



## Review article

## Psychopathy and response inhibition: A meta-analysis of go/no-go and stop signal task performance

Steven M. Gillespie<sup>a,\*</sup>, Jessica Lee<sup>b</sup>, Rachael Williams<sup>a</sup>, Andrew Jones<sup>b,c</sup><sup>a</sup> Department of Primary care and Mental Health, University of Liverpool, UK<sup>b</sup> Department of Psychology, University of Liverpool, UK<sup>c</sup> Liverpool Centre for Alcohol Research, University of Liverpool, UK

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## ABSTRACT

Traditional and more modern conceptualizations of psychopathy cite problems with impulse control. However, the extent to which these problems represent a cardinal feature of the disorder has been debated. In this study, we conducted a preregistered systematic review and meta-analysis, searching Embase, Medline, PsycINFO, and PubMed, for studies from inception to January 6th, 2022. We included 21 studies, published between 2009 and 2021, that reported on the relationship of psychopathy with performance on the go/no-go or stop signal task. A multilevel random-effects meta-analysis, including 43 effect sizes from 17 studies (total  $N = 1394$ ), showed a significant pooled association between psychopathy and response inhibition  $r = -0.143$  (95 % CI:  $-0.250$  to  $-0.034$ ). The relatively small effect size, although statistically significant, calls in to question the extent to which difficulties in response inhibition should be considered a cardinal feature of psychopathic personality. The strength of the relationship did not significantly differ between non-criminal and criminal samples, gender, task type, tasks with or without an affective component, or by psychopathy trait dimension.

## 1. Introduction

Psychopathy is a unique personality disorder that manifests as a constellation of affective (e.g., reduced guilt and empathy), interpersonal (e.g., interpersonal manipulation), and impulsive/irresponsible (e.g., recklessness) features (Hare, 2003; Patrick et al., 2009). Although a diagnosis of psychopathy can only be made in adult populations using the Hare Psychopathy Checklist – Revised (PCL-R; Hare, 2003), psychopathic tendencies can manifest from an early age, and a diagnosis of ‘limited prosocial emotions’ can be made using the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5; American Psychiatric Association, 2013) in children and young people with an existing diagnosis of conduct disorder (American Psychiatric Association, 2013). In children and in adults, psychopathic tendencies are associated with a heightened risk for aggression, and people with elevated psychopathic traits account for a disproportionate amount of all violent crimes, incurring a considerable economic and societal cost (De Brito et al., 2021). Taken together, it has been argued that psychopathy represents a ‘unified theory of crime’, that accommodates categorical and dimensional conceptualizations, has predictive value in understanding aggressive and antisocial phenotypes across diverse

populations, and is applicable across the lifespan (DeLisi, 2009, 2016).

Varying conceptualisations of psychopathy support the presence of either two (Hare, 2003), three (Cooke and Michie, 2001; Patrick et al., 2009), or four (Hare, 2003), separable dimensions, all of which index the affective, interpersonal, and impulsive/irresponsible features of the disorder. These conceptualisations vary in the extent to which they consider antisocial traits as key behavioural criteria, with some suggesting that antisocial behaviour represents a downstream correlate of the other affective, interpersonal, and impulsive/irresponsible features (Cooke and Michie, 2001; Skeem and Cooke, 2010). Although psychopathy has traditionally been studied in forensic contexts, psychopathic traits are distributed along a continuum (Edens et al., 2006; Guay et al., 2007), and are present to varying degrees in samples from the general population (Coid et al., 2009; Neumann and Hare, 2008).

Traditional models of psychopathic personality, including the seminal work of Cleckley (1941), as well as more recent models, share a common consideration that psychopathy is, in part, characterized by impulsivity. For example, the two-factor/four-facet model of the PCL-R (Hare, 2003), the most widely used and best validated assessment of psychopathy in offender populations, includes impulsivity as one of 20 clinician rated features of psychopathic personality. Similarly, the

\* Correspondence to: Department of Primary Care and Mental Health, University of Liverpool, Eleanor Rathbone Building, Liverpool L69 7ZA, UK.

E-mail address: [steven.gillespie@liverpool.ac.uk](mailto:steven.gillespie@liverpool.ac.uk) (S.M. Gillespie).

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Psychopathic Personality Inventory (PPI; Lilienfeld and Andrews, 1996), and its revised edition (PPI-R; Lilienfeld and Widows, 2005), include a higher order factor labelled impulsive antisociality (Benning et al., 2005), and the Triarchic model of psychopathy (Patrick et al., 2009) includes disinhibition as one of its three core components. Conceptually, it has been argued that the construct of psychopathy also captures the essence of low self-control, and holds predictive validity for understanding antisocial, short-lived, and hedonistic behaviours (DeLisi, 2009, 2016).

Although there is considerable theoretical and empirical support for an association of psychopathy with impulse control problems, Poythress and Hall (2011) question the assertion that impulsivity is a cardinal feature of psychopathy. In their non-systematic review of the literature, they cite evidence that: (1) different variants of psychopathy exist that are differentially characterized by impulsive tendencies; (2) the multidimensionality of psychopathic personality, and the construct of impulsivity, calls for a more nuanced view of the psychopathy-impulsivity association; and (3) so-called ‘successful’ psychopaths exist, who live in the community and who use conning and interpersonal manipulation to exploit others, but who have either not engaged in criminal behaviour, or have escaped criminal justice involvement, challenging the notion that these individuals are ‘impulsive’ (Benning et al., 2018; Lilienfeld et al., 2015; Steinert et al., 2017).

One important step toward developing a better understanding of the psychopathy-impulsivity relationship is to consider the usefulness of the term impulsivity. There is now a general consensus that the term ‘impulsive’ is largely problematic, referring to, “behaviour without adequate thought, the tendency to act with less forethought than do most individuals of equal ability and knowledge, or a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions” (International Society for Research on Impulsivity, 2022). Indeed, Strickland and Johnson (2021) went so far as to assert that impulsivity should be ‘rejected’ as a psychological construct, highlighting evidence that impulsive traits and behaviours (e.g., response inhibition, delay discounting) are largely uncorrelated, and that a specific and central neurobehavioral mechanism underlying impulsive behaviours has yet to be identified. Instead, these and other authors have attempted to distinguish different domains that are captured by the broad umbrella term impulsivity, including inattention, response inhibition, impulsive decision making, and shifting (Sharma et al., 2014; Strickland and Johnson, 2021).

The response inhibition domain of impulsivity is central to several neurobehavioral and neurocognitive system models of antisocial deviance (Blair et al., 2018; Patrick et al., 2012). The term prepotent response inhibition refers to active suppression of an action that is inappropriate in relation to its context, or interferes with a goal-driven behaviour (Mostofsky and Simmonds, 2008), and has varying been referred to as response inhibition (Dick et al., 2010), action inhibition (Eagle et al., 2008), or impulsive action (Stamates and Lau-Barraco, 2017; Winstanley et al., 2006).

In a review of fMRI studies in children with antisocial symptom sets, Blair and colleagues distinguished between the neurocognitive systems underlying empathy, the acute threat response, and response inhibition (Blair et al., 2018). While children with conduct problems, both in the presence or absence of psychopathic traits, showed dysfunction in neurocognitive systems relating to empathy, there was some evidence that individuals with psychopathy performed better behaviourally than comparison individuals on tests of executive function (Morgan and Lilienfeld, 2000), and that response inhibition might represent a risk factor for more general antisocial behaviour. However, only one study was identified that compared groups with high and low psychopathic traits during an inhibition task (the Stroop task). Similarly, in their review of neurobehavioral traits related to conduct disorder, antisocial personality, and psychopathy, Patrick et al. (2012) focus on inhibitory control and defensive reactivity. It is suggested in both models (Blair et al.,

2018; Patrick et al., 2012) that a better understanding of neurocognitive system dysfunction in psychopathy will pave the way for the development and testing of more targeted behavioural interventions to reduce antisocial behaviour.

The ability to inhibit a prepotent response is thought to rely on neural systems – including inferior frontal gyrus, anterior insula cortex, and dorsomedial frontal cortex – working together to send a Stop command to intercept the Go process, via the basal ganglia (Aron, 2011; Aron et al., 2014; Cai and Leung, 2011; Chikazoe et al., 2009; Dodds et al., 2011; Meffert et al., 2016). One of the advantages of studying a narrower construct of response inhibition, versus the broader category of impulsivity, is that specific tasks exist that allow for distinct construct measurement.

Two dominant paradigms have been devised to measure response inhibition in laboratory settings: the go/no-go paradigm (Eagle et al., 2008), and the stop signal paradigm (Schachar et al., 2007). The go/no-go task measures action restraint, and requires participants to respond to a prepotent stimulus, displayed on the majority trials, which allows for a habitual response to develop. When a less common, alternative stimulus is presented, participants are asked to withhold their habitual response. Importantly, the decision to inhibit is made upon stimulus presentation – hence action restraint. The stop signal task, on the other hand, measures action cancellation (i.e., inhibiting an already initiated action). Participants are again asked to respond to a prepotent stimulus, but on trials where a rare stop signal occurs, this occurs at some delay following a ‘response required’ stimulus. Thus, when the participant responds, an already initiated motor-response must be cancelled by withholding the response (Verbruggen et al., 2019).

Although other reviews have examined the associations of psychopathic traits and antisocial behaviour with performance on cognitive tasks, none of these reviews have focussed specifically on response inhibition as a particular domain of impulsivity. For example, one meta-analysis focussed on the association of psychopathic traits with performance on a series of tests of cognitive function, highlighting a significant psychopathy related impairment across all tasks ( $d_+ = .25$ ) (Morgan and Lilienfeld, 2000). A separate meta-analysis also showed that psychopathy was associated with impaired performance across a wide range of cognitive tasks that tap a variety of functions ( $d_+ = .42$ ) (Ogilvie et al., 2011). Thus, while there is robust evidence for a small to medium sized inverse relationship between psychopathy and performance on cognitive tasks, a quantitative synthesis of the association of psychopathic traits with response inhibition task performance does not exist.

### 1.1. Aims

Both the go/no-go and stop signal tasks have been used to examine response inhibition abilities in relation to psychopathic traits in non-criminal and criminal samples. However, many of these studies have yielded conflicting results. In this study, our aim was to examine the centrality of response inhibition deficits to psychopathic personality. To do this, we aimed to systematically review and perform a quantitative synthesis of the size and consistency of the relationship between psychopathic traits and response inhibition. We examined this relationship across non-criminal and criminal samples, including studies that assessed response inhibition using one or both commonly used behavioural measures of response inhibition – the go/no-go task (measuring action restraint), and the stop signal task (measuring action cancellation) (Schachar et al., 2007) – which both serve the purpose of distinct construct measurement (Strickland and Johnson, 2021).

When considering criminal versus non-criminal manifestations of psychopathy, the terms ‘successful’ versus ‘unsuccessful’ psychopathy have typically been used, with the label ‘successful’ referring to those individuals with elevated psychopathic tendencies who have not engaged in criminal behaviour, or who have never been caught and convicted for their criminal behaviours (Benning et al., 2018; Lilienfeld et al., 2015; Steinert et al., 2017). A recent comparison of systematic

reviews suggested that severe impulsivity in psychopathy may arise from a pathophysiological mechanism that is unique to severely elevated psychopathic traits that is not reflected in the general population (Korponay and Koenigs, 2021). Thus, the nature of any problems inhibiting a prepotent response may be markedly different in relation to non-criminal and criminal psychopathy. As such, we examined the moderating effects of sample population (non-criminal versus criminal) on the psychopathy-response inhibition relationship. We also aimed to examine the moderating effects of gender, task type (measuring action restraint versus cancellation), the inclusion of affective stimuli, and the method of assessment of psychopathic traits.

## 2. Method

We registered the protocol for a systematic review of the relationship of psychopathic traits with response inhibition in criminal and forensic samples on 1 April 2020 [CRD42020171390] using the International Prospective Register of Systematic Reviews (PROSPERO). We registered a separate protocol for a systematic review and meta-analysis of non-criminal psychopathic traits and response inhibition in community and non-offender samples on 6 July 2020 [CRD42020193362]. To test the hypothesis that psychopathic traits are differentially related to response inhibition in non-criminal versus criminal psychopathy, we have instead included both offender and non-offender samples in the same systematic review and meta-analysis. This decision increases the theoretical relevance of the review question for understanding the pathophysiology of criminal and non-criminal psychopathy. The same search terms were registered in both protocols. The procedures for data extraction and meta-analysis follow those outlined in the protocol registered 6 July 2020.

### 2.1. Search strategy

Electronic searches were conducted in February 2020 and updated on 6 January 2022. The review process followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist (Liberati et al., 2009; Moher et al., 2009). Four electronic databases (Embase, Medline, PsycINFO and PubMed) were searched using the following keyword search terms: (psychopathic OR psychopathy OR “call?us-unemotional” OR “CU traits” OR call?us OR unemotional OR “dark triad”) AND (“stop signal” OR SSRT OR “go no-go”).

### 2.2. Eligibility criteria

Articles were included in the review if the full text was available in English in a peer-reviewed journal, and the study reported the relationship of response inhibition, assessed using the go/no-go task and/or the stop signal task, with psychopathic personality traits, assessed using a validated measure e.g., the Triarchic Psychopathy Measure (TriPM; Patrick et al., 2009), the Levenson Self-Report Psychopathy Scale (LSRP; Levenson et al., 1995), the Psychopathic Personality Inventory (PPI; Lilienfeld and Andrews, 1996; Lilienfeld and Widows, 2005); or the Self-Report Psychopathy Scale Fourth Edition (SRP-4; Paulhus et al., 2016), or its earlier editions. Included studies had to report on adult only samples, including community and college samples, and samples with or without a history of offending or forensic psychiatric care or criminal justice involvement (e.g., in prison or a secure forensic hospital). Samples that included participants with intellectual/learning disability were excluded. Studies that used the go/no-go and/or stop signal task(s) in the context of punishment and reward (Brazil et al., 2013; Howard and Lumsden, 1996; Howard et al., 1997), or learning by trial and error (Newman and Kosson, 1986; Newman et al., 1990; Newman and Schmitt, 1998), were excluded. Where the results of relevant tests were unreported, a request for missing results was sent to the first or corresponding author.

### 2.3. Outcome measures

Outcome measures of interest were performance on the go/no-go task (e.g., proportion of correct responses or number of commission errors on the go/no-go task), or the Stop Signal Reaction Time (SSRT) for the stop signal task.

#### 2.3.1. Study selection

Abstracts and titles were screened for inclusion by one of the authors. Papers were excluded where it was clearly indicated that the paper did not meet the inclusion criteria. Full-text versions of the remaining articles were screened for inclusion. A second rater screened all articles at the title and abstract phase, and a further 10% at the full-text phase. Any uncertainty was resolved through consensus within research team.

To ensure comprehensiveness of the search strategy, further searches were carried out in Google Scholar, reference lists for all included studies were hand searched, and relevant authors were emailed to request any unpublished results.

#### 2.3.2. Data extraction

Data extracted from each study included: (1) publication details: author, title, year; (2) design: type of study (i.e. correlational, case-control); (3) participant details: gender, age, ethnicity; (4) outcome measures: sample size, psychopathy measurement, type of response inhibition task, number of go/no-go/stop trials in each response inhibition task, data used for calculating the effect sizes (e.g., means and SD for between group designs, Pearson's  $r$ ). For go/no-go tasks, we only extracted information about accuracy, and did not extract response time data as these do not reliably measure response inhibition using the go/no-go paradigm. For the stop signal task, we only extracted data relating to the SSRT. A second rater performed a quality check to ensure accuracy of data extraction for all papers, and any disagreements were resolved within the research team.

#### 2.3.3. Quality assessment and risk of bias

Methodological quality and risk of bias was assessed using the Appraisal Tool for Cross-Sectional Studies (AXIS: Downes et al., 2016). The AXIS scaffolds a critical appraisal of all aspects of the study design, analysis, and reporting across five areas (introduction, methods, results, discussion and other). Uncertainty in appraisal decisions were resolved within the research team.

### 2.4. Quantitative synthesis and analytic strategy

In line with recommendations (Polanin and Snijlsteit, 2016), we converted all effect sizes to a common effect size, in this case a correlation coefficient (Pearson's  $r$ ). SMDs using between subject's designs were calculated using the ‘escalc’ function, before converting with the ‘d\_to\_r’ function, of the ‘effectsize’ package (Ben-Shachar et al., 2020), running in R (R Core Team, 2018). Fisher's  $Z$  transformation was used to improve the distribution of the coefficients. Findings were back-transformed for presentation in text and Forest Plots. As several studies included more than one eligible effect size (e.g., a correlation between inhibitory control and multiple indices of psychopathy), we conducted a multilevel, random-effects meta-analysis, in which effect sizes were nested within studies, using the ‘rma.mv’ function of the ‘metafor’ package (Viechtbauer, 2010), running in R (R Core Team, 2018). We examined the influence of individual studies and publication bias using leave-one-out analyses (i.e., excluding the largest and smallest effect sizes). We also conducted a Bayesian Multilevel Meta-analysis using the ‘brms’ package (Bürkner, 2017). We contrasted the estimated effect to a hypothesis of the effect size  $< 0$ , and report the Evidence Ratio (equivalent of a Bayes Factor, see Dienes, 2014) and Posterior Probability of the pooled effect  $< 0$ .

$I^2$  was used as the measure of heterogeneity, with values  $> 50\%$  indicative of moderate and  $> 75\%$  indicative of substantial

heterogeneity. For the initial meta-analysis, we included all identified effect sizes for the association of psychopathy with response inhibition task performance, and prioritised the inclusion of continuous effect sizes at the facet or factor level, followed by continuous effect sizes for the total score, and finally standard mean difference effect sizes converted to  $r$ . We also performed a series of moderator analyses on sample type (non-criminal versus criminal), gender, task type (go/no-go vs. stop signal), the inclusion of an affective component in the response inhibition task (e.g., the inclusion of affective and neutral images or words), and psychopathy trait dimension (interpersonal/affective vs. lifestyle/antisocial). Psychopathic traits were categorised according to the two-factor solution synonymous with the PCL-R (Hare, 2003) as moderator analyses based on a three-factor solution were not feasible based given the available data. The categorization of subscales as synonymous with Factor 1 and Factor 2 is documented in Supplemental Material 1, Table S1). Analysis scripts and data can be found on Open Science Framework [<https://osf.io/nqb4h/>].

### 3. Results

#### 3.1. Number of studies identified and included

Updated searches, completed on 6 January 2022, identified 133

papers, of which 67 were duplicates. After reviewing the remaining full texts, 13 papers were considered suitable for inclusion. A further eight papers were identified via cited articles, reference list checks, and email correspondence. This resulted in a total of 21 papers that met inclusion criteria for the review. One author read all cited articles and reference lists, and emailed authors/presenters where critical analyses were unreported. A PRISMA (Liberati et al., 2009; Moher et al., 2009) flowchart is provided in Fig. 1.

#### 3.2. Overview of study and participant characteristics

Participant demographics and study characteristics for eligible papers are presented in Table 1. All studies were published in peer-reviewed journals between 1995 and 2021. Ten studies used a correlational design, and the remaining 11 studies used a group-based design. All but two of the non-offender samples were recruited using convenience sampling methodology, with six studies recruiting from university student populations, three studies recruiting from local communities, and one study recruiting from patients presenting at an accident and emergency room. Forensic and offender samples were recruited from prisons, medium and high-secure services, correctional facilities, and criminal justice agencies (e.g., offender programmes, probation services, associations providing support to ex-prisoners).

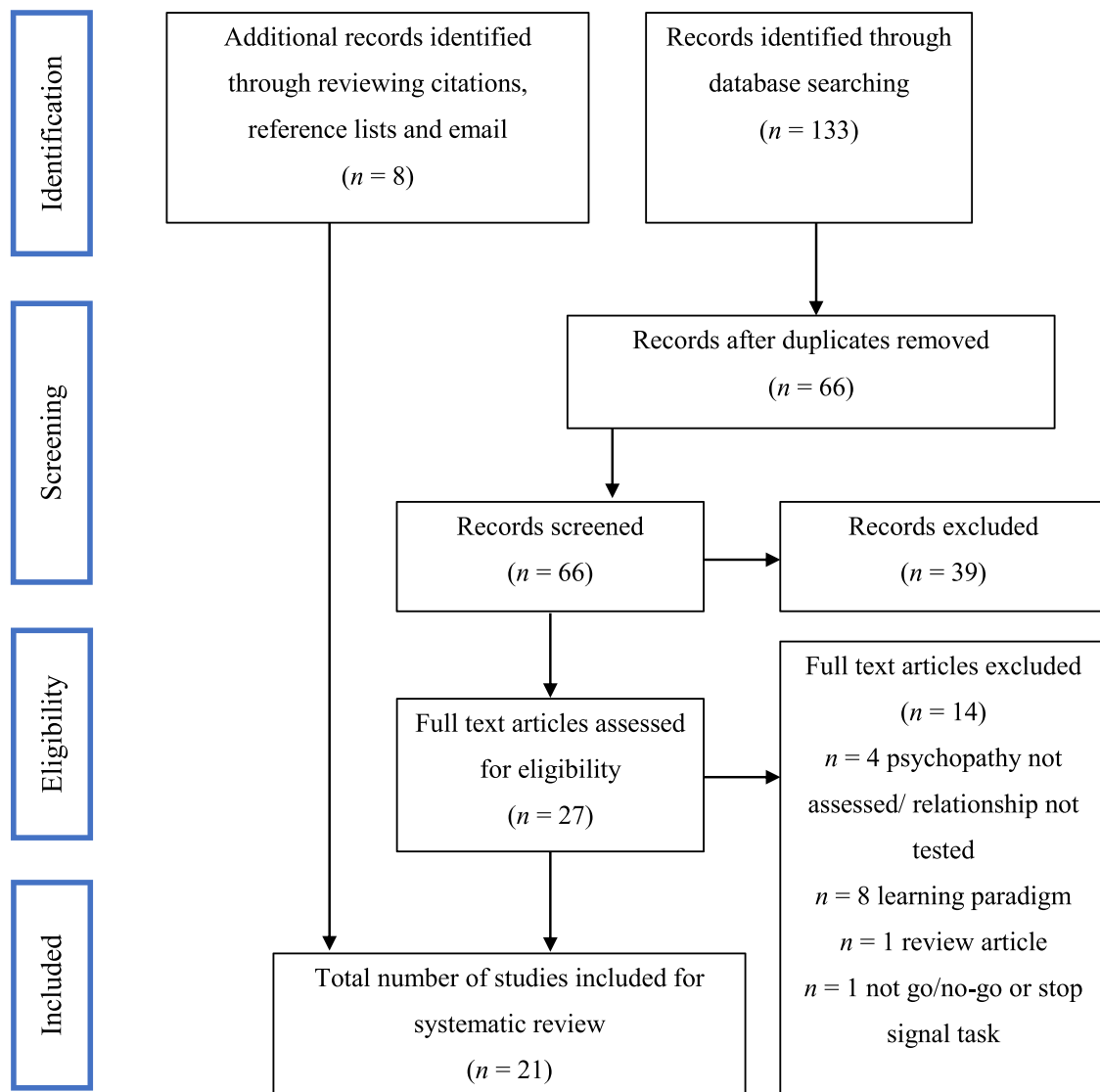


Fig. 1. PRISMA flowchart of the study selection process.

**Table 1**  
Characteristics of included studies.

Author (Year), location	Format of publication	Study design, sample	Sample size (N)	Gender (%), Mage, SD (years)	Ethnicity (%)	Psychopathy measure	RI task (No. of trials), ratio of Go/No-Go trials
Fournier et al. (2021), USA	Journal article	Correlational, community	Total N = 114	Females (50), males (48), transgender (2), Mage = 29.41, SD = 6.58	Caucasian (55.250) Black / African American (29.82) Asian (4.39) American Indian (3.51) Other (6.14)	SRPS-II	Emotional-linguistic Go/No-Go (576) Go:No-Go = 71.88:28.13
Heritage and Benning (2013), USA	Journal article	Correlational, Emergency room	Total N = 66	Male (44), Mage = 26, SD = 12	N/S	MPQ-BF	Stop-Signal (600) W/NW: 75/25
Iria and Barbosa (2009), Portugal	Journal article	Group-based, separate community and forensic comparisons	Total N = 62, high PCL:SV offenders (n = 22), low PCL:SV offenders (n = 11), high PCL:SV non-offenders (n = 16), low PCL:SV non-offenders (n = 13)	Male (100) High PCL:SV offenders Mage = 30.09, low PCL:SV offenders Mage = 27.36, high PCL:SV non-offenders Mage = 28.13, low PCL:SV non-offenders Mage = 28.31	Caucasian (100)	PCL:SV (Portuguese version)	Go/No-Go (56) Go:No-Go = 39:61
Iria et al. (2012), Portugal	Journal article	Group-based, separate community and forensic comparisons	Total N = 113, high PCL:SV Factor 1 offenders (n = 25), low PCL:SV Factor 1 offenders (n = 37), high PCL:SV Factor 1 non-offenders (n = 12), low PCL:SV Factor 1 non-offenders (n = 39)	Male (100), high PCL:SV Factor 1 offenders Mage = 40.76, low PCL:SV Factor 1 offenders Mage = 38.70, high PCL:SV Factor 1 non-offenders Mage = 36.75, low PCL:SV Factor 1 non-offenders Mage = 37.87	Caucasian (100)	PCL:SV (Portuguese version)	Go/No-Go (144) Go:No-Go = N/S
Kiehl et al. (2000) Canada	Journal article	Group-based, forensic	Total N = 36, low PCL-R schizophrenia (N = 12), high PCL-R offenders (n = 13), low PCL-R offenders (n = 11)	Male (100), low PCL-R schizophrenia Mage = 33.0, high PCL-R offenders Mage = 28.0, low PCL-R offenders Mage = 27.0	NA	PCL-R	Go/No-Go (540) Go:No-Go = 50:50
Kim and Jung (2014), South Korea	Journal article	Group-based, undergraduate	Total N = 30, high PPI-R (n = 15), low PPI-R (n = 15)	Male (33), high PPI-R Mage = 19.9, SD = 1.6, low PPI-R Mage = 20.5, SD = 1.9	N/S	PPI-R	Go/No-Go (240) Go:No-go = 66.67/33.4
Krakowski et al. (2015) USA	Journal article	Group-based, forensic	Total N = 38, high PCL:SV offenders (n = 16), low PCL:SV healthy controls (n = 22)	High PCL:SV offenders Male (94), Mage = 41.7, low PCL:SV healthy controls Male (77), Mage = 41.4	High PCL:SV African American (81.3), Low PCL:SV healthy controls African American (59.1)	PCL:SV	Go/No-Go (478) Go:No-Go = 85/15
Lapierre et al. (1995) Canada	Journal article	Group-based, forensic	Total N = 60, high PCL-R offenders (n = 30), low PCL-R offenders (n = 30)	Gender NA, Mage NA, age range 18–55	French-Canadian (100)	PCL-R	Go/No-Go Block A (50) Go:No-Go = 100/0 Block B (150) Go:No-Go = 50/50
Malesza and Ostaszewski (2016), Poland	Journal article	Correlational, undergraduate	Total N = 298	Male (46), Mage = 21.8, SD = 1.52	N/S	SRPS-III	Stop-Signal Go:Stop = 75:25
Maurer et al. (2016) USA	Journal article	Correlational, forensic	Total N = 121	Female (100), Mage = 33.94	Hispanic/Latino (55), White (34), Black/African American (6), American Indian (4), > than one ethnic category (1)	PCL-R	Go/No-Go (490) Go:No-Go = 84/16
Michalowski et al. (2015), Poland	Journal article	Group-based, undergraduate	Total N = 26, low IA (n = 12), high IA (n = 14)	Males (19), Mage = N/S	N/S	PPI (Polish version)	Stop-Signal (128) Go:Stop = 75:25 Go/No-go (128) Go:No-Go = 75:25
Morgan et al. (2011), UK	Journal article	Correlational, community	Total N = 80	Males (38), Mage = 21.16, SD = 2.42	N/S	PPI-R	Go/Stop (N/S)
Munro et al. (2007) Canada	Journal article	Correlational, forensic	Total N = 15	Male (100), Mage = 45.9	N/S	PCL-R	Go/No-Go (550) Go:No-Go = 67/34
			Total N = 50		N/S		

(continued on next page)

Table 1 (continued)

Author (Year), location	Format of publication	Study design, sample	Sample size (N)	Gender (%), <i>Mage</i> , <i>SD</i> (years)	Ethnicity (%)	Psychopathy measure	RI task (No. of trials), ratio of Go/No-Go trials
Paiva et al. (2020), Portugal	Journal article	Correlational, community	Total N = 142	Female (50) Male (50) <i>Mage</i> = 26.6; <i>SD</i> = 6.12	N/S	TriPM (Portuguese version)	Go/No-Go (500) Go:No-Go = 95:30
Ribes-Guardiola et al. (2020), Spain	Journal article	Correlational, undergraduate		Males (29), <i>Mage</i> = 20.58, <i>SD</i> = 4.69		TriPM (Spanish adapted version)	Go/No-Go (1200) Go:No-Go: 80:20
Sprague and Verona (2010), USA	Journal article	Group-based, undergraduate	Total N = 81 High IA ( <i>n</i> = 42), low IA ( <i>n</i> = 39)	Males (44), age range = 18–21, <i>Mage</i> = NS	Caucasian (64) Asian (20) Hispanic (7) Other (6) African American (3)	SRPS-II & PPI-Short form	Emotional-linguistic Go/No-Go (96) Go:No-Go = 68.75:31.25
Steele et al. (2016) USA	Journal article	Correlational, forensic	Total N = 104	Male (93), <i>Mage</i> = 34.53	White (46) Hispanic (44) American-Indian (20) Other (17) Black/African American (10) Asian (6)	PCL-R	Go/No-Go (490) Go:No/Go = 84/16
Varlamov et al. (2011) UK	Journal article	Group-based, forensic	Total N = 69, high PCL-R secure patients ( <i>n</i> = 27), low PCL-R secure patients ( <i>n</i> = 22), low PCL:SV healthy controls ( <i>n</i> = 20)	Male (100), high, PCL-R secure patients <i>Mage</i> = 31.55, low PCL-R secure patients <i>Mage</i> = 33.78, low PCL:SV healthy controls <i>Mage</i> = 32.55	NR	PCL-R and PCL:SV	Go/No-Go (195) Go:No-Go = 67:34
Verona et al. (2012) USA	Journal article	Group-based, forensic	Total N = 55, high PCL-R offenders with APD ( <i>n</i> = 14), low PCL-R offenders with APD ( <i>n</i> = 16), low PCL-R offenders without APD ( <i>n</i> = 15)	Male (74), high PCL-R offenders with APD <i>Mage</i> = 36, low PCL-R offenders with APD, <i>Mage</i> = 30.44, low PCL-R offenders without APD <i>Mage</i> = 30	High PCL-R offenders with APD: European American (57.1), African American (42.9), low PCL-R offenders with APD: European American (50), African American (43.8), Hispanic (6.3), low PCL-R offenders without APD: European American (53.3), African American (46.7)	PCL:SV	Go/No-Go (576) Go:No-Go = 72/28
Weidacker et al. (2017a), UK	Journal article	Correlational, forensic	Total N = 77	Male (100), <i>Mage</i> = 41.18	White British (90.9)	PCL:SV	Parametric Go/No-Go Stage 1 (150) Go:No-Go = 100/0 Stage 2 (180) Go:No-Go = 40/10 <sup>a</sup> Stage 3 (180) Go:No-Go = 40/10 <sup>a</sup>
Weidacker et al. (2017b), UK	Journal article	Correlational, undergraduate	Total N = 86	Males (35), <i>Mage</i> = 22.99, <i>SD</i> = 5.15	N/S	PPI-R	Parametric Go/No-Go Stage 1 (180) Go:No-Go = 25:75 Stage 2 (360) Go:No-Go = 20:5 Stage 3 (360) Go:No-Go = 20:5

*Note.* IA: Impulsive antisociality, MPQ-BF: Multi-dimensional Personality Questionnaire – Brief Version, W: word, NW: non-word, PT group: psychopathic trait group, C Group: control group, PPI-R: Psychopathic Personality Inventory – Revised, SRPS-III: Self Report Psychopathy Scale version 3, NS: Not stated, nCP: Non-criminal psychopath, nCnP: Non-criminal non-psychopath; Scz = Schizophrenia; APD = Antisocial Personality Disorder; CG = Control group; NR = information was not reported; PCL-R = Psychopathy Checklist Revised; PCL:SV = Psychopathy Checklist Screening Version; a there was no Go/No-Go rule applied to the remaining 50 % of trials.

Control participants in studies that included a non-forensic comparison group were recruited from prison staff (Munro et al., 2007), via local employment services (Iria and Barbosa, 2009), or via local advertisements (Iria et al., 2012; Varlamov et al., 2011). Two studies included samples of both offenders and non-offenders (Iria and Barbosa, 2009; Iria et al., 2012). Total sample sizes ranged from  $N = 15$ –298.

### 3.3. Measurement of psychopathic traits

The measures used to assess psychopathic traits varied between studies. Of those studies that recruited non-offending samples, five studies used the PPI-R (Lilienfeld and Widows, 2005), and its translated or short-form versions. Although the PPI-R is intended for use as a dimensional measure, some studies employed arbitrary cut-off scores to identify high and low scoring groups (Kim and Jung, 2014; Michalowski et al., 2015), but the precise approach used to identify these groups varied between studies. Michalowski et al. (2015) specifically focused on the impulsive-antisociality subscale of the PPI-R and used upper and

lower interquartile ranges to identify high and low scorers. Sprague and Verona (2010) used composite scores based on the antisocial subscales of the PPI Short Form (PPI-S; Lilienfeld and Andrews, 1996) and the SRP-II (Hare, 1991), and two measures of borderline personality features, to identify participants with an emotionally dysregulated profile, characterised by combined high antisocial and borderline features. Two studies (Iria and Barbosa, 2009; Iria et al., 2012) used the Portuguese translation of the Psychopathy Checklist: Screening Version (PCL:SV; Hart et al., 1995), but employed different (arbitrary) cut-off scores to determine high and low scoring groups. Ribes-Guardiola et al. (2020) and Paiva et al. (2020) used the TriPM (Patrick et al., 2009), while Fournier et al. (2021) and Malesza and Ostaszewski (2016) used the SRP-III (Paulhus et al., 2009). Heritage and Benning (2013) used the Multi-dimensional Personality Questionnaire-Brief Version (Patrick et al., 2002) to estimate fearless dominance and impulsive antisociality scores. Studies that recruited from criminal and forensic samples all used clinician rating instruments for the assessment of psychopathic traits, with five studies using the PCL-R (Hare, 2003), five studies using the

**Table 2**  
Results of quality assessment.

Author (Year)	Introduction			Methods							
	1	2	3	4	5	6	7	8	9	10	11
Fournier et al. (2021)	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Heritage and Benning (2013)	Yes	Yes	No	Yes	Yes	Yes	Partial	Yes	Yes	Yes	Yes
Iria and Barbosa (2009)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Iria et al. (2012)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Kiehl et al. (2000)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Kim and Jung (2014)	Yes	Yes	No	Yes	Yes	Partial	No	Yes	Yes	Partial	Yes
Krakowski et al. (2015)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Lapierre et al. (1995)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Malesza and Ostaszewski (2016)	Yes	Yes	No	Yes	Yes	Partial	No	Yes	Yes	Yes	Yes
Maurer et al. (2016)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Michalowski et al. (2015)	Yes	Yes	No	Yes	Yes	Partial	No	Yes	Yes	Partial	Yes
Morgan et al. (2011)	Yes	Yes	No	Yes	Yes	Partial	No	Yes	Yes	Partial	Yes
Munro et al. (2007)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Paiva et al. (2020)	Yes	Yes	No	Yes	Yes	Partial	Partial	Yes	Yes	Yes	Yes
Ribes-Guardiola et al. (2020)	Yes	Yes	No	Yes	Yes	Partial	Partial	Yes	Partial	Yes	Yes
Sprague and Verona (2010)	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Steele et al. (2016)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Varlamov et al. (2011)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Verona et al. (2012)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Weidacker et al. (2017a)	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Weidacker et al. (2017b)	Yes	Yes	No	Yes	Yes	Partial	No	Yes	Yes	Yes	Yes
Author (Year)	Results		13	14	15	16	Discussion		Other		
Author (Year)	12		13	14	15	16	17	18		19	20
Fournier et al. (2021)	Yes	No		Yes	Yes	Yes	Yes	Yes		Yes	Partial
Heritage and Benning (2013)	Yes	No	No	No	Yes	Yes	Yes	Yes		NS	NS
Iria and Barbosa (2009)	Yes	No	No	No	Yes	Yes	Yes	No		NS	NS
Iria et al. (2012)	Yes	No	No	No	Partial	Yes	Yes	Yes		NS	Yes
Kiehl et al. (2000)	Yes	No	No	No	NC	Yes	Yes	Yes		Yes	NS
Kim and Jung (2014)	Yes	No	No	No	Partial	Yes	Yes	No		Yes	NS
Krakowski et al. (2015)	Yes	No	No	No	Yes	Yes	Yes	Yes		Yes	Yes
Lapierre et al. (1995)	Yes	No	No	No	Yes	Yes	Yes	Yes		Yes	Yes
Malesza and Ostaszewski (2016)	Yes	No	No	No	Partial	Yes	Yes	Yes		Yes	NS
Maurer et al. (2016)	Yes	No	No	No	Yes	Yes	Yes	Yes		Yes	Yes
Michalowski et al. (2015)	Yes	No	No	No	Yes	Yes	Yes	No		Yes	NS
Morgan et al. (2011)	Yes	No	No	No	NC	Yes	Yes	Yes		Yes	NS
Munro et al. (2007)	Yes	No	No	No	Partial	Yes	Yes	Yes		NS	Yes
Paiva et al. (2020)	Yes	No	No	No	Partial	Yes	Yes	Yes		Yes	NS
Ribes-Guardiola et al. (2020)	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes		No	NS
Sprague and Verona (2010)	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes		Yes	NS
Steele et al. (2016)	Yes	No	No	No	Yes	Yes	Yes	Yes		Yes	Yes
Varlamov et al. (2011)	Yes	No	No	No	NC	Yes	Yes	Partial		NS	Yes
Verona et al. (2012)	Yes	No	No	No	Yes	Yes	Yes	Yes		NS	NS
Weidacker et al. (2017a)	Yes	No	No	No	Yes	Yes	Yes	Yes		NS	Yes
Weidacker et al. (2017b)	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes		NS	Yes

Note: 1 = Clear aims & objectives; 2 = Appropriate study design; 3 = Sample size justification; 4 = Population clearly defined; 5 = Appropriate sample frame; 6 = Representative selection process; 7 = Categorisation of non-responders; 8 = Variables appropriate to the aim; 9 = Validated measures used; 10 = Clear reporting of statistical significance; 11 = Methods described for replication; 12 = Descriptive statistics reported; 13 = Concern for non-response bias; 14 Detail of any non-responding; 15 = Internal consistency of results; 16 = Analysis as described in method; 17 = Justification of discussion and conclusion; 18 = Limitations discussed; 19 = Any funding or conflict of interest; 20 = Ethical approval or consent obtained; NC = Not clear; NS = Not stated; Partial = some of the required information is available.

PCL:SV (Hart et al., 1995), and one study using both the PCL-R and the PCL:SV.

### 3.4. Assessment of response inhibition

Sixteen studies used a variation of the standard go/no-go task to measure response inhibition, two studies used a stop signal task, and one study employed both the go/no-go and stop signal tasks. One study used a task that involved the presentation of a stop signal after a trial had started, but the variable of interest was the proportion of correctly inhibited stop trials, rather than the Stop Signal Reaction Time (Morgan et al., 2011). Stimuli used in response inhibition tasks differed depending on the overarching aims of the studies, with a variety of go, no-go or stop cues employed, including words, shapes, letters, numbers, and auditory tones.

### 3.5. Risk of bias within studies

A summary of the quality assessment of all papers included in the review is displayed in Table 2. All of the studies clearly reported their aims and objectives, had an appropriate study design, clearly defined their population of choice, used validated measures, selected an appropriate sampling frame and variables appropriate to the study aims, had a replicable methods section and accurate reporting of descriptive statistics, reported results from all analyses reported to have been undertaken (although because these studies were not pre-registered, any changes to the analysis pipeline following data collection cannot be ruled out), and reported conclusions that were justified based on the results. Two studies were of particularly high quality (Fournier et al., 2021; Sprague and Verona, 2010) and showed little risk of bias.

Except for Paiva et al. (2020), all the studies failed to report a power analysis, and some studies reported relatively sample sizes, including Munro et al. (2007) ( $N = 15$ ) and Michałowski et al. (2015) ( $N = 26$ ). Twelve studies declared a conflict of interest and/or funding, eight studies failed to include a conflict-of-interest statement, and one paper (Ribes-Guardiola et al., 2020) clearly stated no conflict of interest or funding awarded.

Although most studies detailed limitations of their research, three papers (Iria and Barbosa, 2009; Kim and Jung, 2014; Michałowski et al., 2015) failed to discuss any methodological issues that might have affected the results. Overall, the findings reported by Michałowski et al. (2015) should be interpreted with a relatively higher degree of caution; clear statistical significance was only partially reported, with  $p$ -values for non-significant tests omitted, and details about methodological limitations, ethical approval and informed consent, and conflicts of interest or sources of funding (if any) were unreported.

### 3.6. Quantitative synthesis of results (meta-analyses)

Table 3 summarises the results, including effect sizes, for each individual study. The multilevel random-effects meta-analysis contained 43 effect sizes from 17 studies (total  $N = 1394$ ). Data were unavailable for four studies, which were excluded from the quantitative synthesis (Kim and Jung, 2014; Sprague and Verona, 2010; Weidacker et al., 2017a,b). The pooled effect was statistically significant and showed a small effect size ( $r = -0.143$  [95 % CI:  $-0.250$  to  $-0.034$ ],  $Z = 2.56$ ,  $p = .011$ ; see Fig. 2). There was substantial heterogeneity ( $I^2 = 76.2$  %). Removal of the smallest ( $r = -0.159$  [95 % CI:  $-0.265$  to  $-0.052$ ],  $p = .004$ ,  $I^2 = 75.0$  %), and largest ( $r = -0.101$  [95 % CI:  $-0.174$  to  $-0.028$ ],  $p = .006$ ,  $I^2 = 50.0$  %), effect sizes did not substantially influence the pattern of results. The evidence ratio for the pooled effect  $r < 0$  was 84.11, and the posterior probability = 99%. This can be interpreted as substantial evidence of the alternative hypothesis (vs the null).

Based on the pooled effect size, the median statistical power to detect this effect across all studies was approximately 19.6 % (min 7.9 % – max

69 %): see Fig. 3 for sunset plot. To detect a correlation of  $r = -0.143$ , 301 participants would be needed for 80 % power and 415 participants for 90 % power (one-tailed).

### 3.7. Moderator analyses

#### 3.7.1. Sampling: Non-criminal versus criminal

The subgroup effect comparing associations in non-criminal versus criminal samples was not statistically significant ( $X^2(1) = 0.555$ ,  $p = .456$ ). The association in non-criminal samples was  $r = -0.135$  [95 % CI:  $-0.191$  to  $-0.079$ ],  $Z = 4.68$ ,  $p < .001$ ,  $I^2 = 18.3$  %, and the association in criminal samples was  $r = -0.137$  [95 % CI:  $-0.337$  to  $0.074$ ],  $Z = 1.27$ ,  $p = .203$ ,  $I^2 = 86.1$  %.

#### 3.7.2. Gender: Male only vs. mixed/female only

The subgroup effect comparing male only samples versus mixed/female only samples was not statistically significant ( $X^2(1) = 1.104$ ,  $p = .293$ ). The association in males only was  $r = -0.150$  [95 % CI:  $-0.290$  to  $-0.004$ ],  $Z = 2.02$ ,  $p = .044$ ,  $I^2 = 52.8$  %, and the association in mixed/female only samples was  $r = -0.071$  [95 % CI:  $-0.148$  to  $0.006$ ],  $Z = 1.80$ ,  $p = .071$ ,  $I^2 = 45.4$  %.

#### 3.7.3. Task type: Go/no-go vs. stop signal

Because the study by Morgan et al. (2011) included a go/stop task, where the outcome variable of interest was the proportion of correct responses, we excluded this study from the moderator analysis. The subgroup effect comparing go/no-go versus stop signal tasks was not significant ( $X^2(1) = 0.732$ ,  $p = .392$ ). The association in go/no-go tasks was  $r = -0.140$  [95 % CI:  $-0.274$  to  $-0.001$ ],  $Z = 1.967$ ,  $p = .049$ ,  $I^2 = 80.6$  %, and in stop signal tasks was  $r = -0.211$  [95 % CI:  $-0.427$  to  $0.028$ ],  $Z = 1.736$ ,  $p = .083$ ,  $I^2 = 77.1$  %.

#### 3.7.4. Task stimuli: Affective component vs. no affective component

The subgroup effect comparing response inhibition tasks with an affective component versus not was not statistically significant ( $X^2(1) = 0.022$ ,  $p = .880$ ). The association in tasks with an affective component was  $r = -0.133$  [95 % CI:  $-0.289$  to  $0.022$ ],  $Z = 1.69$ ,  $p = .092$ ,  $I^2 = 59.6$  %, and in tasks with no affective component was  $r = -0.151$  [95 % CI:  $-0.298$  to  $0.002$ ],  $Z = 1.93$ ,  $p = .054$ ,  $I^2 = 83.6$  %.

#### 3.7.5. Psychopathy dimensions: Interpersonal/affective vs. lifestyle/antisocial

Relationships for different psychopathy trait dimensions were reported separately in nine studies, which yielded a total of 25 individual effect sizes (14 x interpersonal/affective, 11 x lifestyle/antisocial). The subgroup effect comparing interpersonal/affective and lifestyle/antisocial was not statistically significant ( $X^2(1) = 0.480$ ,  $p = .489$ ). The association for interpersonal/affective traits was  $r = -0.036$  [95 % CI:  $-0.128$  to  $0.056$ ],  $Z = 0.77$ ,  $p = .439$ ,  $I^2 = 51.0$  %), and the association for lifestyle/antisocial traits was  $r = -0.059$  [95 % CI:  $-0.151$  to  $0.035$ ],  $Z = 1.23$ ,  $p = .218$ ,  $I^2 = 46.7$  %).

#### 3.7.6. Method of assessment: Self-report vs. clinician rating scale

Moderator analyses on method of assessment could not be performed. Using the available data, psychopathic traits were assessed using clinician rating scales in all studies that included forensic samples. The population sample (non-criminal vs. criminal) would therefore have confounded any comparison between self-report and clinician-rating scale methods of assessment.

## 4. Discussion

In this study, we conducted a systematic review and meta-analysis of the relationship between psychopathic traits and response inhibition in non-criminal and criminal samples. A total of 21 papers were identified for inclusion that were published in peer-reviewed journals between

**Table 3**

Summary information of methods and results for included studies.

Author (Year)	N included in analyses	Stimuli	Variables of interest	Psychopathy variables	M (SD)/r, p
Fournier et al. (2021)	114	Affective and neutral words	Go/No-Go commission errors	SRP-III Facet 1: IPM SRP-III Facet 2: CA SRP-III Facet 3: ELS SRP-III Facet 4: ASB	$r = -0.03, p > .05$ $r = 0.16, p > .05$ $r = -0.04, p > .05$ $r = 0.21, p < .05$
Heritage and Benning (2013)	66	Affective and neutral words	SSRT	MPQ-BF estimated FD MPQ-BF estimated IA	$r = -0.09, p > .05$ $r = 0.32, p < .05$
Iria and Barbosa (2009)	62	Affective images	Go/No-Go commission errors	High PCL:SV offenders Low PCL:SV offenders High PCL:SV non-offenders Low PCL:SV non-offenders	$M = 4.64, SD = 4.51$ $M = 4.91, SD = 3.96$ $M = 4.06, SD = 4.85$ $M = 2.23, SD = 2.42$
Iria et al. (2012)	113	Affective images	Go/No-Go commission errors	High PCL:SV F1 offenders    Low PCL:SV F1 offenders    High PCL:SV F1 non-offenders    Low PCL:SV F1 non-offenders	Anger: $M = 9.06, SD = 3.81$ Fear: $M = 11.92, SD = 5.61$ Sad: $M = 8.30, SD = 4.14$ Anger: $M = 5.87, SD = 3.78$ Fear: $M = 11.18, SD = 5.56$ Sad: $M = 6.89, SD = 4.17$ Anger: $M = 6.86, SD = 3.93$ Fear: $M = 11.39, SD = 5.78$ Sad: $M = 7.33, SD = 5.30$ Anger: $M = 3.72, SD = 3.83$ Fear: $M = 8.09, SD = 5.63$ Sad: $M = 5.44, SD = 3.62$
Kiehl et al. (2000)	36	Neutral shapes	Go/No-Go commission errors	Low PCL-R schizophrenia patients High PCL-R offenders Low PCL-R offenders	$M = 9.01, SD = 5.8$ $M = 5.60, SD = 5.5$ $M = 4.26, SD = 3.1$
Kim and Jung (2014)	30	Shapes	No-Go Accuracy	High PPI-R Low PPI-R	$M = 91.0$ $M = 90.7$
Krakowski et al. (2015)	38	Affective and neutral images	Go/No-Go commission errors	High PCL:SV offenders Low PCL:SV offenders	N/R N/R
Lapierre et al. (1995)	60	Neutral shapes	Go/No-Go commission errors	High PCL-R offenders Low PCL-R offenders	$M = 16.47, SD = 8.32$ $M = 3.30, SD = 3.85$
Malesza and Ostaszewski (2016)	298	Neutral words and letters	SSRT	SRP-III total score	$r = 0.16, p < .05$
Maurer et al. (2016)	121	Neutral shapes	Go/No-Go commission errors	PCL-R Facet 1 PCL-R Facet 2 PCL-R Facet 3 PCL-R Facet 4	$r = -0.10, p > .05$ $r = -0.02, p > .05$ $r = -0.08, p > .05$ $r = -0.15, p > .05$
Michalowski et al. (2015)	26	Letters	Go/No-Go commission errors  SSRT	High PPI-R IA Low PPI-R IA High PPI-R IA Low PPI-R IA	$M = 18.5, SD = 12.5$ $M = 15.1, SD = 13.0$ $M = 327.0, SD = 58.9$ $M = 280.5, SD = 44.5$
Morgan et al. (2011)	80	Numbers	Go/Stop commission errors	PPI-R S-CI PPI-R FD, PPI-R CH	$r = -0.02, p > .05$ $r = -0.12, p > .05$ $r = -0.02, p > .05$
Munro et al. (2007)	15	Neutral letters	Go/No-Go commission errors	Total PCL-R	$r = -0.46, p = .13$
Paiva et al. (2020)	41	Letters	Go/No-Go commission errors	TriPM Boldness TriPM Meanness TriPM Disinhibition	$r = -0.09, p < .05$ $r = 0.11, p < .05$ $r = -0.12, p < .05$
Ribes-Guardiola et al. (2020)	142	Letters	Go/No-Go commission errors	TriPM Boldness TriPM Meanness TriPM Disinhibition	$r = -0.14, p > .05$ $r = -0.21, p < .05$ $r = -0.12, p > .05$
Sprague and Verona (2010)	81	Neutral, negative, and diagnostic specific negative words	Go/No-Go commission errors	High composite BA scale Low composite BA scale	N/R N/R
Steele et al. (2016)	93	Neutral letters	Go/No-Go commission errors	PCL-R Facet 1 PCL-R Facet 2 PCL-R Facet 3 PCL-R Facet 4	$r = 0.12, p > .05$ $r = 0.1, p > .05$ $r = -0.1, p > .05$ $r = -0.1, p > .05$
Varlamov et al. (2011)	69	Neutral shapes	Go/No-Go commission errors		$M = 13.96, SD = 11.5$

(continued on next page)

Table 3 (continued)

Author (Year)	N included in analyses	Stimuli	Variables of interest	Psychopathy variables	M (SD)/r, p
				High PCL-R secure patients	
				Low PCL-R secure patients	M = 9.09, SD = 7.75
				Low PCL:SV healthy controls	M = 7.5, SD = 5.24
Verona et al. (2012)	45	Affective words	Go/No-Go commission errors	PCL:SV Factor 1	r = -0.13, p > .05
Weidacker et al. (2017a)	77	Neutral letters	Go/No-Go commission errors	PCL:SV Factor 2	r = -0.11, p > .05
				PCL:SV Facet 1	N/R
				PCL:SV Facet 2	N/R
				PCL:SV Facet 3	N/R
Weidacker et al. (2017b)	86	Letters	Go/No-Go percentage correctly inhibited trials	PPI-R Fearlessness	N/R
				PPI-R Impulsive	N/R
				Nonconformity	N/R
				PPI-R Coldheartedness	N/R
				PPI-R Carefree	N/R
				Nonplanfulness	N/R
				PPI-R Machiavelian	N/R
				Egocentricity	N/R
				PPI-R Social Potency	N/R
				PPI-R Blame	N/R
				Externalization	

Note. MPQ-BF = Multidimensional Personality Questionnaire – Brief Form; SSRP-III = Self Report Psychopathy Scale-III; PPI-R = Psychopathic Personality Inventory-Revised; PCL:SV = Psychopathy Check-List Screening Version; SSRT = Stop Signal Reaction Time; IA = Impulsive Antisociality; FD = Fearless Dominance; F = Fearlessness; IN = Impulsive Nonconformity; C = Coldheartedness; CN = Carefree Nonplanfulness; ME = Machiavellian Egocentricity; SP = Social Potency; BE = Blame Externalization; SI = Stress Immunity, S-CI = Self-Centred Impulsivity, AD = Antisocial Deviance, BA = Borderline-Antisocial, IPM = Pathological Lying and Manipulation; CA = Callous-affect; ELS = Erratic-lifestyle; ASB = Antisocial Behaviour; NS = Not Stated.

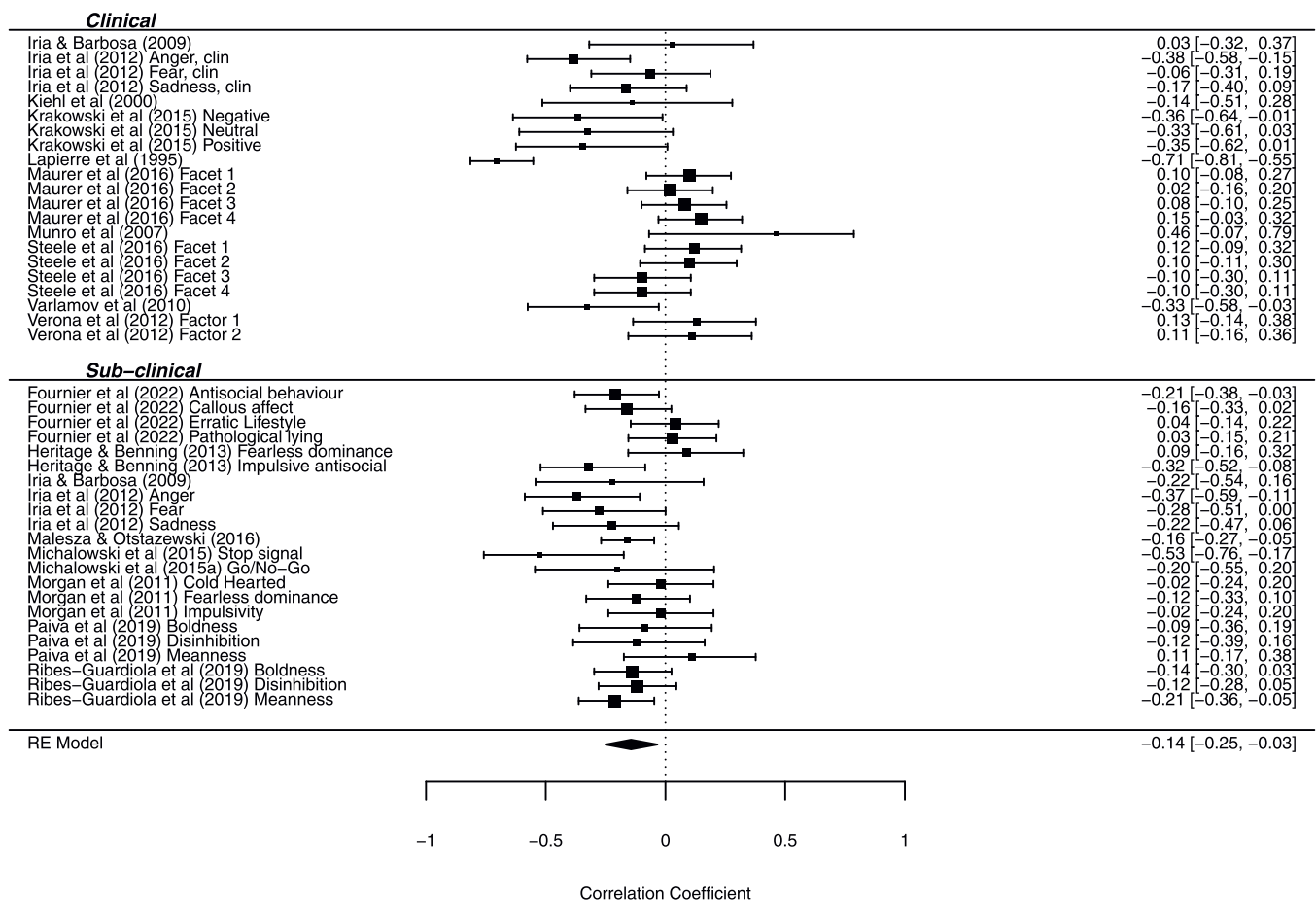


Fig. 2. Forest plot for the association between psychopathic traits and response inhibition.

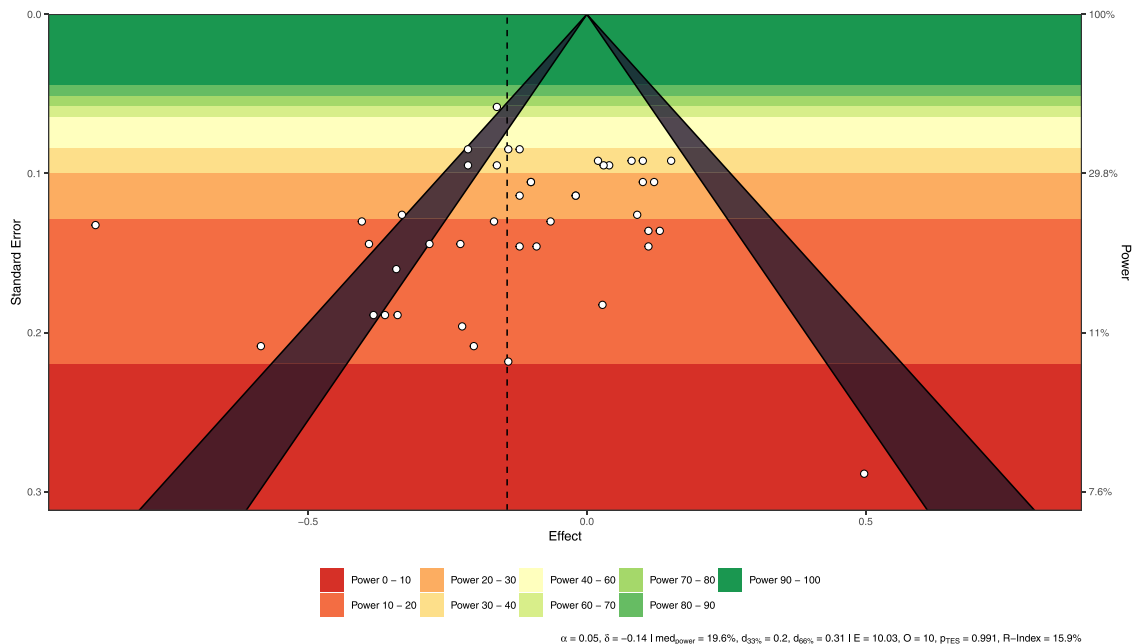


Fig. 3. Sunset plot showing effect sizes plotted against their standard error and statistical power.

2009 and 2021 (total  $N = 1668$ ), with 43 available effect sizes from 17 different studies included in a multilevel, random-effects meta-analysis (total  $N = 1394$ ). The analysis showed that the association of psychopathic traits with response inhibition, assessed using either the go/no-go or stop signal tasks, was significant, with increasing psychopathic traits associated with poorer response inhibition task performance. The association was robust to the removal of individual effect sizes. Despite evidence for considerable heterogeneity ( $I^2 = 75.8\%$ ), moderator analyses showed that the effects of sample type (non-criminal vs. criminal), gender (male vs. female/mixed), task type (go/no-go vs. stop signal), presence of an affective component (affective words, pictures), and psychopathy trait dimension (interpersonal/affective vs. lifestyle/antisocial), were all non-significant. Overall, we report only a relatively modest association of psychopathy with difficulties in response inhibition, which supports the argument of Poythress and Hall (2011) that these difficulties may not represent a cardinal feature of the psychopathic personality.

Although impulsivity is a commonly recognised risk factor for violence and criminality more generally, we found no differences in the size of the relationship between psychopathy and response inhibition in criminal and non-criminal samples. These findings have some, indirect relevance for various models of so called 'successful' psychopathy, outlined by Lilienfeld et al. (2015). It is suggested by each of these models that the presence of impulsive tendencies or other factors that might protect against criminality allows some individuals to either avoid criminality or avoid detection and subsequent arrest and conviction for their criminal behaviours. Although our meta-analysis does not represent a direct test of these models, it is informative about the strength of the relationship between psychopathy and response inhibition in these different population samples. It would also be worthwhile to examine the associations of non-criminal and criminal psychopathic traits with other factors that might protect against criminality, including intelligence, exceptional talent, educational opportunity, socioeconomic status, highly effective socialization, or independent aspects of temperament (Benning et al., 2018). Importantly, our results do not support or refute any arguments about the role of response inhibition in the decision to act in criminal or antisocial ways.

We also showed that the pooled effect size was similar for both interpersonal/ affective and lifestyle/ antisocial psychopathic traits. However, while the general construct of psychopathy, across factors and

facets, was found to be associated with impaired response inhibition, the individual trait dimensions both showed non-significant associations with response inhibition. This finding may appear somewhat surprising, especially given that the lifestyle/antisocial trait dimension indexes features including irresponsibility, impulsivity, and recklessness, compared to features of narcissism, lack of empathy, and a conning and manipulative interpersonal style indexed by the interpersonal/ affective dimension.

Theoretically, it is proposed in the dual-process model that the two trait dimensions are underpinned by distinct aetiological processes (Fowles and Dindo, 2009), with the interpersonal/ affective features attributable to low fear reactivity, and lifestyle/ antisocial features attributable to impairments in emotional and behavioural control. However, developmental trajectories resulting from either process can produce similar phenotypic traits, and risk-factors for different trait dimensions are not considered to be mutually exclusive, but instead may combine in complex ways (Fowles and Dindo, 2009; Patrick et al., 2009). Ultimately, our results suggest that impairments in response inhibition are a feature of the general construct of psychopathic personality and are not driven by either trait dimension in isolation. It is also noteworthy that the effect sizes reported here do no account for suppressor effects between the two trait dimensions, and the use of partialling or residualized scores may yield different results (Hicks and Patrick, 2006; but also see Lynam et al., 2006).

Methodological arguments might also account for the lack of association with specific trait dimensions. First, it has been shown that self-report measures of impulsive responding and other near neighbour constructs seldom correlate with cognitive task-based measures of the constructs they claim to assess (Strickland and Johnson, 2021). Second, the test of moderation for trait dimensions may have been limited by statistical power, where we were limited to including only those studies that reported effect sizes for specific trait dimensions.

Our review had several strengths, including a comprehensive search strategy across four databases, duplicating the selection of papers for inclusion (100% of all papers at title and abstract stage and 10 % at full text stage) and data extraction, and employing a quality assessment tool. However, there are nonetheless some limitations. First, based on the pooled effect size reported here, we calculated that a sample size of between 301 and 415 participants would be needed to detect a correlation  $r$  of  $-0.143$  with 80–90 % statistical power. We also calculated

that the average statistical power to detect this effect across all studies included in the quantitative synthesis was low, approximately 19.6% (min 7.9 % – max 69 %). Although the pooling of smaller studies to increase statistical power is a considerable strength of the meta-analytic approach (Cohn and Becker, 2003), the individual findings reviewed here should be interpreted with caution. Our review highlights the need for more robust, highly powered research looking at the effects of psychopathic tendencies on response inhibition.

We also limited our review to published research only, and estimates suggest that this may inflate effect sizes (McAuley et al., 2000). However, there are concerns that unpublished literature has poorer reporting quality (Adams et al., 2017). It was also notable that only three studies that met inclusion criteria for this review included the stop signal task, and that none of these studies employed the stop signal task in criminal/forensic samples. Future research should therefore consider using both the go/no-go task, and the stop signal task, to measure action restraint and action cancellation processes in forensic samples. There was considerable heterogeneity in the measures used to assess psychopathic tendencies (and the use of different factor structures between measures), the use of arbitrary cut-offs to delineate high and low scoring groups, and the reporting of facet level analyses. Studies that recruited from forensic populations used clinician rating scales for the assessment of psychopathic tendencies (e.g., the PCL-R or the PCL:SV), meaning that a potential moderator analysis based on measurement type (i.e., self-report versus clinician rating scale) would be confounded by criminal history. Similarly, there was considerable variability in the design of the response inhibition tasks, although moderator analyses showed no effect of either task type or the inclusion of affective stimuli.

Future research should seek to understand the associations of individual psychopathy trait dimensions with discrete components of response inhibition, including action restraint and action cancellation. The inclusion of other techniques, including measures of physiological and autonomic nervous system activity, would be constructive in helping to resolve conflicting findings in this area from a multisystem approach (Hagan et al., 2020). For example, our findings cannot altogether rule out the possibility that different pathophysiological mechanisms underly impulsive responding in non-criminal and criminal psychopathy. The findings of one review of brain imaging studies concluded that severe impulsivity in psychopathy may arise from a pathophysiological mechanism that is unique to severely elevated psychopathic traits that is not reflected in the general population (Korponay and Koenigs, 2021). A programme of work using a multisystem approach would therefore help to elucidate the pathophysiological underpinnings of non-criminal versus criminal psychopathy, and associations of response inhibition with individual psychopathy trait dimensions.

Our findings also have implications for understanding the association of psychopathic traits with other processes that share neurobiological underpinnings with response inhibition. For example, offender profiles characterized by difficulties in emotion regulation are associated with higher affective and lifestyle psychopathic traits (Garofalo et al., 2018), while structural equation models show relationships of affective and lifestyle psychopathic traits with difficulties in emotion regulation, and interpersonal psychopathic traits with better emotion regulation (Garofalo et al., 2019). Importantly, the ability to regulate negative affective states is dependent on the functional architecture of prefrontal regions that overlap with those underlying response inhibition abilities, including the anterior insula cortex and the dorsomedial frontal cortex (Etkin et al., 2015; Morawetz et al., 2020). Our findings may therefore suggest that functional impairments in these circuits may account not only for psychopathy related impairments in response inhibition, but also difficulties in emotion regulation. Future research should explore these possibilities. Again, a multi-system approach that includes measures of heart rate variability, a biological marker of emotion regulation (Gillespie et al., 2018, 2012), may be most helpful.

#### 4.1. Conclusions

Our systematic review and meta-analysis showed that both non-criminal and criminal psychopathic tendencies, across both interpersonal/affective and lifestyle/antisocial features of the disorder, are associated with impaired performance on tests of response inhibition. We found no evidence that this effect was moderated by sample type, gender, task type, presence of an affective component to the task, or psychopathy trait dimension. Our findings suggest that the association of psychopathy with response inhibition may be driven by the overall psychopathy construct, rather than being dependent on a particular psychopathy trait dimension, and that the size of the effect is relatively small, which calls in to question the extent to which impulse control problems, and specifically response inhibition, should be considered a cardinal feature of the psychopathic personality. Additional research using a multisystem approach is needed to better understand the pathophysiology of non-criminal versus criminal psychopathy, and the extent to which functional impairments in the neural circuitry underpinning response inhibition may also account for other problems, including difficulties in emotion regulation.

#### Data availability

Analysis scripts and data can be found on Open Science Framework [<https://osf.io/nqb4h/>].

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#### Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.neubiorev.2022.104868](https://doi.org/10.1016/j.neubiorev.2022.104868).

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