

# Impulsivity, aggression, and suicidality relationship in adults: A systematic review and meta-analysis

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## Summary

**Background** The aim of the study was to crystallize the nature of relationships between impulsivity, aggression, and suicidality. We reviewed studies of adults with published, psychometric measures of impulsivity and aggression, and measures of suicidality.

**Methods** Our primary data source was Web of Science (from inception to 31st December 2021). Quality of articles was assessed using a Joanna Briggs Appraisal Tool and publication bias using Trim and Fill. We synthesised results using random effects meta-analyses and explored moderation by measure of impulsivity, aggression, and suicidality, and population.

**Findings** 77 studies were included in our analysis. We found weak positive relationships between impulsivity ( $r = 0.19$  [95% CI: 0.15–0.22]), aggression (0.23 ([0.17, 0.29])), and impulsive aggression (0.16 [0.1, 0.22]) with suicidality. Heterogeneity was significant and only partially explained by moderators. Limitations included the lack of studies which assess impulsivity or aggression proximal to suicidality.

**Interpretation** Given small effect sizes and significant heterogeneity, the study suggests that additional studies are needed in the field to analyze the relation between impulsivity and aggression with suicidality.

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**Keywords:** Impulsivity; Aggression; Impulsive aggression; Suicide; Meta-analysis

## Introduction

Despite a large body of research identifying population-level risk factors we remain poor at identifying individuals at risk of suicide.<sup>1–9</sup> Counting risk factors alone performs no better than chance,<sup>6</sup> actuarial risk assessment tools are wrong 95% of the time,<sup>7,8</sup> and clinicians report low confidence in risk assessment.<sup>9</sup> This may reflect limited validity of the theoretical models which underpin risk assessment. Here we focus on two key constructs widely believed to be related to suicidality and included in multiple theoretical models and risk assessment tools, but whose relationships are characterized

by inconsistency in the empirical literature: impulsivity and aggression.

Impulsivity is a broad construct that has multiple operationalizations.<sup>10,11</sup> Definitions include risk-taking, sensation-seeking, behavioral disinhibition, preference for small immediate rewards over large distal rewards, deficits in planning, and urgency.<sup>10</sup> It is included in three leading theoretical models of suicide.<sup>12</sup> In Beck et al.'s Cognitive Model,<sup>13,14</sup> impulsivity is a dispositional trait which increases suicidality. In Baumeister's Escape Theory,<sup>15</sup> suicidality increases when individuals can no longer resist impulsive urges to remove themselves from distress via increased behavioral disinhibition. In the Integrated Motivational Volitional Model,<sup>16,17</sup> impulsivity acts as a volitional moderator between suicidal ideation and action.

Unsurprisingly, given the lack of theoretical consensus, empirical tests of associations yield conflicting results. From their meta-analysis of associations

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### Research in context

#### Evidence before this study

Web of Science, EMBASE, MEDLINE, CINAHL, PubMed, Cochrane Library, PsychInfo, and PsychArticles were searched on 31st Dec 2021 using the following keywords: ("impuls\*" OR "disinhib\*" OR "inhib\*" OR "risk taking" OR "risk-taking" OR "behav\* control" OR "adventuresomeness" OR "sensation seeking" OR "sensation-seeking" OR "novelty seeking" OR "novelty-seeking" OR "urgency" OR "premeditation" OR "perseverance" OR "response inhib\*" OR "distractor interference" OR "proactive interference" OR "delay\* response" OR "delay\* discount" OR "distortions in elapsed time" OR "inattention") AND ("aggress\*" OR "ang\*" OR "hostil\*" OR "irritabil\*" OR "violen\*") AND ("suicid\*"). Impulsivity and aggression are included as predictors in multiple psychological models of suicide. Empirical work, however, has shown inconsistent directions and strengths of relationships. Existing reviews and meta-analyses suggest that this may be due to the diversity of operationalisations of impulsivity, aggression, and suicidality.

#### Added value of this study

In this meta-analysis of 77 studies, we found impulsivity, aggression, and impulsive aggression to be weak predictors of suicidality. Heterogeneity was significant and only partially explained by moderators. Impulsive aggression was a stronger predictor of suicidality in non-clinical populations, and for measures of suicidal ideation. Impulsivity and aggression were stronger predictors of suicide risk than other measures of suicidality. Given small effect sizes and significant heterogeneity, we suggest that additional studies are needed before conflation of impulsivity and aggression with suicidality, and argue for the ongoing development of nuanced, person-centered, suicide risk formulation and management.

#### Implications of all the available evidence

Our findings confirm the lack of consistency in relationships between impulsivity and aggression with suicidality. This work will inform development of future studies on clinical suicide risk assessment.

between trait impulsivity and suicidal behavior, Anestis et al.<sup>10</sup> concluded that the association is weak at best and that impulsivity acts indirectly via exposure to painful experiences. Conversely, Gvion and Apter<sup>11</sup> concluded from a systematic review that the relationship is well established across different types of impulsivity, and clinical and non-clinical populations (although they point to likely differences in relation to suicidality between trait and state impulsivity, which we discuss later in this section). Vast diversity in the definitions of both suicidality and impulsivity are likely to contribute to such discrepancies.<sup>11,18,19</sup> A recent meta-analysis, for

example, found cognitive impulsivity to be a stronger predictor of suicidality than behavioral impulsivity.<sup>20,21</sup>

More recently, models of impulsivity and suicide have incorporated a role of aggression or *impulsive aggression*.<sup>11,22</sup> Aggression, too, has multiple definitions. Broadly, it is any behavior intended to harm another person.<sup>11</sup> Aggression, irritability, hostility, and anger, however, are often used interchangeably. Specifically, anger encompasses annoyance, hostility, and displeasure<sup>23</sup>; irritability is sensitivity to provocation<sup>24</sup>; and hostility is cynicism, mistrust, and denigration.<sup>25</sup> Aggression can also be conceptualized as reactive (a response to perceived threat that is impulsive and emotionally charged) or proactive (premeditated and controlled).<sup>11</sup> As with impulsivity, while there is evidence that aggression correlates with suicidality, the nature of the relationship is unclear.<sup>11,26</sup>

Impulsivity and aggression may also form part of a larger psychopathology.<sup>27–29</sup> Brent and Mann<sup>30,31</sup> argue that impulsivity, hostility, and aggression, are part of a disinhibitory psychopathology operationalized as impulsive aggression. Aggression appears in Wenzel and Beck's<sup>14</sup> update of Beck et al.'s Cognitive Model<sup>13</sup> in the same role as impulsivity (a dispositional trait which increases vulnerability to suicidality). Impulsive aggression also plays a role in Brent and Mann's Clinical-Biological Model<sup>31</sup> and in Plutchik, van Praag, and Conte's Two Stage Model.<sup>32</sup> In the former, impulsive aggression is a familial trait which mediates between psychopathology and suicidality. In the latter, aggressive impulses are triggered by stress, and the likelihood of them being expressed against the self increases when coupled with psychiatric symptoms.

That impulsivity and aggression are conceptualised and measured as both states and traits may further contribute to complexity.<sup>11</sup> In the models above, they are viewed as dispositional traits,<sup>10,13,14,30</sup> state responses to adversity,<sup>21,32</sup> or a combination of both.<sup>15,16</sup> This distinction is relevant as trait and state measures of impulsivity correlate weakly with one another<sup>33–37</sup> and differ in their relation to self-harm.<sup>38</sup> Furthermore, Liu et al.<sup>20</sup> found time between a suicide attempt and the assessment of impulsivity moderated the relationship (i.e. the relationship was strongest when there was less than one month between a suicide attempt and assessment of impulsivity).

The aim of the current synthesis was to further our understanding of the nuanced pathways by which impulsivity and aggression influence suicidality in order to inform the development of accurate suicide risk assessment. To achieve this, we sought to answer the following research questions: (1) to what extent do impulsivity, aggression, and impulsive aggression, predict suicidality?, and (2) are these relationships moderated by (a) measure of impulsivity, aggression, or impulsive aggression, (b) measure of suicidality, or (c) population?

## Methods

### Search strategy and eligibility criteria

Eligibility criteria, search strategy, data collection, and analytic strategy were registered as a review protocol to PROSPERO (CRD42020160631). The review was conducted in accordance with PRISMA (2020) guidelines.<sup>39</sup>

We included only those studies which met the following criteria: (1) use of peer reviewed and published, psychometric measures of impulsivity and aggression (i.e. not pre-prints or unvalidated measures of impulsivity or aggression); (2) use of measures of suicidality (i.e. we excluded studies which assessed non-suicidal self-harm as, although this may develop into suicidality, they can have distinct aetiologies and functions<sup>40–42</sup>); (3) all participants were adults (i.e. 16 years and over); (4) were empirical, original, assessments of the relationships of interest (i.e. not reviews or case studies). We did not specify a start date for publications in our search and did not exclude publications on the basis of geographic location or research design. Where English language versions of articles were not available, we contacted authors to request English versions. If these were unavailable, the study was excluded from the sample. FM conducted the search and screened articles, and HM independently screened and evaluated a subset of 10% of articles.

The search terms were agreed by the study authors based on expertise in psychological predictors of suicidality and a scoping review of operationalizations of impulsivity, suicide, and aggression. The search string was: (“impuls\*” OR “disinhib\*” OR “inhib\*” OR “risk taking” OR “risk-taking” OR “behav\* control” OR “adventuresomeness” OR “sensation seeking” OR “sensation-seeking” OR “novelty seeking” OR “novelty-seeking” OR “urgency” OR “premeditation” OR “perseverance” OR “response inhib\*” OR “distractor interference” OR “proactive interference” OR “delay\* response” OR “delay\* discount\*” OR “distortions in elapsed time” OR “inattention”) AND (“aggress\*” OR “ang\*” OR “hostil\*” OR “irritabil\*” OR “violen\*”) AND (“suicid\*”). The search was conducted most recently on 31/12/21 on Web of Science using the above search terms with no limits or filters. We ran confirmatory searches using EMBASE, MEDLINE, CINAHL, PubMed, Cochrane Library, PsychInfo, and PsychArticles, which revealed no additional items.

### Data extraction

Each article which met eligibility criteria was screened against the Joanna Briggs Checklist for Analytical Cross-Sectional Studies by FM, with a subsample of 10% screened by HM.<sup>43</sup> Here, each study was screened against 8 quality criteria including assessment of validity and reliability of measurements, descriptions of

inclusion criteria and subjects, appropriateness of statistical analysis, and identification and management of relevant confounding variables. FM extracted effect sizes, or statistical information required to calculate effect sizes, for associations between impulsivity and aggression with suicidality. In all cases, the quality and appropriateness of statistical analyses was rated as acceptable for extraction of effect sizes (e.g. assumption of linearity had been checked where appropriate). HM independently extracted data from a subset of 10% of articles, yielding no discrepancies. We recorded the following study characteristics to be tested as moderators in analyses: population (clinical or non-clinical), and measures of impulsivity, aggression, and suicidality. Where some effect sizes were reported and others were missing ( $n = 5$  studies), we followed the Cochrane Handbook for Systematic Reviews of Interventions.<sup>44</sup> In all instances, missing data were for outcomes that failed to reach statistical significance. In four<sup>45–48</sup> the proportion of missing values was relatively small (all < 39%). For one<sup>49</sup> we considered the proportion of missing values to be too high (73%) and excluded the study.

### Data analysis

We used Pearson’s Correlation Coefficient ( $r$ ) as our measure of effect size since various research designs were included in our sample (e.g. comparison of impulsivity in groups with and without a history of suicide attempt, or cross-sectional tests of relationships between suicidal ideation and impulsivity). We adhered to Cohen’s<sup>50</sup> classification of small ( $r = 0.1$ ), medium ( $r = 0.3$ ), and large ( $r = 0.5$ ) effect sizes. Where  $r$  was not reported, we calculated it from statistical information available in the article (or made available by the article’s authors).

We used Hedge’s random effects models to incorporate subject and sampling error. We first assessed the pooled weighted effect sizes for associations between all measures of impulsivity, aggression, and impulsive aggression, across samples. As there were insufficient numbers of studies which assessed potential covariates, we did not include these in any models. As there were multiple results for each study, the unit of analysis was average study effect size. Heterogeneity was assessed using  $I^2$ , Cochran’s  $Q$ , and confidence intervals.

Population was coded as clinical (in- and out-patients, and deaths by suicide), non-clinical (community), or both.

We grouped suicidality outcome measures as: history of suicide attempt, current or previous suicidal ideation, number of lifetime suicide attempts, age at first suicide attempt, suicide risk, lethality of suicide attempt (s), and cause of death (see Supplementary Material 3 for a description of the measures of each of these outcomes).

We categorised impulsivity in two ways. We treated self-report measures as 'trait' and neuropsychological measures as 'state'.<sup>20,21</sup> An exception was the Impulsivity Rating Scale<sup>51</sup> which assesses self-reported impulsive behaviors in the past week and was treated as 'state'. Second, we grouped outcomes by broad domains of impulsivity. Recent reviews suggest that impulsivity can be meaningfully categorized as either cognitive or behavioral.<sup>20</sup> Cognitive impulsivity is the inability to weigh the consequences of proximal and distal events in order to delay gratification, and behavioral impulsivity is deficits in response inhibition.<sup>52,53</sup> The two facets are weakly correlated and underpinned by distinct neural pathways.<sup>52</sup> See Supplementary Material 4 for a description of the measures of each of these categories.

Measures of aggression were grouped as either state (aggression in the past week) or trait (aggression across the lifespan), and broad domain of aggression. Domains were general aggression, anger, hostility, irritability, physical aggression, verbal aggression, and premeditated aggression. Finally, we included a category for impulsive aggression. See Supplementary Material 5 for a description of the measures of each of these categories.

Each moderator was tested separately using meta-regression. Here, all results were included for each study to allow us to detect moderation by measures of impulsivity, aggression, and suicidality for which there were frequently multiple measures per study. While this approach has traditionally been advised against due to non-independence of multiple results from the same study, modern methods of meta-regression are robust to this independence.<sup>54</sup> To correct for repeated sampling and reduce risk of Type I error we used the Hartung-Knapp correction.<sup>54</sup> In all cases we carried out sensitivity analyses by running the moderation analyses using one effect size per sample. As deviations were minimal ( $< 0.01$  per effect size), we were confident that the Hartung-Knapp correction had minimised the issue of dependence. Where a level of a moderator was reported in less than three studies we excluded those studies from moderation analyses.

Publication bias was visualised using funnel plots and assessed using Duval and Tweedie's Trim and Fill method. All analyses were conducted with Comprehensive Meta-Analysis Version 3.<sup>55</sup>

### Role of the funding source

The study was not supported by a funding source. All authors had access to the dataset and agreed to submission for publication.

### Results

The search returned 10,348 items. FM screened titles and removed duplicates and studies outside the area of

interest, reviews or theoretical reports, case studies, or those that stated inclusion of participants under 16 ( $n = 9622$ ). FM screened the abstracts of the remaining 726 articles in accordance with eligibility criteria. Forward and backward searching of reference lists of all eligible studies identified one additional item. Full articles were screened against the Joanna Briggs Checklist for Analytical Cross-Sectional Studies.<sup>43</sup> As our eligibility criteria closely matched the Checklist criteria, there was no instance where a study met our inclusion criteria but scored below 100% on relevant items (i.e. 8 out of 8 if identification and management of confounding variables was appropriate, and 6 out of 6 if not) on the Checklist. Given the lack of variance in scores, we did not include this measure of quality as a covariate in analyses. HM independently evaluated a sub-set of 10% of the articles, yielding concordance of 98% (i.e. two discrepancies which were discussed and agreed). Our decision to check concordance in a subset of the articles was based on the large number of articles to be screened, and the high rate of concordance across the 10% sample, from which we concluded further testing would be unlikely to improve the validity of the sample. Seventy-seven studies from 75 samples were included in the review. See Figure 1 for a summary of the selection process.

PRISMA flow diagram showing the process by which articles were screened and selected.

Seventy-seven publications reporting 445 results from 75 samples were included in analyses. Table 1 summarises sample characteristics. For references and study characteristics for all articles included in analyses, see Supplementary Material 2. For full data included in analyses, including effect sizes, see linked data file.

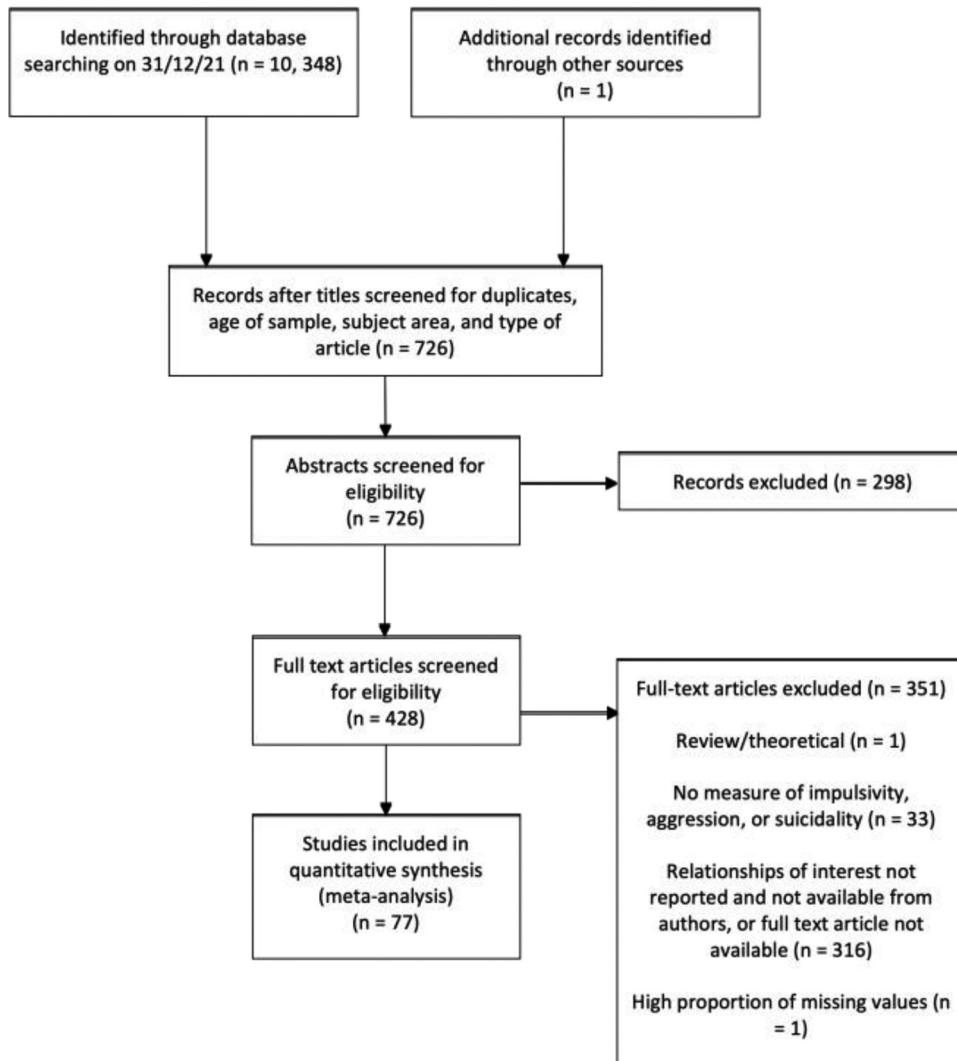
Supplementary Material 3–5 describe measures of suicidality, impulsivity, and aggression reported across articles, and the number of studies employing each measure.

### Impulsivity, aggression, and suicidality

The mean pooled effect size across all 75 samples was small, positive, and significant ( $r = 0.21$  [95% CI: 0.17–0.25],  $z = 9.57$ ,  $p < 0.001$ ). There was significant heterogeneity ( $I^2 = 96.96$ ,  $Q(74) = 2432.15$ ,  $p < 0.001$ ). Duval and Tweedie's Trim and Fill method estimated 8 studies to be missing to the right of the mean. With these imputed, the effect size increased to 0.24 (95% CI: 0.2, 0.28). For all funnel plots see Supplementary Material 6. Across all 501 results, psychological construct (impulsivity, aggression, impulsive aggression) did not moderate the relationship with suicidality ( $F(2498) = 1.74$ ,  $p = 0.1765$ ,  $R^2$  analog = 0.09).

### Impulsivity and suicidality

The mean pooled effect size for relationships between impulsivity and suicidality ( $n = 71$  samples) was small,



**Figure 1.** Screening and selection of articles.

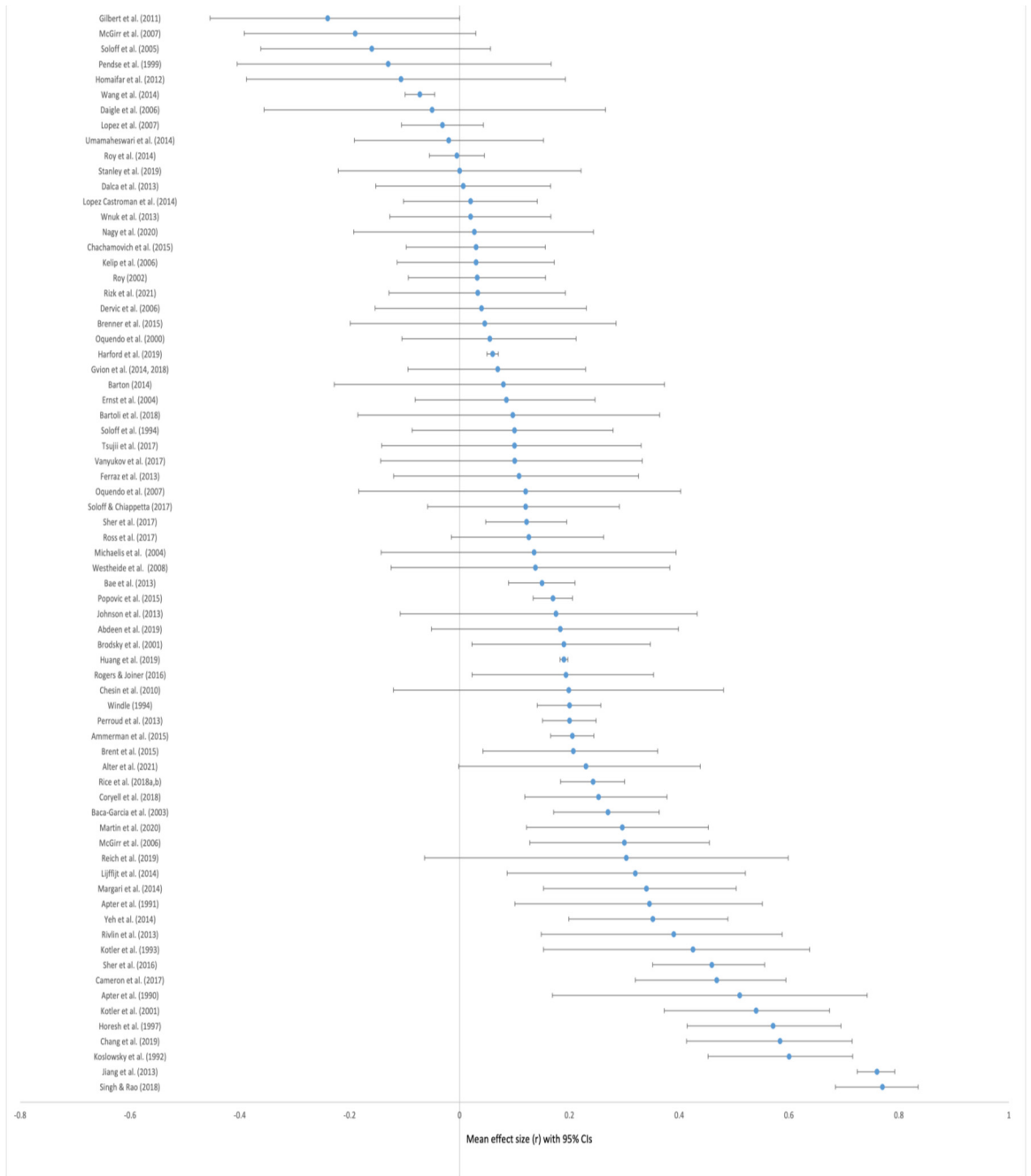
positive, and significant ( $r = 0.19$  [95% CI: 0.15–0.22],  $z = 9.91$ ,  $p < 0.001$ ; [Figure 2](#)). There was significant heterogeneity ( $I^2 = 95.37$ ,  $Q(70) = 1512.14$ ,  $p < 0.001$ ). Duval and Tweedie’s Trim and Fill estimated four studies to be missing from the right of the mean. When

imputed, the effect size decreased to 0.14 (95% CI: 0.11, 0.19).

Points represent effect sizes for each study, and bars indicate the 95% CI. Exact effect sizes and 95% CI are provided in the dataset.

Publication Year	Region	Clinical/non-clinical	Sex	Age
1990–1999 $n = 8$	Africa $n = 2$	Both $n = 15$	Female biased $n = 34$	Range = 16–81
2000–2009 $n = 16$	Asia $n = 13$	Clinical $n = 54$	Male biased $n = 24$	
2010–2019 $n = 49$	Australasia $n = 2$	Non-clinical $n = 8$	Both $n = 19$	
2020–2021 $n = 4$	Europe $n = 12$			
	North America $n = 47$			
	Multicenter $n = 1$			

**Table 1: Characteristics of the 77 studies included in analyses.**



**Figure 2.** Forest plot showing mean and 95% CI of effect sizes for impulsivity in relation to suicidality.

Measure of impulsivity (state or trait) moderated the relationship ( $F(1,199) = 9.31, p = 0.0026, \text{Tau}^2 = 0.04, I^2 = 96.9\%, R^2 \text{ analog} < 0.01$ ). The relationship between suicidality and (a) state impulsivity was small, negative, and non-significant ( $r = -0.05$  [95% CI:  $-0.19, 0.1$ ],  $p = 0.519, n = 15$ ) and (b) trait impulsivity was small, positive, and significant ( $r = 0.17$  [95% CI:  $0.14, 0.2$ ],  $p < 0.0001, n = 184$ ). For Figures showing

significant moderation effects, please see Supplementary Material 7.

Type of impulsivity (cognitive, behavioral, or both) moderated the relationship ( $F(2,197) = 9.58, p = 0.0001, \text{Tau}^2 = 0.02, I^2 = 93.3\%, R^2 \text{ analog} = 0.49$ ). The relationship between suicidality and (a) behavioral impulsivity was small, positive, and significant ( $r = 0.23$  [95% CI:  $0.19, 0.27$ ],  $p < 0.0001, n = 54$ ), (b) cognitive



impulsivity was small, positive, and non-significant ( $r = 0.01$  [95% CI:  $-0.04, 0.06$ ],  $p = 0.63$ ,  $n = 43$ ), and (c) both types of impulsivity was small, positive, and significant ( $r = 0.17$  [95% CI:  $0.14, 0.21$ ],  $p < 0.0001$ ,  $n = 102$ ). For Figures showing significant moderation effects, please see Supplementary Material 7.

Measure of suicidality moderated the relationship ( $F(6193) = 4.39$ ,  $p = 0.0003$ ,  $\text{Tau}^2 = 0.05$ ,  $I^2 = 96.69\%$ ,  $R^2 \text{ Analog} < 0.01$ ). The relationship between impulsivity and (a) suicidal ideation was small, positive, and significant ( $r = 0.1$  [95% CI:  $0.03, 0.17$ ],  $p = 0.005$ ,  $n = 38$ ), (b) lethality was small, positive, and significant ( $r = 0.08$  [95% CI:  $0.01, 0.15$ ],  $p = 0.03$ ,  $n = 19$ ), (c) number of suicide attempts was small, positive, and significant ( $r = 0.13$  [95% CI:  $0.07, 0.2$ ],  $p < 0.0001$ ,  $n = 14$ ), (d) suicide risk was medium, positive, and significant ( $r = 0.35$  [95% CI:  $0.26, 0.43$ ],  $p < 0.0001$ ,  $n = 23$ ), (e) cause of death was small, positive, and non-significant ( $r = 0.06$  [95% CI:  $<0.0001, 0.13$ ],  $p = 0.051$ ,  $n = 11$ ), and (f) history of suicide attempts was small, positive, and significant ( $r = 0.18$  [95% CI:  $0.12, 0.23$ ],  $p < 0.0001$ ,  $n = 92$ ). Age at first suicide attempt was excluded from this analysis due to sample size ( $n = 2$ ). Population (clinical, non-clinical, or both) did not moderate the relationship (all  $p > 0.05$ ). For Figures showing significant moderation effects, please see Supplementary Material 7.

### Aggression and suicidality

The mean pooled effect size ( $r$ ) for the relationship between aggression and suicidality across 65 samples was small, positive, and significant ( $0.23$  [95% CI:  $0.17, 0.29$ ],  $Z(62) = 7.17$ ,  $p < 0.0001$ ; Figure 3). There was significant heterogeneity ( $I^2 = 98.65$ ,  $Q(62) = 4581.53$ ,  $p < 0.0001$ ). Duval and Tweedie's Trim and Fill method estimated 26 studies to be missing from the right of the mean. When imputed, the effect size increased to  $0.39$  (95% CI:  $0.33, 0.45$ ).

Points represent effect sizes for each study, and bars indicate the 95% CI. Exact effect sizes and 95% CI are provided in the dataset.

Measure of suicidality moderated the relationship across 211 results ( $F(7204) = 8.16$ ,  $p < 0.0001$ ,  $\text{Tau}^2 = 0.04$ ,  $I^2 = 97.15\%$ ,  $\text{Analog } R^2 = 0.15$ ). The relationship between aggression and (a) age at first suicide attempt was small, negative, and significant ( $r = -0.1$  [95% CI:  $-0.13, 0.07$ ],  $p < 0.0001$ ,  $n = 3$ ), (b) suicidal ideation was small, positive, and significant ( $r = 0.24$  [95% CI:  $0.16, 0.31$ ],  $p < 0.0001$ ,  $n = 38$ ), (c) lethality was small, positive, and significant ( $r = 0.1$  [95% CI:  $0.06, 0.15$ ],  $p < 0.0001$ ,  $n = 23$ ), (d) number of suicide attempts was small, positive, and non-significant ( $r = 0.04$  [95% CI:  $-0.01, 0.09$ ],  $p = 0.033$ ,  $n = 18$ ), (e) suicide risk was medium, positive, and significant ( $r = 0.46$  [95% CI:  $0.36, 0.56$ ],  $p < 0.0001$ ,  $n = 17$ ), (f) cause of death was small, positive, and significant ( $r = 0.16$  [95% CI:  $0.06, 0.26$ ],  $p = 0.002$ ,  $n = 12$ ), and

(g) history of suicide attempts was small, positive, and significant ( $r = 0.21$  [95% CI:  $0.17, 0.25$ ],  $p < 0.0001$ ,  $n = 100$ ). For figures showing significant moderation, please see Supplementary Material 7.

Population, and state or trait aggression did not moderate the relationship between aggression and suicidality (all  $p > 0.1$ ).

### Impulsive aggression and suicidality

The mean pooled effect size ( $r$ ) for the relationship between impulsive aggression and suicidality across 28 samples was small, positive, and significant ( $0.16$  [95% CI:  $0.1, 0.22$ ],  $Z(27) = 4.96$ ,  $p < 0.0001$ ; Figure 4). There was significant heterogeneity ( $I^2 = 84.38$ ,  $Q(27) = 166.41$ ,  $p < 0.001$ ). Duval and Tweedie's Trim and Fill method estimated one study to be missing from the left of the mean. When imputed, the mean effect size dropped to  $0.15$  (95% CI:  $0.09, 0.21$ ).

Points represent effect sizes for each study, and bars indicate the 95% CI. Exact effect sizes and 95% CI are provided in the dataset.

The relationship was moderated by population across 89 results ( $F(3,87) = 3.84$ ,  $p = 0.0252$ ,  $\text{Tau}^2 = 0.01$ ,  $I^2 = 81.85\%$ ,  $R^2 \text{ analog} = 0.18$ ). The relationship between impulsive aggression and suicidality was (a) small, positive, and significant across studies with samples drawn from clinical populations ( $r = 0.18$  [95% CI:  $0.13, 0.23$ ],  $p < 0.001$ ,  $n = 63$ ); (b) small, positive, and significant in those studies whose samples were drawn from non-clinical populations ( $r = 0.27$  [95% CI:  $0.2, 0.34$ ],  $p < 0.001$ ,  $n = 6$ ), and (c) small, positive and significant in those studies whose samples were drawn from both ( $r = 0.09$  [95% CI:  $0.07, 0.11$ ],  $p < 0.0001$ ,  $n = 21$ ). For figures showing significant moderation, please see Supplementary Material 7.

Measure of suicidality moderated the relationship ( $F(5,81) = 7.33$ ,  $p < 0.0001$ ,  $\text{Tau}^2 = 0.01$ ,  $I^2 = 81.43\%$ ,  $\text{Analog } R^2 = 0.24$ ). Suicide risk was excluded due to small sample size ( $n = 2$ ). The relationship between impulsive aggression and (a) suicidal ideation was small, significant, and positive ( $r = 0.27$  [0.17, 0.37],  $p < 0.0001$ ,  $n = 16$ ), (b) lethality was small, non-significant, and negative ( $r = -0.02$  [ $-0.08, 0.04$ ],  $p = 0.472$ ,  $n = 7$ ), (c) number of suicide attempts weak, significant, and positive ( $r = 0.18$  [0.05, 0.15],  $p < 0.0001$ ,  $n = 15$ ), (d) cause of death was weak, significant, and positive ( $r = 0.17$  [0.1, 0.24],  $p < 0.0001$ ,  $n = 8$ ), and (e) history of suicide attempts was weak, significant, and positive ( $r = 0.18$  [0.14, 0.21],  $p < 0.0001$ ,  $n = 40$ ). For figures showing significant moderation, please see Supplementary Material 7.

### Discussion

In the largest meta-analysis to date, we found small, significant, positive relationships between impulsivity, aggression, and impulsive aggression, with suicidality.

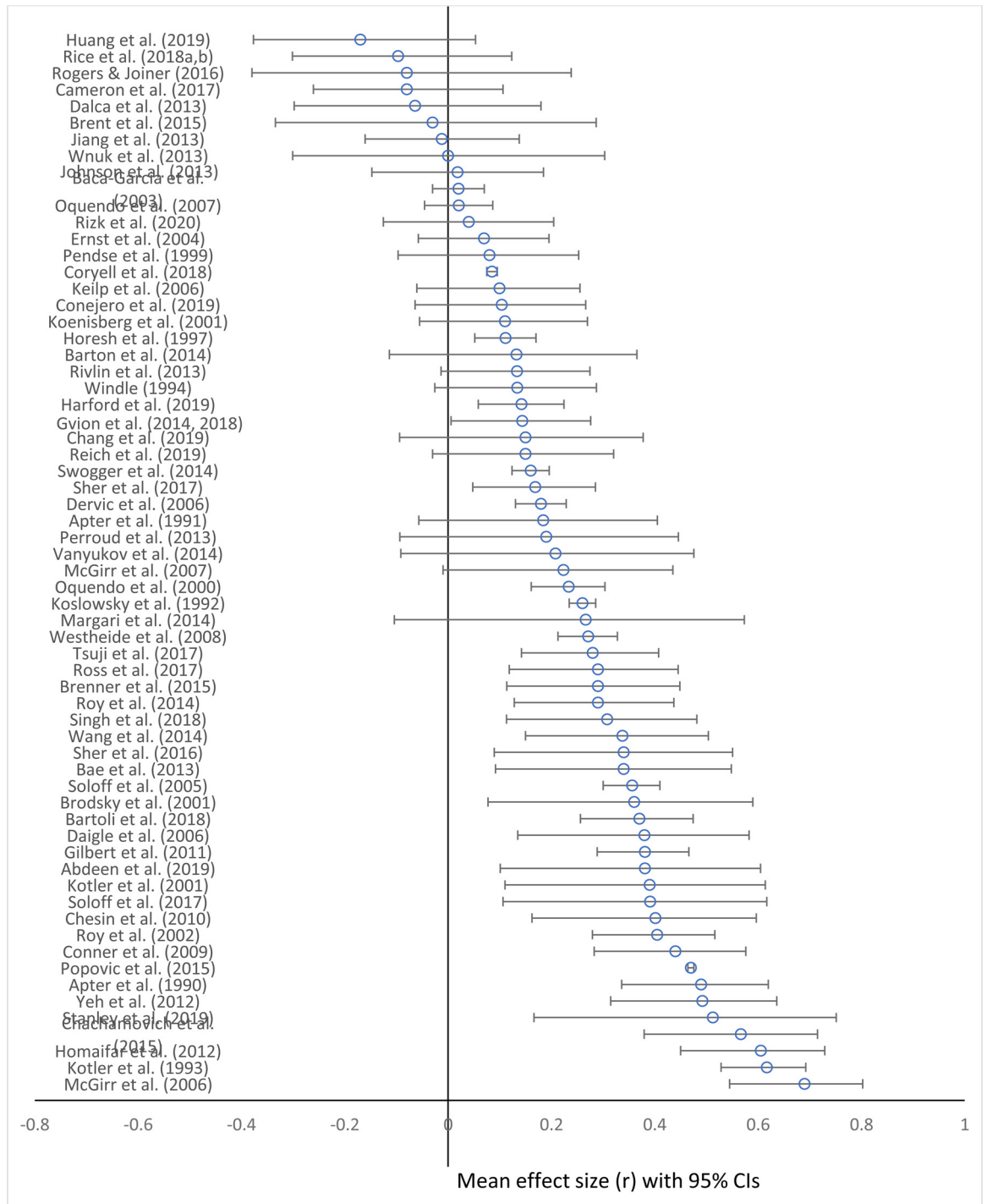


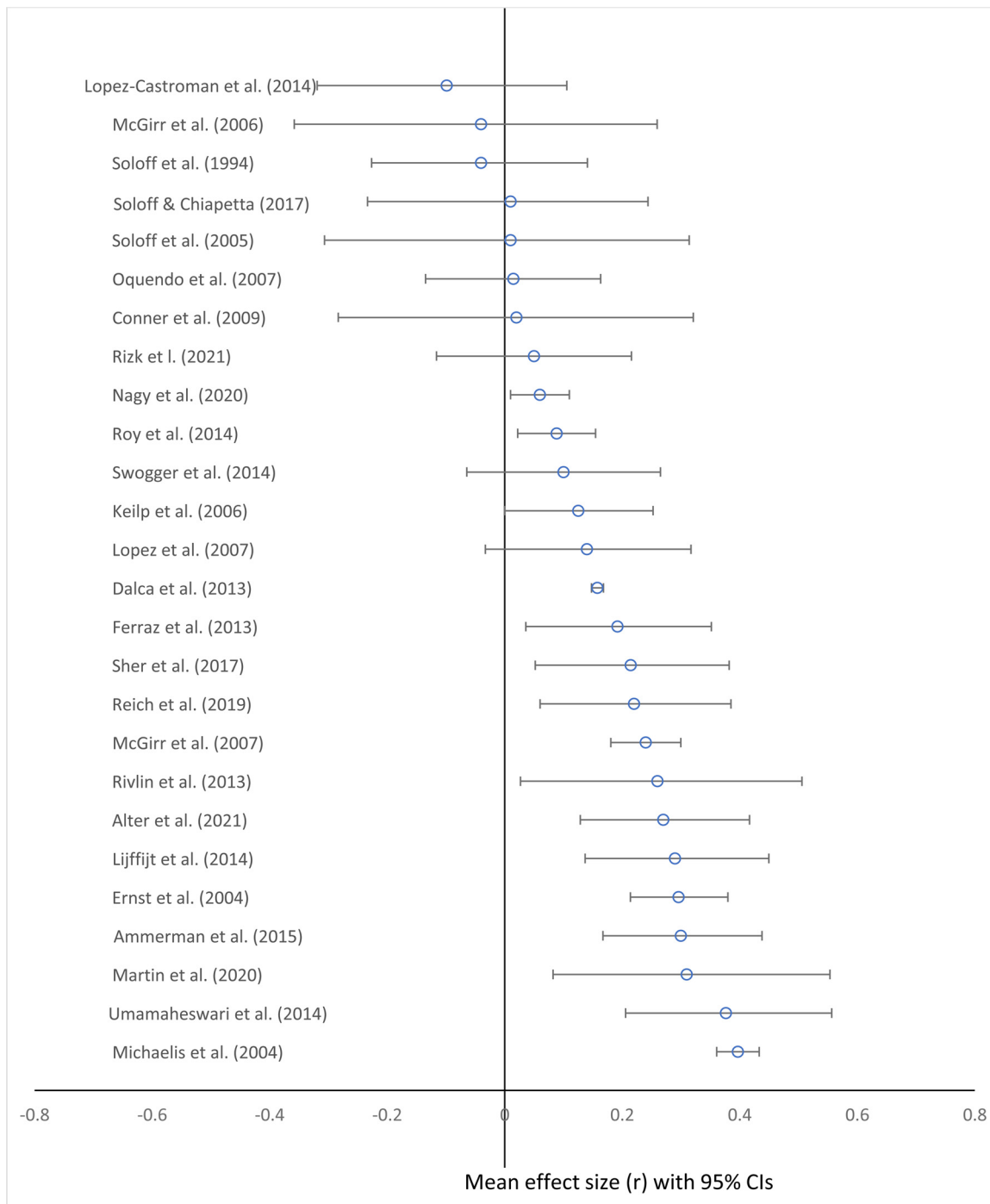
Figure 3. Forest plot showing mean and 95% CI of effect sizes for aggression in relation to suicidality.

There was significant heterogeneity in all cases, which was only partially explained by demographic and methodological moderators. As we will discuss in the following, we argue that our synthesis suggests that does not support the conflation of impulsivity and aggression

with suicidality and call for additional studies in the field to better address this question.

Trait impulsivity was a stronger predictor of suicidality than was state impulsivity. This is consistent with models in which trait impulsivity serves as a distal risk





**Figure 4.** Forest plot showing mean and 95% CI of effect sizes for impulsive aggression in relation to suicidality.

factor for increased vulnerability to suicide,<sup>10,13,14,16,17,30</sup> and contrasts with those in which impulsivity increases risk when elevated under stress.<sup>20,21,32</sup> We could not, however, control for length of time between suicidality and assessment of impulsivity. The state-based tasks which assess impulsivity at one time-point may not

correlate with past suicidality. There were no prospective studies of state impulsivity and suicidality, which would be the strongest test of this relationship. Furthermore, although the relationship between state and trait impulsivity is weak,<sup>33,34,35,37</sup> the ways in which an impulsive disposition translates to state impulsivity and

suicidality during times of distress is unknown. We argue that, while our results support a role of trait impulsivity in suicidality, further work is required to determine how this relates to state impulsivity and suicidality under distress. We also note that the relationship between trait impulsivity and suicidality, while stronger than that of state impulsivity, was however small.

The relationship between impulsivity and suicidality was stronger for behavioral than cognitive impulsivity. This is in contrast with Liu et al.<sup>20</sup> Liu et al. included only neuropsychological measures of impulsivity, whereas we included self-reported assessments.<sup>20</sup> Self-report may be less objective than neuropsychological assessments<sup>20</sup> and, as there were significantly more results for self-report than neuropsychological assessments in our sample (70 and 7, respectively; Supplementary Material 4), it is possible that our sample was prone to Type I error. Given the number of results testing both behavioral ( $n = 54$ ) and cognitive ( $n = 43$ ) impulsivity, however, we are confident that our finding has some validity and argue that behavioral impulsivity (particularly when assessed through self-report) merits further research to explore its value as a target for suicide risk assessment and management. Again, however, we note the small effect size and significant heterogeneity.

The relationship between aggression and suicidality was not moderated by population or by measure of aggression. Unlike impulsivity there were no differences between state and trait measures of aggression, meaning that it is not possible to conclude with which psychological model of aggression and suicide our findings fit most closely. For both aggression and impulsivity, the relationship was moderated by measure of suicidality such that, in both cases, the relationship was strongest for measures of suicide risk. For both, relationships to suicide risk were medium, and to all other measures of suicidality were small. Two measures of suicide risk were employed (Suicide Probability Scale and Suicide Risk Scale).<sup>56,32</sup> Both include items that explicitly assess aggression, but may also assess aspects of impulsivity (e.g. 'Have you ever been so angry you that you felt you might kill someone?';<sup>32</sup> 'When I get mad I throw things'<sup>56</sup>), therefore we are cautious in interpreting a link between aggression, impulsivity, and suicide risk using these measures. Furthermore, nearly all studies which assessed suicide risk were based on psychiatric inpatient samples and participants provided answers to risk measures as part of a clinical interview. We question, therefore, whether these populations are representative of links between suicidality, impulsivity, and aggression more broadly. For example, given evidence that risk assessment tools perform worse than chance,<sup>7,8</sup> these relationships may reflect cultural or organizational beliefs about the contribution of impulsivity and aggression to suicidality, erroneously conflating the three and inflating clinician's ratings of risk. Furthermore, it would be insightful to know whether

self-report assessment of risk correlates with clinician assessments (e.g. based on clinical interviews) and/or whether these relationships differ depending upon the population of interest. Aggression may be interpreted and treated differently in forensic psychiatric versus community populations, for example. In light of this, meta-analysis of standardized regression coefficients which control for demography and mental health would be desirable. In our dataset, however, there was insufficient consistency in the ways in which demography and mental health were assessed to facilitate this.

The relationship between suicidality and impulsive aggression was moderated by population, such that it was stronger in studies of non-clinical populations. This may, again, reflect differences in the ways in which individuals rate or report their own suicidality compared to clinician judgement or objective measures such as cause of death. Clinical samples were more likely to include clinician-completed ratings of risk or lethality, or cause of death, than were non-clinical populations which relied more heavily on self report.

While we excluded studies which did not include reliable, validated, psychometric measures of impulsivity and aggression, we have relatively less confidence in the quality of measures of impulsive aggression. Furthermore, the majority of studies which measured impulsive aggression did so with the Buss and Durkee Hostility Index,<sup>57</sup> which was developed as a measure of hostility and there is, to our knowledge, no convincing evidence that it provides a valid or reliable measure of impulsive aggression. Contributing to this is a lack of consensus regarding the definition of the construct. It is unclear from our review of the relevant theory and data, for example, whether we should predict individuals high in impulsive aggression to be highly aggressive and highly impulsive, or to be highly impulsive in the expression of aggression specifically. Is it, as argued by Brent and Mann,<sup>30,31</sup> a hostile reaction to provocation? Is it reactive, rather than proactive, aggression? Is aggression a distinct variable that sits alongside impulsivity under a disinhibition psychopathology? While we did not find evidence that impulsivity, aggression, or impulsive aggression, differed in their relation to suicidality, perhaps supporting the existence of a disinhibition psychopathology, it is unclear what underlying constructs were being assessed by measurements of 'impulsive aggression'. Without more precisely delineated parameters, measurement, and understanding of the clinical relevance of the construct, we have been unable to answer these questions.

Given the significant heterogeneity revealed in our synthesis, which remained despite the inclusion of methodological moderators, it is important to qualify any interpretation of our results with the disclaimer that methodological limitations may have impacted upon the extent to which our pooled effect sizes are statistically or clinically meaningful.<sup>61</sup> For example, where

available we used  $r$  as a measure of effect size, and converted available effect sizes to  $r$  where alternative measures were reported. In the latter instances, we were unable to check for linearity which is an assumption of Pearson's  $r$ . We may have failed to consider more relevant moderators which better explain the variance. Gender, age, mental health diagnosis, or substance use, are all examples which may conceivably influence the relationships of interest. In particular, we note the bodies of evidence linking substance use to each of impulsivity,<sup>58</sup> aggression,<sup>59</sup> and suicidality,<sup>60</sup> and argue that work which explores these pathways will contribute to our understanding. As described above, the inclusion of covariates via multivariate regression coefficients in meta-analyses may provide greater precision around the nature of relationships. However, consistent assessment of potential covariates was not present in our sample. Alternatively, it is possible that there are too few studies at each level of our moderators and/or methodological differences between them are too substantial, for us to detect moderation. Additionally, while impulsivity and, to a lesser extent, aggression, are defined and operationalized in the literature, assessments of these in the context of suicidality is difficult, and the lack of studies which assessed either proximal to suicidality is a limitation of the literature.

In conclusion, then, we have suggested impulsivity (particularly behavioral and trait impulsivity) and aggression to correlate with suicidality. Associations, however, were weak, with extremely high heterogeneity that was only partially explained by methodological moderators. We argue that, while both constructs may be of some relevance in assessing and managing suicide risk, caution should be exercised in conflating such broad constructs with suicidality when the evidence to date does not seem to support clear or meaningful links. While our results are consistent with a model in which impulsivity and aggression form part of a wider psychopathology of relevance to suicidality, we argue that greater clarity on the definition, clinical relevance, and measurement of impulsive aggression is required. Future research which takes a person-centered approach to understanding the ways in which impulsivity and aggression interact with distal and proximal, and static and dynamic, risk factors in the lives of individuals will contribute to greater precision in predicting risk, and in understanding the mechanisms by which individual circumstances translate into suicidality.<sup>62</sup>

#### Declaration of interests

We declare no competing interests.

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None.

#### Data sharing

The full dataset (including article meta-data and effect sizes) is available from [fhionnarosemoore@gmail.com](mailto:fhionnarosemoore@gmail.com).

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#### Contributors

FM conceived and designed the work, acquired and analysed data, interpreted results, and wrote the manuscript supervised by RO and CA. HM provided critical evaluation of the manuscript and processed a sub-sample of the data to allow assessment of inter-rater reliability. HD and TN contributed significantly to scoping of the data prior to the final search strategy and provided critical evaluation of the manuscript. All authors have access to and verify the final dataset, provided final approval for the work to be published and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

#### Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:[10.1016/j.eclinm.2022.101307](https://doi.org/10.1016/j.eclinm.2022.101307).

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