


Posttraumatic stress and delay discounting: a meta-analytic review

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Review Article

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Abstract

Delay discounting—the extent to which individuals show a preference for smaller immediate rewards over larger delayed rewards—has been proposed as a transdiagnostic neurocognitive process across mental health conditions, but its examination in relation to posttraumatic stress disorder (PTSD) is comparatively recent. To assess the aggregated evidence for elevated delay discounting in relation to posttraumatic stress, we conducted a meta-analysis on existing empirical literature. Bibliographic searches identified 209 candidate articles, of which 13 articles with 14 independent effect sizes were eligible for meta-analysis, reflecting a combined sample size of $N = 6897$. Individual study designs included case-control (e.g. examination of differences in delay discounting between individuals with and without PTSD) and continuous association studies (e.g. relationship between posttraumatic stress symptom severity and delay discounting). In a combined analysis of all studies, the overall relationship was a small but statistically significant positive association between posttraumatic stress and delay discounting ($r = .135$, $p < .0001$). The same relationship was statistically significant for continuous association studies ($r = .092$, $p = .027$) and case-control designs ($r = .179$, $p < .001$). Evidence of publication bias was minimal. The included studies were limited in that many did not concurrently incorporate other psychiatric conditions in the analyses, leaving the specificity of the relationship to posttraumatic stress less clear. Nonetheless, these findings are broadly consistent with previous meta-analyses of delayed reward discounting in relation to other mental health conditions and provide further evidence for the transdiagnostic utility of this construct.

Introduction

Posttraumatic stress disorder (PTSD) can develop following exposure to one or more traumatic events (Goldstein et al., 2016; Koenen et al., 2017), with symptoms including intrusions (e.g. distressing memories or dreams related to the trauma), alterations to cognitions and mood (e.g. exaggerated negative beliefs pertaining to self, others, or the world; persistent negative emotional state), heightened arousal and reactivity (e.g. hypervigilance, concentration difficulties), and avoidance of stimuli associated with the traumatic event (APA, 2022). Epidemiological studies in North America have found lifetime PTSD prevalence rates that range from 5.0 to 6.8% in the general population (Kessler, Chiu, Demler, Merikangas, & Walters, 2005; Statistics Canada, 2022). Despite ongoing efforts to understand and implement effective psychotherapeutic treatments for PTSD, there remain substantial treatment challenges, in part due to the heterogeneity of its presentation, an incomplete understanding of the etiological and maintenance factors, and features of the disorder that hinder treatment, potentially including impulsive decision making (e.g. substance use) leading to premature treatment discontinuation (Back, Waldrop, & Brady, 2009; Lewis, Roberts, Gibson, & Bisson, 2020; Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008; Zoellner, Pruitt, Farach, & Jun, 2014).

Recent efforts have been aimed at developing a psychiatric nosology that emphasizes transdiagnostic processes in the development and maintenance of multiple disorders, offering potential for novel treatment targets (Cuthbert, 2022; Dalgleish, Black, Johnston, & Bevan, 2020; Kozak & Cuthbert, 2016) and greater treatment efficiency (Barlow, Harris, Eustis, & Farchione, 2020). One relevant neurocognitive process showing promise as a transdiagnostic factor is delay discounting, a behavioral economic index that assays individual preference

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for smaller but immediate rewards over those that are larger but delayed. Delay discounting has historically been considered a behavioral index of impulsivity, but there remains controversy about the nature of impulsivity as a psychological construct, how delay discounting fits into such a conceptualization, and the degree to which delay discounting may be truly transdiagnostic (Bailey, Romeu, & Finn, 2021; Levitt et al., 2022; Stein, MacKillop, McClure, & Bickel, 2023; Strickland & Johnson, 2021).

Delay discounting is typically measured using intertemporal choice tasks, in which individuals make decisions between two rewards that vary in magnitude and delay to their receipt (Madden & Bickel, 2010). Systematic reviews and meta-analyses for continuous association and case-control studies both suggest that delay discounting is positively associated with a variety of mental health symptoms and conditions, including substance use quantity/frequency and addiction severity (Amlung, Vedelago, Acker, Balodis, & MacKillop, 2017; MacKillop et al., 2011), behavioral addictions (e.g. gambling and internet gaming disorders; Weinszok, Brassard, Balodis, Martin, and Amlung, 2021), attention-deficit/hyperactivity disorder (ADHD; Jackson & MacKillop, 2016), and both dysregulated eating (Stojek & MacKillop, 2017) and obesity (Amlung, Petker, Jackson, Balodis, & MacKillop, 2016). In a recent meta-analysis of case-control studies for an array of mental health conditions, Amlung et al. (2019) reported that, relative to controls, elevated rates of delay discounting were found among individuals with major depressive disorder, schizophrenia, borderline personality disorder, bipolar disorder, binge eating disorder, and bulimia nervosa, providing broad support for the transdiagnostic significance of delay discounting. While promising, fewer studies have examined delay discounting in relation to posttraumatic stress, and it remains unclear whether similar associations are present.

There are specific features of PTSD that might contribute to steep discounting of delayed rewards. A defining feature of PTSD is avoidance, whereby individuals make persistent attempts to reduce distress in the short-term by attempting to avoid internal triggers (i.e. thoughts, feelings, physiological sensations) and/or external stimuli associated with the trauma. Short-lived reductions in distress contribute to a negatively reinforced cycle that promotes increasing reliance on avoidance, while simultaneously limiting engagement in activities that are positively reinforcing, disconfirming of trauma-related cognitions, or that may foster habituation or extinction of trauma-related emotions (Foa & Cahill, 2001; Foa, Hembree, Rothbaum, & Rauch, 2019; Olin et al., 2022; Rauch & Foa, 2006). Thus, like delay discounting, avoidance in PTSD is a short-term strategy to maximize the immediate smaller reward of distress reduction at the expense of long-term symptom recovery. PTSD symptom severity can also be associated with increased perceptions of uncontrollability and unpredictability (Bolstad & Zinbarg, 1997), and in previous versions of the *Diagnostic and Statistical Manual of Mental Disorders*, a characteristic of PTSD was having a sense of foreshortened future (i.e. a sense of foreboding or that one could die at any time; Ratcliffe, Ruddell, and Smith, 2014). Collectively, these features are consistent with behavioral economic theory and the process of delay discounting.

Despite theoretical and conceptual reasons to expect a positive association between posttraumatic stress and delay discounting, individual studies examining this link have thus far produced mixed results. For example, in a large sample of community adults, Levitt et al. (2022) reported statistically significant correlations between posttraumatic stress symptom severity and delay discounting ($r_s = 0.14$ to 0.16). In contrast, using a smaller sample

of active or retired military personnel, Olin et al. (2022) found a non-significant negative association between similar variables ($r = -0.050$). Using a case-control design, Morris et al. (2020) found that delay discounting was significantly elevated among individuals with PTSD relative to those without ($d = 0.32$), whereas Peck, Nighbor, and Price (2021) found non-significant elevations in delay discounting among individuals with PTSD (without opioid use disorder) relative to healthy controls ($d = 0.14$) (calculated based on information provided in text).

To address this question, the present study meta-analyzed the primary empirical literature with the goal of characterizing the presence, direction, and strength of the relationship between post-traumatic stress symptoms and delay discounting. Secondary goals were to examine study-level moderating variables that may account for heterogeneity in effects, and to estimate the extent to which findings may be influenced by small study (publication) bias.

Method

Study selection

The methods of this review were pre-registered (CRD42022369226) in the International Prospective Register of Systematic Reviews (PROSPERO). Systematic searches were conducted in PsycINFO, Medline/PubMed, Embase, and Web of Science for articles through November 25, 2022. Boolean terms included the following: (post-traumatic* OR post-traumatic* OR PTSD OR trauma* OR acute stress disorder) AND (delay discounting OR temporal discounting OR intertemporal choice OR impulsive choice). Studies were eligible for inclusion if they met the following criteria: published in a peer-reviewed journal, available in English, human participants, included a delay discounting measure(s), reported on differences between groups (e.g. trauma exposed *v.* not; PTSD *v.* no PTSD) or a continuous measure of association between posttraumatic stress symptom severity and delay discounting. Studies were excluded for the following reasons: involved an experimental stress manipulation or a clinical intervention, due to possible confounding (pre-intervention/pre-manipulation associations were eligible, although none of the included studies ultimately met this criteria); were primarily focused on the association between traumatic brain injury and delay discounting, given a different scope; measured trauma using total scores for adverse childhood events (ACEs), to disambiguate traumatic events as per Criterion A of the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition Text Revision* (DSM-5-TR; APA, 2022) from other forms of adversity included among ACEs. To be maximally inclusive, no restrictions were placed on the type of delay discounting reinforcer, or the measures used to assess posttraumatic stress or delay discounting.

Two authors of the present work (BMB and EEL) independently performed article screening and coding/extraction using the Covidence systematic review software (Veritas Health Innovation), and discrepancies were resolved through consensus ratings. Inter-rater agreement was excellent for title/abstract (92.8% agreement, $\kappa = 0.75$) and full text screening (100% agreement). For eligible studies that did not report enough information to derive the effect of interest, the lead author of the present work contacted the corresponding authors of prior studies with a request for the effect size (or information to derive the effect size) of interest. Studies were excluded if a response was not received after two attempts to obtain the requested information.

Characteristics of included studies

The following information was coded or extracted from the included studies: title, year of publication, study design (case-control or cross-sectional with continuous associations), sample sizes after exclusions, percent female, percent White/European ancestry, mean age of the sample, measure of trauma and/or posttraumatic stress, and measure of delay discounting. If age was reported separately by group (e.g. PTSD *v.* Controls), we took the weighted average of the ages. In one instance, age was binned in various categories, in which case we used the median as an estimate of average age. If an average effect was derived for a study that reported more than one relevant outcome or comparison within the same sample (see meta-analytic approach section), and sample sizes were also reported separately for each outcome (and/or each group), we used the respective average *n* for meta-analysis, which resulted in a non-discrete sample size for one study. If the percentage of females was not reported for the final sample (i.e. the sample after exclusions), we used the percentage of females in the pre-exclusion sample as a corresponding estimate. In two instances, the authors reported the number of 'women' or 'men', which are traditionally used as terms referring to gender rather than sex. Given that no separate descriptive statistics were provided for sex, these numbers were used to estimate the percentage of females for the respective samples. In two other instances, the descriptive heading was 'gender' but included the number or percentage of 'male' or 'female' (traditional sex terms) and thus were assumed to be corresponding estimates of biological sex.

Meta-analytic approach

The primary effect of interest was the correlation (Pearson's *r*) between posttraumatic stress and delay discounting, which is converted to Fisher's *Z* for analysis (Borenstein, Hedges, Higgins, & Rothstein, 2021). Effects from categorical designs were also converted into the same indicator. If the correlation of interest was not reported in the article, effects were initially derived based on available information that allowed an effect to be calculated (see online Supplementary Material Table S2 for information by article). For example, when available, means and standard deviations were extracted for articles reporting on differences between groups. Effects from categorical studies were based on dichotomous categorical variables (e.g. PTSD *v.* no PTSD group) and continuous delay discounting outcomes. In instances where authors reported multiple statistical models (or examined differences for more than one potential group of relevance), we derived effects based on the least confounded model/group. To avoid issues of non-independence and artificially assigning more statistical weight to an individual sample in the meta-analysis (Borenstein et al., 2021), for studies that reported more than one delay discounting outcome within the same sample ($k = 5$), we averaged the estimates (i.e. average of the associations between posttraumatic stress and delay discounting outcomes) to form a single effect size. In each instance, delay discounting outcomes were from the same task (e.g. small, medium, and large reward magnitudes for the Monetary Choice Questionnaire; Levitt et al., 2022). In two studies, correlations were reported between different posttraumatic stress symptom clusters and delay discounting, in which case we preferentially used the correlation for the overall posttraumatic stress symptom index as the most representative estimate.

Some studies of delay discounting report results using an area under the curve (AUC) analysis—which quantifies distances

between observed values (subjective value of a reward) and delay to their receipt (Myerson, Green, & Warusawitharana, 2001). With higher (steeper) delay discounting, AUC values decrease. Frequently, other studies use metrics that are positively associated with discounting of delayed rewards. For example, delay discounting can be modeled with a hyperbolic function ($V = A/(1 + kD)$), where *V* is the current value of the delayed reward, *A* is the amount of the delayed reward, *D* is the reward delay, and *k* is a derived parameter that indicates the discounting rate, with higher *k* values indicating steeper discounting of delayed rewards (Mazur, 1987). One study from the current meta-analysis used AUC analysis, and thus the effect from this study had its sign reversed prior to inclusion (given inverse association between AUC values and *k* indices).

Effect size conversion, and the meta-analysis itself, were principally performed with Comprehensive Meta Analysis V 4.0 (Borenstein, Hedges, Higgins, & Rothstein, 2022). When the studies in the meta-analysis are not functionally identical, the random effects model is often recommended over the fixed-effects model as the former accounts for both within-study sampling error and between-study variance, while guarding against inflated effect estimates (Borenstein et al., 2021; Schmidt, Oh, & Hayes, 2009). Given that true effect sizes are likely to vary with the different sample characteristics and study designs, a random effects meta-analytic approach was employed. Heterogeneity in effects was quantified with Cochran's *Q* test (a test of the null hypothesis that the included studies share a common effect size) and the I^2 statistic (the proportion of observed variance that reflects true differences in effect rather than sampling error; Higgins and Thompson, 2002). A one-study-removed analysis was also conducted to determine the extent to which individual studies disproportionately affected the summary effect. When there were sufficient sample sizes, moderator analyses were conducted to determine if categorical differences (subgroups analysis) or continuous variables (meta-regression) accounted for a significant portion of effect heterogeneity.

Risk of bias was assessed with multiple indices. Rosenthal's classic fail-safe *n* (Rosenthal, 1979) is an estimate of the number of effects averaging an effect size of 0 that would be required to nullify the summary effect of the meta-analysis. The funnel plot for the summary effect was also visually inspected for symmetry of effect sizes around the mean effect. Examination of the funnel plot was supplemented by (1) the Begg-Mazumdar rank correlation test of the association between the treatment effect and the standard error (Begg & Mazumdar, 1994) and (2) Egger's one-tailed test of the regression intercept (Egger, Davey Smith, Schneider, & Minder, 1997), both of which provide indications of whether small sample studies are more likely published due to having larger effect sizes. Lastly, an adjusted estimate of the summary effect was computed using the trim-and-fill approach (Duval & Tweedie, 2000), which re-calculates the summary effect after imputing studies that are likely to be missing to the left of the summary effect in the forest plot.

Results

Preliminary analyses

The Preferred Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram (Page et al., 2021) appears in Fig. 1. Following exclusion of non-eligible studies, and three studies whose authors did not respond to data requests, the meta-analysis sample included a total of 13 studies, 14 independent effect sizes, and a combined sample size of $N = 6897$.

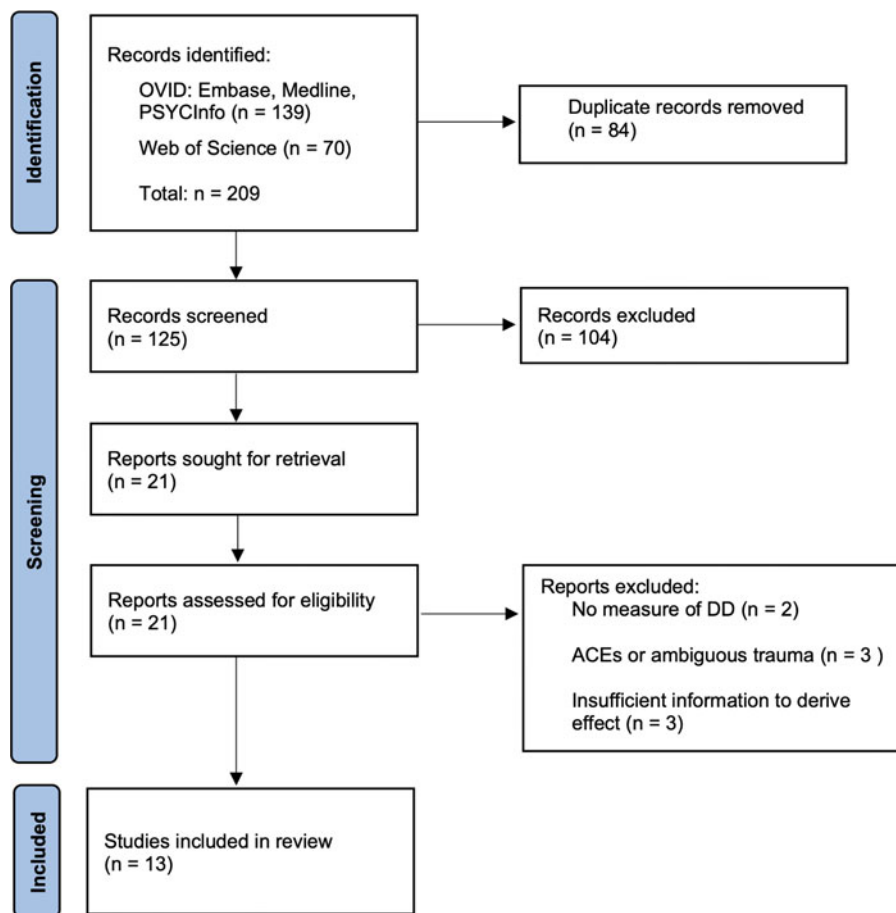


Figure 1. PRISMA flow diagram for study selection.
Note. DD, Delay discounting; ACEs, Adverse childhood events.

Characteristics of the included samples are depicted in Table 1. Mean ages ranged from 8.3 to 76.2 years (mean weighted age across samples = 35.7, median = 37.1 years). Study design was evenly split between case-controls (trauma exposure *v.* controls, $k = 3$; groups with PTSD *v.* controls, $k = 4$) and continuous associations ($k = 7$). Traumatic exposure and/or posttraumatic stress severity was most commonly indexed using self-report (or in one instance, parent-report) measures ($k = 10$), followed by clinician-administered semi-structured interviews ($k = 4$). Delay discounting was indexed with measures that employ pre-configured items (Monetary Choice Questionnaire [MCQ] or an adapted version thereof, $k = 5$; brief MCQ, $k = 2$; incentivized choice task, $k = 1$) or an adjusting amount task (ED_{50} , $k = 3$; other, $k = 3$). See Table 1 for a list of measures and tasks. Nearly 80% of studies addressed possible influence of a skewed delay discounting distribution, either by using transformed delay discounting indicators ($k = 9$), by using a non-parametric test ($k = 1$), or by reporting that skewness and kurtosis were within normal limits ($k = 1$). Three studies did not report whether transformations were used or required.

Meta-analysis results

Meta-analytic results, in addition to a forest plot, appear in Fig. 2. The analysis revealed a statistically significant positive summary effect of small magnitude ($r = 0.135$, $p < 0.0001$, 95% CI [0.074, 0.195]), with high heterogeneity across studies ($Q = 67.974$, $p < 0.001$; $I^2 = 81\%$). The one-study-removed analysis revealed that the summary effect was not unduly influenced by any single

effect size (estimates = 0.112–0.151, all $ps < 0.001$; see online Supplementary Material Table S1).

Risk of bias

The fail-safe N indicated that an additional 353 studies would be required to nullify the significant summary effect. The Begg-Mazumdar test (Kendall's $\tau = 0.07$, one-tailed $p = 0.37$), and Egger's test of the regression intercept (intercept = 0.11, $p = 0.47$) were both non-significant. Examining the funnel plot revealed symmetric distribution around the mean effect for studies with moderate to larger samples, with two smaller sample studies located to the right of the mean effect (see online Supplemental Materials Fig. S1). The trim and fill technique (Duval & Tweedie, 2000) suggested the possibility that one study was missing to the left of the mean effect, although the random effects point estimate remained of similar magnitude at 0.128 (95% CI 0.07–0.19). Collectively, the various indices suggest that there is minimal evidence or influence of publication bias.

Moderator analysis

For categorical moderators, the effect size was statistically significant for continuous association studies ($r = 0.092$, $p = 0.027$) and those employing case-control designs ($r = 0.179$, $p < 0.001$), with no statistically significant difference between the two ($Q = 1.98$, $p = 0.160$). Similarly, the effect size was statistically significant for studies assessing traumatic exposure and/or PTSD symptoms

Table 1. Characteristics of studies included in the meta-analysis

Article	Country	Study Design	Group Characteristics	Total <i>N</i>	Age (<i>M</i>)	% Female	Trauma/PTSD Index	DD Task
Bryan and Bryan (2021)	US	Case-control	General community –PTSD/–SA and +PTSD/–SA	574	38.71	58.57	PC-PTSD-5	MCQ 21
Engelmann, Maciuba, Vaughan, Paulus, and Dunlop (2013)	US	Case-control	General community MDD + PTSD and HC	25	37.04	75.00	SCID for DSM-IV	DDT ^a
Levitt et al. (2022)	Canada	Continuous	General community	1344	38.99	57.90	PCL-5	MCQ
Luciano, Acuff, McDevitt-Murphy, and Murphy (2020)	US	Continuous	General community + TE	91	26.53	36.26	PCL-5	bMCQ
Matsuyama et al. (2020)	Japan	Case-control	General community ± TE	129.5	8.30	49.10	Traumatic exposure ^b	ICT
Minhas et al. (2020)	Canada	Continuous	General community + HED	728	21.44	52.60	PCL-5	ED ₅₀
Minhas et al. (2020)	US	Continuous	General community + HED	602	22.63	57.30	PCL-5	ED ₅₀
Minhas, Cooper, Sousa, Costello, and MacKillop (2022)	Canada	Continuous	Inpatient addiction tx + SUD	712	42.00 (median)	27.90	PCL-5	MCQ ^c
Morris et al. (2020)	US	Case-control	Online sample ± PTSD	1609	37.20	59.00	PCL-5	ED ₅₀
Olin et al. (2022)	US	Continuous	U.S. Military ≥1 combat deployment	106	37.11	12.70	CAPS-5	bMCQ
Olson et al. (2018)	US	Continuous	General community + TE	40	32.27	41.18	CAPS-IV	DDT ^d
Peck et al. (2021)	US	Case-control	General community + PTSD/–OUD and HC	87	27.22	79.31	PCL-5	MCQ
Simmen-Janevska, Forstmeier, Krammer, and Maercker (2015)	Switzerland	Case-control	Former child laborers (TE) and HC	153	76.23	44.44	Traumatic exposure (CTQ-SF)	MCQ ^e
van den Berk-Clark, Myerson, Green, and Gruzza (2018)	US	Case-control	Alcohol tx AUD + TE and HC	696	41.70	53.00	Traumatic exposure (SSAGA-II)	DDT ^f

Note: ± indicates the presence/absence of a group characteristic; group characteristics and sample sizes reflect those for which effect sizes were extracted or derived and thus may differ from the original study; DD, Delay discounting; PTSD, Posttraumatic Stress Disorder; SA, Suicide Attempt; MDD, Major Depressive Disorder; HC, Healthy Controls; TE, Trauma Exposure; HED, Heavy Episodic Drinking; SUD, Substance Use Disorder; OUD, Opioid Use Disorder; AUD, Alcohol Use Disorder; tx, treatment; SCID for DSM-IV, Structured Clinical Interview for the DSM-IV (First & Gibbon, 2004); CAPS for DSM-IV and DSM-5, Clinician-Administered Posttraumatic Stress Disorder Scale for the DSM-IV (Blake et al. 1995) and DSM-5 (Weathers et al. 2013); CTQ-SF, Childhood Trauma Questionnaire Short Form (Bernstein et al. 2003); SSAGA-II, The Semi-Structured Assessment for the Genetics of Alcoholism-II (Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999); PC-PTSD-5, The Primary Care PTSD Screen for the DSM-5 (Prins et al. 2016); MCQ 21, Monetary Choice Questionnaire 21-item version (Kirby & Maraković, 1996); MCQ, Monetary Choice Questionnaire (Kirby, Petry, & Bickel, 1999); DDT, Delay Discounting Task (note that 'DDT' was used to indicate general delay discounting tasks that were not named in the original publications, rather than the name of a particular task); bMCQ, Brief MCQ (also referred to as Brief Delay Discounting Task; Gray, Amlung, Acker, Sweet, and MacKillop, 2014); ICT, Incentivized Choice Task (Angerer, Lergetporer, Glätzle-Rützler, & Sutter, 2015); ED₅₀, Effective Delay 50 (also referred to as the 5-item DDT; Koffarnus and Bickel, 2014).

^aWittmann, Leland, and Paulus (2007).

^bParent reported child exposure to trauma (for the current meta-analysis, we used an average estimate from 'witnessing someone being swept away by a tsunami' and 'saw a dead body' to align most closely with the definition of a Criterion A traumatic event in the DSM-5-TR); the non-discrete *N* reflects the sum of average *N*s for witnessed (*N* = 6.5) and did not witness (*N* = 123) a traumatic event.

^cOnly medium magnitude reward levels used.

^dYoung (2017).

^eSwiss-German version (Forstmeier & Maercker, 2011).

^fDu, Green, and Myerson (2002).

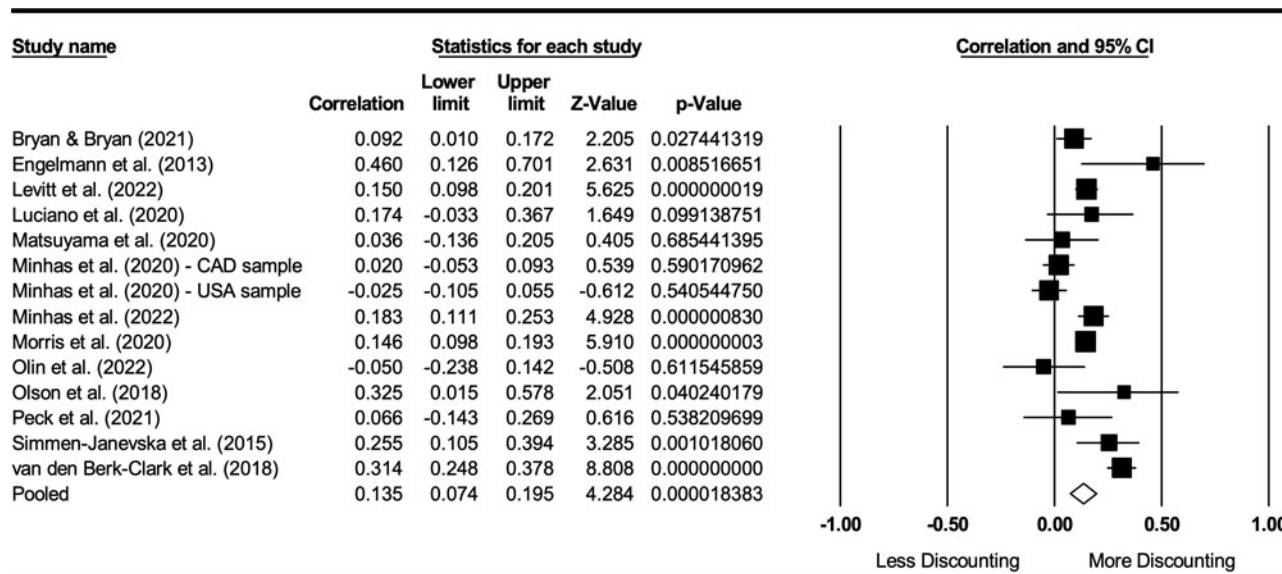


Figure 2. Forest plot of studies included in the meta-analysis. Box size is proportional to study weight. Black diamond depicts the summary effect, indicating a positive meta-analytic association between posttraumatic stress and delay discounting.

with self-report instruments (includes Matsuyama et al. (2020), which assessed trauma using parent report) ($r = 0.107$, $p < 0.001$) and clinician-assessed with semi-structured interview ($r = 0.249$, $p = 0.028$), with no statistically significant difference between the two ($Q = 1.53$, $p = 0.216$). The effect size was also statistically significant for studies employing adjusting amount delay discounting tasks ($r = 0.166$, $p = 0.011$) and pre-configured items delay discounting tasks ($r = 0.132$, $p < 0.001$), with no statistical difference between the two ($Q = 0.24$, $p = 0.625$).

Meta-regression for continuous moderators produced significant estimates for year of publication (coefficient = -0.052 , $p = 0.017$) and mean sample age (coefficient = 0.008 , $p = 0.009$), suggesting that effect sizes tended to decrease over time and increase with the mean age of the sample. Caution is warranted, however, given the relatively low number of samples, and the restricted range for year of publication (2015–2022). Percent female (coefficient = 0.003 , $p = 0.295$) and percent White/European ancestry (coefficient = -0.002 , $p = 0.327$) were not significantly associated with effect size.

Discussion

The current meta-analysis provides the first synthesis of literature on the association between posttraumatic stress and delay discounting. The principal analysis identified a small but significant positive association between posttraumatic stress and delay discounting. Given the heterogeneity in associations, it is possible that steep delay discounting is a feature of posttraumatic stress, but not a defining one, and thus may be variably present. It is worth considering that delay discounting can be influenced by one's context, including stimulating social environments (Bixter & Luhmann, 2021; Gilman, Curran, Calderon, Stoekel, & Evins, 2014; Martínez-Loredo, 2023). For some individuals, PTSD-related avoidance may reduce their exposure to such environments that otherwise might enhance delay discounting. The extent to which this contributes to variability in the associations examined in the current study, and whether other factors (e.g. the number and nature of traumatic events experienced) play

important roles, requires further study. More generally, the overall effect size here is consistent with recent arguments that complex psychological phenomena—which are likely determined by a multitude of causes—may have small but important effects that contribute to a cumulative psychological science (Götz, Gosling, & Rentfrow, 2022).

Various metrics indicate that the summary effect was unlikely to be unduly influenced by publication bias. Examination of key candidate moderators revealed no statistical difference between study designs (case-control *v.* continuous association studies) or methodology (traumatic exposure and/or posttraumatic stress severity measured with self-report instrument *v.* clinician-administered semi-structured interview; adjusting amount *v.* pre-configured item delay discounting tasks). The effect size did tend to decrease with year of publication and increase with mean age of the sample, but interpretation is limited by the relatively small number of samples in the analysis and the relatively recent publication of articles. As additional studies accumulate, it may be useful for future meta-analytic work to examine age and year of publication as moderators of effect size.

As noted, a defining feature of PTSD is avoidance of stimuli associated with the trauma, likely contributing to a negatively reinforced cycle of short-lived reductions in distress and increasing reliance on avoidance. Past work has also shown that relative to trauma-exposed controls without PTSD, individuals with PTSD show lower future specificity (i.e. the generation and description of possible future events in one's life) in response to positive (Kleim, Graham, Fihosy, Stott, & Ehlers, 2014) and neutral word cues (Brown et al., 2013). PTSD may thus be at least partly characterized by a relatively greater focus on the short-term and/or a pessimistic future perspective, which may be reflected in the positive association between posttraumatic stress and delay discounting.

Notably, the findings here are broadly consistent with evidence for a link between delay discounting and other psychiatric disorders or measures of psychiatric symptomology, such as addiction severity and substance use disorders, major depressive disorder, bulimia nervosa and binge eating disorder, ADHD, schizophrenia

spectrum disorders, bipolar disorder, and borderline personality disorder (Amlung et al., 2019; 2017; Jackson & MacKillop, 2016; MacKillop et al., 2011; Stojek & MacKillop, 2017). Further, findings are of similar magnitude to prior meta-analysis of continuous associations, specifically (e.g. $r = 0.14$ for addiction severity/quantity-frequency and delay discounting; Amlung et al., 2017).

Limitations and future directions

Despite using a relatively large aggregated sample for the current report, the literature on the association of posttraumatic stress with delay discounting remains small relative to other psychiatric disorders (e.g. substance use). Further, although candidate moderators for design and methods were not statistically significant, results suggest the possibility that as further studies accumulate, it may reveal larger effects for case-control over continuous association studies, and/or for those employing clinician-administered semi-structured interviews over self-report measures of post-traumatic stress symptom severity. As the literature develops, it will be useful for future meta-analyses to more closely examine these potential effect moderators, which may help clarify and explain the significant heterogeneity that was observed.

Other aspects of methodological heterogeneity remain important considerations for future work. Most studies transformed the delay discounting variable, which is frequently skewed, but in other instances, it was unclear whether these steps were performed or necessary. It is recommended that future studies on PTSD symptoms and delay discounting report whether transformations were performed prior to analysis. With one exception, the studies in the current meta-analysis also focused on samples of adults. While current diagnostic criteria for PTSD in children is similar (in children 6 years or younger) or the same (in children older than 6 years) to those for adults, some symptom expression may differ (APA, 2022). Additionally, the extent to which individuals discount delayed rewards can change across development (Klein, Collins, & Luciana, 2022). It remains unknown to what degree traumatic exposure and/or PTSD symptoms may have a differential association with delay discounting across developmental stages, representing a fruitful area for future research.

Additional consideration can be given to the measurement of posttraumatic stress. Only two studies examined associations between PTSD cluster scores and delay discounting (Olin et al., 2022; Olson, Kaiser, Pizzagalli, Rauch, & Rosso, 2018), and the results were inconsistent. Olson et al. (2018) found that an avoidance symptom cluster was most strongly positively correlated (controlling for age and sex) with delay discounting ($r = 0.388$) relative to hyperarousal ($r = 0.320$) or re-experiencing ($r = 0.144$). Olin et al. (2022), on the other hand, found weak, non-significant correlations between delay discounting and clusters of avoidance ($r = -0.05$), intrusions ($r = -0.04$), negative alterations in cognition and mood ($r = -0.07$), and alterations in arousal and reactivity ($r = 0.02$). Nevertheless, some previous work has found differential associations between individual PTSD cluster scores and behaviors associated with delay discounting, such as substance use (Livingston, Farmer, Mahoney, Marx, & Keane, 2022; Sullivan & Holt, 2008). As more studies employing this parsing appear in the literature, it may therefore also be useful for future meta-analytic work to directly examine associations between PTSD symptom clusters and delay discounting.

PTSD is highly comorbid with several other psychiatric disorders, including those with well-established links with delay

discounting. It remains largely unknown to what degree comorbidities are relevant or indeed responsible for PTSD associations with delay discounting. Many of the studies included in the current meta-analysis did not systematically measure and incorporate (i.e. control for) other psychiatric conditions in a manner that would allow for meta-analysis, making the specificity of the relationship of delay discounting to posttraumatic stress less clear. It thus remains possible that the association between post-traumatic stress and delay discounting is a function of an unmeasured third variable (e.g. PTSD is highly comorbid with substance use disorder [Pietrzak, Goldstein, Southwick, and Grant (2011)], which is strongly associated with delay discounting [MacKillop et al. (2011)]). As additional studies become available, future work may benefit from examining the degree to which the presence or number of psychiatric comorbidities affects the strength of association with delay discounting, or whether broader disorder categories (e.g. internalizing *v.* externalizing) show differential associations with delay discounting. It seems plausible that delay discounting would be further elevated among individuals with greater levels of psychiatric comorbidity. Determining the extent to which this is true will require a direct test in future work.

To the degree that PTSD is partly characterized by an excessive focus on the short-term (e.g. avoidance and short-term distress relief) and deficits in prospective cognition, important questions can be raised about whether modifying one's focus to be more future-oriented may be useful for such populations. One candidate of rapidly growing interest is episodic future thinking (EFT), which refers to the simulation or imagination of possible future events (Atance & O'Neill, 2001). Meta-analytic work has recently shown that interventions aimed at improving EFT can reduce delay discounting, particularly when the future event is positively valenced (Ye et al., 2022). The extent to which expanding the temporal horizon out of the present toward the future would be useful for individuals with PTSD, in addition to whether modifications would be required to overcome potential interference from traumatic memories, remains an empirical question. Further questions can be raised about the extent to which interventions for PTSD—with or without co-occurring conditions—may lead to clinical change, in part, due to indirect effects of future thinking experiential exercises (e.g. setting values-consistent, long-term goals, as in acceptance and commitment therapy; Meyer et al., 2018) on delay discounting.

Conclusion

The current meta-analysis provides the first quantitative synthesis showing that traumatic exposure and posttraumatic stress are significantly and positively associated with delay discounting. The findings are consistent with previous meta-analytic findings for other psychiatric disorders and provide additional support for delay discounting as a likely transdiagnostic process. Although further refinement of these relations is needed, especially regarding potential confounders, these results nonetheless support investigating future orientation as a novel clinical target for PTSD.

Supplementary material. The supplementary material for this article can be found at <https://doi.org/10.1017/S0033291723003069>

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