the inclusion of studies drawing on RMH tasks potentially ranging in validity (e.g., degree to which a dominant response set is established over a secondary stimulus). Some of the tasks included in the review are commonplace in the RM literature (e.g., go/no-go, picture word task, instructed fear task), whereas others could be considered more exploratory (e.g., BART, global-local processing, visual search). MacCoon et al. (2004) suggested that the dominant, or primary, versus secondary aspects of a RMH task can be conceptualized dimensionally; however, few or no explicit criteria exist for assessing the quality of the operationalization of RM tasks. As such, it is difficult to interpret the variation in effect sizes across this review, as they may reflect to some extent variation in the quality of RM measures. In the lone case in which we were able to examine a clear-cut prediction regarding the magnitude of a dominant response set, namely, the comparison of pretreatment with non-pretreatment go/no-go studies, we found no support for RMH predictions. As the body of research on RM expands, proponents of the RMH should make concerted efforts to clarify criteria for evaluating RM tasks.

Finally, when coding effect sizes for the present review, for each publication we were guided by the hypotheses of the authors. Such an approach was undertaken to minimize the odds of potential bias on the part of the authors of the present review. Nevertheless, this approach could serve as a potential limitation in that some studies (e.g., Arnett et al., 1997) postulated a priori hypotheses that are open to legitimate debate, such as those relating to physiological outcome measures (e.g., heart rate). In addition, some of the studies yielding negative findings used methodologies that may contribute to the lack of support for the RMH. For example, Vitale et al. (2005) and Arnett et al. (1997) used an unusually long intertrial interval on the go/no-go task, which may have contributed to the negative findings for passive avoidance errors. Nevertheless, sensitivity analyses removing the studies in question revealed no differences in the overall effect size. It is also worth noting that both of the aforementioned studies found support for the RMH on other measures used (e.g., picture word, skin conductance) on the same samples.

Future Directions

Our results highlight the importance of several potentially fruitful directions for research. Research on the causes of psychopathy is still in its relative infancy. Understanding the etiology of psychopathy and related psychological disorders is important for the development of treatment and prevention efforts (Gough, 1971). We address four important areas for future research.

First, the inconsistency in effect sizes across experimental tasks highlights important concerns regarding the use of laboratory paradigms in psychological research. Although RMH researchers have used a wide variety of laboratory tasks across studies, few studies have examined more than one or two tasks simultaneously. Paradigms used to assess RM deficits necessarily rely on laboratory data or "t-data," defined as data drawn from standardized situations created in a laboratory, where behavior can be largely objectively observed and measured (Block, 1977; Cattell, 1965). Despite the utility of t-data for the objective measurement of behavior, such data have been criticized for their unreliability and tenuous associations with other relevant indicators of personality, including those measured by other forms of t-data (Block, 1977; Epstein, 1979). Thus, the measurement error inherent in laboratory data is almost certain to attenuate the true relations between psychopathy and RM deficits. Moreover, few researchers have examined the extent to which different RM measures are correlated, rendering unclear whether these measures are detecting the same construct. Similarly, given that RMH researchers attempt to relate measures of personality to behavioral performance and physiological outcomes, method variance across variables may limit the magnitude of effect sizes (see Blonigen et al., 2010, for a discussion of this issue in the psychopathy literature). The use of multiple RM tasks within the context of the same study would allow for their aggregation into a latent variable using statistical techniques, such as structural equation modeling.

Indeed, Patrick et al. (2013) highlighted the utility of examining neurobehavioral traits as intermediate phenotypes between laboratory data and real-world clinical problems. Such a neurobehavioral trait approach draws on a multimeasurement, latent variable approximation across many manifest indicators. Patrick et al. illustrated this approach through an examination of the construct of trait inhibition-disinhibition. First, trait inhibition-disinhibition was linked empirically to relevant phenotypic features, such as externalizing behavior. Second, trait inhibition-disinhibition was linked empirically to relevant neurological functions. Ultimately, a construct-network was modeled statistically using trait inhibition-disinhibition as a bridge between externalizing behavior and underlying neurophysiology. Patrick and colleagues emphasized the importance of tying laboratory indicators to well-validated trait measures and in turn clinical problems to clarify the role of neurobiological processes in psychological disorders. Future research on the RMH would benefit from such an approach to address issues of measurement error and clarify the real-world expression of RM deficits.

Moreover, this approach is broadly consistent with the framework of the Research Domain Criteria (RDoC) initiative proposed recently by the National Institute of Mental Health (Cuthbert & Insel, 2013). This approach encourages the use of multiple endophenotypic indicators, including laboratory measures, to detect variation in psychobiological systems. Several units in the current RDoC matrix seem

particularly relevant to further testing of the RMH as well as competing models of the etiology of psychopathy, such as the low fear model. For example, within the domain of cognitive systems, the RDoC matrix emphasizes the area of attention, which is particularly relevant to recent incarnations of the RMH. The RDoC initiative also includes threat-based systems, which are particularly relevant to motivational models of psychopathy (e.g., Fowles & Dindo, 2009; Lykken, 1995). The RDoC model might ultimately help to better test the validity of the RMH against that of the low fear model (see also Blair, 2015, for a discussion).

Second, one crucial question that remains unanswered is whether RM deficits are causally implicated in psychopathy rather than merely descriptive. It is possible that RM deficits are merely manifestations or consequences of other core deficits in the disorder, including dysfunctional emotional/motivational systems. For example, one potential reason why psychopathic individuals may not attend highly to fear relevant stimuli in the context of fear potentiated startle paradigms (e.g., Baskin-Sommers et al., 2013) is that they are not particularly frightened of these stimuli to begin with. If so, the causal directionality may be the opposite from that posited by the RMH in the context of FPS and similar fear-related paradigms. An instructive example can be found in the literature on anxiety disorders. Recent evidence raises the possibility that attentional biases toward threat may be causally implicated in anxiety disorders, although the evidence is somewhat equivocal. At the same time, evidence also suggests that changes in anxiety levels may in turn contribute to changes in attention (Van Bockstaele et al., 2014). In the case of the psychopathy literature, researchers should similarly be cognizant of the possibility that attentional deficits may result from, instead of or in addition to contributing to, the core features of the disorder.

In this regard, longitudinal designs may allow researchers to conduct "theoretically riskier" (Meehl, 1978) tests of causal models, including attentional models generated by the RMH. For example, it will be important for researchers to demonstrate that early RMH deficits presage the development of CU traits in children, which may themselves be precursors of psychopathy (see Frick & White, 2008 for a review). Longitudinal designs will be needed to establish whether or not RM deficits are merely correlates of psychopathy, risk factors (i.e., correlates that precede the disorder), or causal risk factors (i.e., risk factors that are etiologically involved in psychopathy; Kraemer et al., 1997). At this point, RM deficits can be construed only as correlates of psychopathy.

Third, it will be essential for researchers to place the RMH within the context of well-established personality dimensions. Growing data indicate that scores on psychopathy and its subdimensions can be well approximated by scores on normal-range personality dimensions, such as those from the FFM of personality. In particular, global psychopathy seems to be especially tied to low levels of conscientiousness and agreeableness as well as certain facets of extraversion and neuroticism (Lilienfeld et al., 2014; Lynam & Derefinko, 2006). Given the diversity of largely distinct personality traits with which psychopathy is closely associated, it may be more reasonable to expect the RMH or other etiological models to account for only some personality features of psychopathy rather than all such features. Although early versions of the RMH were tied explicitly to motivational models of personality such as those of Eysenck and Gray (e.g., Newman, 1987), more recent attentional models of the RMH have increasingly moved away from linking psychopathic deficits to personality traits.

Nevertheless, the RMH may serve as an adequate explanation of at least certain personality features of psychopathy, such as those tied to risk for disinhibited or impulsive behavior. The triarchic model (Patrick et al., 2009) may provide a useful framework for the continued examination of psychopathy within the context of the RMH. This model posits that psychopathy is an amalgam of three largely separable personality traits, namely, boldness, meanness, and disinhibition. Disinhibition may be particularly relevant to the RMH. According to Patrick and colleagues, disinhibition refers to a lack of planfulness, failure to delay gratification, and deficits in restraining behavior. In fact, they identified impulsive behavior resulting in maladaptive consequences (e.g., punishment) as one key behavioral manifestation of disinhibition. The findings of Newman et al. (1987), in which forced reflection on the consequences of behavior diminished RM deficits, suggest that impulsivity or disinhibition may be an important explanatory component of the RMH. Although our findings, which revealed a nonsignificant difference in effect sizes between the two psychopathy dimensions, yielded equivocal support for this possibility, future research would benefit from a more nuanced examination of the dimensions of psychopathy most related to RM deficits within the context of the triarchic model.

The RMH may also help to explain certain features of psychopathy within dual process models (e.g., Fowles & Dindo, 2009), which posit that psychopathy is a joint product of fearlessness and disinhibition. Further work may indicate that RM deficits are more specific to one of these etiological pathways, although again our meta-analysis found little support for this possibility within the framework of the classic two-factor model of psychopathy. Nevertheless, further work with measures that provide relatively pure indicators of these divergent etiological pathways (e.g., the Triarchic Psychopathy Measure; Patrick, 2010) may yield different results.

A fourth and final future direction concerns the implications of the RMH for ameliorating and ultimately preventing the deficits of psychopathy. Even if the RMH is not an adequate causal model of psychopathy, ameliorating psychopaths' attentional deficits might nonetheless be a useful therapeutic strategy. This might be true even if the attentional deficits of psychopathy are linked bidirectionally to the disorder, as may be the case for certain anxiety disorders (Van Bockstaele et al., 2014). Indeed, recent work by Baskin-Sommers, Curtin, and Newman (2015) suggest that attentional retraining strategies may diminish the deficits of psychopathic individuals on well-established RMH tasks. Nevertheless, as the authors themselves acknowledged, more work will be needed to ascertain whether these attentional training regimens generalize outside of the laboratory to psychopaths' real world behavior, such as their risk for antisocial activity and substance use. Other studies suggest that some of the emotional deficits associated with psychopathy may also be ameliorated by attentional-based interventions. For example, manipulations instructing psychopathic individuals to feel the emotions of others appear to increase empathic response as evidenced by neural activation (e.g., Meffert, Gazzola, den Boer, Bartels, & Keyers, 2013). Moreover, although the efficacy of attentional retraining for anxiety disorders, mood disorders, and other conditions appears to have a mixed scientific track record (Van Bockstaele et al., 2014;

Cristea, Kok, & Cuijpers, 2015), such retraining may prove to be effective for at least certain psychopathy dimensions if it is targeted explicitly to the attentional deficits posited by the RMH.

The present review provides tempered support for the RMH as an etiological model of psychopathy. It seems likely that RM deficits are at least one element of the clinical picture of psychopathy, although their causal status requires clarification. Still, our review raises significant concerns regarding the RMH's robustness and comprehensiveness. In many respects, our conclusions and unresolved questions bring us back full circle to the central animating question that motivated this review, namely, the etiology of psychopathy. Like many literature reviews, our examination of the evidentiary basis of the RMH raises more questions than answers. In particular, in light of the anomalies we have identified that render the RMH difficult to reconcile with some of the extant psychopathy literature, especially the presence of clear-cut laboratory deficits even in the absence of a dominant response set, it seems unlikely that the RMH, at least in its current form, can offer a complete story of the origins of psychopathy.

At the same time, the growing evidence that psychopathy appears to be a constellation or configuration of numerous personality traits that are themselves empirically distinct (Lilienfeld et al., 2014; Lynam & Derefinko, 2006) gives us pause. Such evidence suggests that the longstanding assumption that psychopathy is a monolithic entity may be in error (Lilienfeld, 2013; Lilienfeld & Fowler, 2006; Patrick et al., 2009). Furthermore, such evidence raises the deeper question of whether any single theoretical model, whether it be the RMH or its competitors, including the low fear model, can fully explain the enormously diverse clinical picture of psychopathy. Put somewhat differently, given the phenotypic heterogeneity of psychopathy, it may be asking too much of any one model to offer a comprehensive account of its causes. If so, the shortcomings of the RMH identified by our review may point to a need to reexamine our traditional conceptualizations of psychopathy at least as much as the model itself.

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Received February 8, 2014
Revision received April 13, 2015
Accepted May 12, 2015 ■