# A transdiagnostic meta-analysis of physical and social Anhedonia in major depressive disorder and schizophrenia spectrum disorders

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## ABSTRACT

*Introduction:* Anhedonia is a transdiagnostic construct conceptualized as physical or social, however, the extent to which these subtypes differ across psychotic and mood pathology remains poorly understood. We aimed to quantify the severity of physical and social anhedonia across Major Depressive Disorder (MDD) and Schizophrenia Spectrum Disorder (SSDs).

*Methods:* We conducted meta-analyses of the Chapman Physical and Social Anhedonia Scales (PAS;SAS). We reviewed data from participants with MDD, and SSDs separately.

*Results:* Our first meta-analysis ($n = 8$ studies, 409 participants) with MDD revealed elevated SAS and PAS in MDD compared to controls. Within-group differences were not significant. Depressive symptom severity moderated the between-group effect of PAS. Our second meta-analysis ($n = 44$ studies, 3352 participants) revealed elevated SAS and PAS in SSDs compared to controls. We detected a moderate difference between the SAS and PAS within the SSD group. Age moderated within-group differences of SAS and PAS.

*Discussion:* People with SSD or MDD experience elevated SAS and PAS compared to controls. People with SSDs endorse greater challenges experiencing social rewards relative to physical rewards. People with MDD experience social and physical rewards similarly. The moderating role of depressive symptoms in MDD suggests that physical anhedonia is more state-like than social anhedonia.

## 1. Introduction

Anhedonia is characterized by a reduced experience of pleasure, at least partly reflected by impaired reward processing (Zhang et al., 2016), that can limit goal-directed activity and associated psychosocial functioning (Barch and Dowd, 2010; Kring and Barch, 2014). As a Diagnostic and Statistical Manual (DSM-5) Criterion A symptom of Major Depressive Disorder, anhedonia is central to the diagnosis of depression. It is also central to the diagnosis of Schizophrenia Spectrum Disorders (i.e., Schizophrenia and Schizoaffective Disorder) as one of five core negative symptoms (APA, 2013). Furthermore, anhedonia is a risk factor for the onset of both SSDs (Gooding et al., 2005; Kwapil et al., 1997; Velthrost et al., 2009) and MDD (Bress et al., 2012; Morgan et al., 2013). Anhedonia is thus a key transdiagnostic symptom and common intervention target (Barkus and Badcock, 2019; Bedwell, Gooding, Chan, and Trachick, 2014).

Early conceptualizations considered the experience of anhedonia to be similar across SSDs and MDD. However, recent innovations in the assessment of emotion experience have painted a more complex and multifaceted picture. Specifically, assessments of "in-the-moment," or consummatory pleasure experiences (i.e., 'liking'), on the whole do not find differences in pleasant emotion experience between people with versus without SSDs. The assessment of in-situ responses to pleasant smells, images, social interactions, or other stimuli, using event-based ecological momentary assessment (EMA) and laboratory tasks, shows that people with SSDs demonstrate the capacity to experience pleasure (Blanchard et al., 1998; Burbridge and Barch, 2007; Cohen et al., 2010; Horan & Blanchard, 2003, Horan et al., 2006). However, trait-based assessments (i.e., those that ask people to report a gestalt view of their emotional experiences) continue to show general pleasure deficits across daily life in people with SSDs compared to those without (Cho, Gonzalez, Lavaysse, Pence, Fulford, & Gard, 2017; Culbreth et al., 2016; Edwards, et al., 2015, Gerritsen, 2015). Other self-report measures and lab-based studies consistently demonstrate deficits in anticipatory

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pleasure (i.e., looking forward to future events and/or feeling pleasure in anticipation of those events; also known as 'wanting') among people with SSDs (Gard, Kring, Gard, Horan, Green, 2007). Taken together, people with SSDs appear to retain the capacity to experience and report the same amount of consummatory pleasant emotions compared to people without SSDs, yet show deficits when reporting trait-based and anticipatory pleasure experiences. Thus, rather than a general hedonic deficit, low positive affect typical in SSDs may instead reflect cognitive alterations in the way hedonic experiences are stored, represented, maintained, and ultimately reported (Barch & Dowd, 2019; Kring and Barch, 2014).

There is evidence that anhedonia in the context of MDD, however, may cut across state- (consummatory and anticipatory) and trait-based pleasure experiences. Regardless of the method of assessment, people with MDD report reduced pleasure across multiple contexts (e.g., physical, interpersonal, monetary rewards; Barch, Pagliaccio, and Luking, 2015; Bylsma et al., 2008). Furthermore, there is less evidence for a contribution of impaired cognitive control systems to anhedonia in MDD, unlike in SSDs (Barch et al., 2015). These findings suggest that, unlike in SSDs, anhedonia in MDD may represent a reduced capacity to experience pleasure, more consistent with a generalized hedonic deficit.

Other innovations in the assessment of anhedonia reflect the different *types* of stimuli used to evoke emotion. The vast majority of studies assess anhedonia in the context of either physical (e.g., pleasant smells, images, sounds) or social (e.g., dynamic interpersonal interactions) stimuli. Lab-based tasks using static, sensory stimuli demonstrate consistent and robust findings of physical anhedonia among those with MDD (Barch et al., 2015; Bylsma et al., 2008) but intact affective responding among those with SSDs (Barch et al., 2015). Similarly, EMA studies assessing anhedonia in the context of real-life social interactions suggest that individuals with SSDs may demonstrate normative social pleasure in daily life (Mote and Fulford, 2020), while depressed individuals report social pleasure deficits in these contexts (Silk et al., 2011).

To date, few studies have directly compared physical and social anhedonia within or across groups. It may be that social and physical anhedonia differentially impact meaningful outcomes that could be targeted by different treatment approaches. Some recent research has found that social anhedonia, for example, is associated with functional deficits, illness severity, and other aversive outcomes such as loneliness, suicidality, and poor quality of life across both SSDs and MDD (Ritsner, Ratner, Mendyk, Gooding, 2018; Sagud et al., 2021). Furthermore, preliminary findings from neuroimaging and behavioral studies indicate blunted responses to social relative to nonsocial rewards across SSDs (Bjorkquist & Herbener, 2012; Catalano, Heerey, and Gold, 2018; Lee, Jimenez, Reavis, Horan, Wynn, & Green, 2018). Although such findings are inconsistent with previous EMA studies indicating increased positive affect in social contexts within SSDs, they suggest that at on a neural and behavioral level, anhedonia may manifest differently across social and non-social stimuli (Fulford et al., 2018).

The most commonly used measures of trait-based social and physical anhedonia are the Chapman Anhedonia Scales (Chapman et al., 1976; Eckblad et al., 1982). They were the original scales designed to examine physical (The Physical Anhedonia Scale [PAS]) and social (The Social Anhedonia Scale [SAS]) anhedonia as separate constructs. Together, the scales reflect a quick, viable means of detecting trait-level anhedonia across contexts, including over time (e.g., test-retest reliability over 6-moth intervals) and between cultures (e.g., North American and Chinese college students; Chan et al., 2015). Early studies in clinical populations showed that people with SSDs reported both physical and social anhedonia stably over time (Blanchard et al.,1998; Herbener and Harrow, 2002), while those with MDD reported anhedonia more episodically, covarying with clinical severity (Blanchard et al.,1998; Schankman et al., 2010).

Other studies highlight the potential clinical utility of the scales. Among those with SSDs, the Chapman Scales are negatively correlated with positive affect, social functioning, and pre-morbid functioning (Blanchard et al.,1998). Within MDD, higher PAS scores are associated with poorer social functioning and increased rates of rehospitalization over time (Shankman et al., 2010). Both scales are also positively correlated with lifetime suicide attempts and suicidal ideation in MDD (Sagud et al., 2020). Furthermore, psychometric assessments of the Chapman Scales found that approximately 24% of healthy individuals with elevated SAS scores endorsed an SSD diagnosis at the 10-year follow-up, compared to only 1% of those with elevated PAS scores (Chapman, Chapman, Kwapil, Eckblad, and Zinser, 1994; Kwapil, Miller, Zinser, Chapman, and Chapman, 1997; Kwapil, 1998). Thus, trait-like anhedonia, as assessed by the Chapman Scales, provides useful clinical information, such as the severity of psychopathology and related need for intervention. Furthermore, compared to the PAS, the SAS may provide greater utility as a predictor of the development of severe psychopathology over time among healthy individuals.

Despite the utility and ubiquity of the use of the Chapman Scales (as well as the differentiation between social and physical anhedonia broadly) in psychopathology research, there is limited evidence that trait-based physical and social anhedonia vary in their relative presentation in mood and psychotic disorders. Quantification of such differences may have important implications for how trait-based anhedonia contributes to psychosocial impairment, within and across disorders. For example, if trait-based social anhedonia is more pronounced in SSDs vs. controls, relative to MDD vs. controls, such impairment could reflect different biopsychosocial mechanisms that could be targeted in treatments. In other words, if social or physical anhedonia is more primary in some diagnoses, and more generalized in others, it may clarify the granularity of anhedonia, informing more precise treatment approaches. As emotion experience assessments continue to advance in differentiating between consummatory, anticipatory, and other trait-based experiences, it remains vital to understand the utility of continuing to view trait-based social and physical anhedonia as separable constructs among two of the most debilitating mental health concerns.

In the current-meta-analysis we quantified the within- and between-group (i.e., clinical vs. controls) differences in trait-like, self-reported physical and social anhedonia across MDD and SSDs, specifically, those with Schizophrenia and Schizoaffective disorder. First, we examined the magnitude of difference between each diagnostic group and controls on the Chapman Scales. Next, we quantified the discrepancies between physical and social anhedonia *within* each clinical group by examining the magnitude of difference between means scores on the Chapman Scales. We further aimed to investigate the degree to which clinical symptoms, including depression and negative symptoms, moderated between-group differences in physical and social anhedonia. Although anhedonia is considered a negative symptom, we examined interview-rated negative symptom severity as a moderator of Chapman Scale scores to identify the degree to which the scales capture variance in gold-standard clinical measures. Finally, we examined additional conditions and characteristics as moderators of such differences, including age, sex, race, and mood episode and medication status.

## 2. Methods

This review was registered on July 2020 on the Open Science Framework (OSF) with a PRISMA guided protocol: https://osf.io/pmk2c/.

### 2.1. Search strategy

We conducted two separate searches of articles published between January 1976 (the year the Chapman Anhedonia scales were published) and April 2020 using the PubMed and PsycINFO databases. We first searched for articles assessing anhedonia in MDD using the following search terms: "major depressive disorder" or "MDD" or "major depression" and "social" or "physical" and "anhedonia" or "pleasure". Next, we

searched for articles assessing anhedonia and schizophrenia using similar search terms: "schizo*" or "psychosis" and "social" or "physical" and "anhedonia" or "pleasure". We also used professional listservs to identify any unpublished data to incorporate in the analyses. We used the following inclusion criteria: (1) samples consisting of SSDs and/or MDD, (2) adults (ages 18 or older), (3) empirical study published in a peer-reviewed journal, (4) in the English language or translated, (5)

using the Chapman Anhedonia Scales, and (6) the revised version of the SAS. We restricted our search to the revised SAS as the original SAS was found to include items tapping into social anxiety, rather than social anhedonia (Eckblad, Chapman, and Chapman, 1982). We also used the following exclusion criteria: (1) systematic reviews or (2) case studies; (3) studies with participants with co-occurring substance use disorders, (4) Schizotypal Personality Disorder (5) at clinical high-risk for
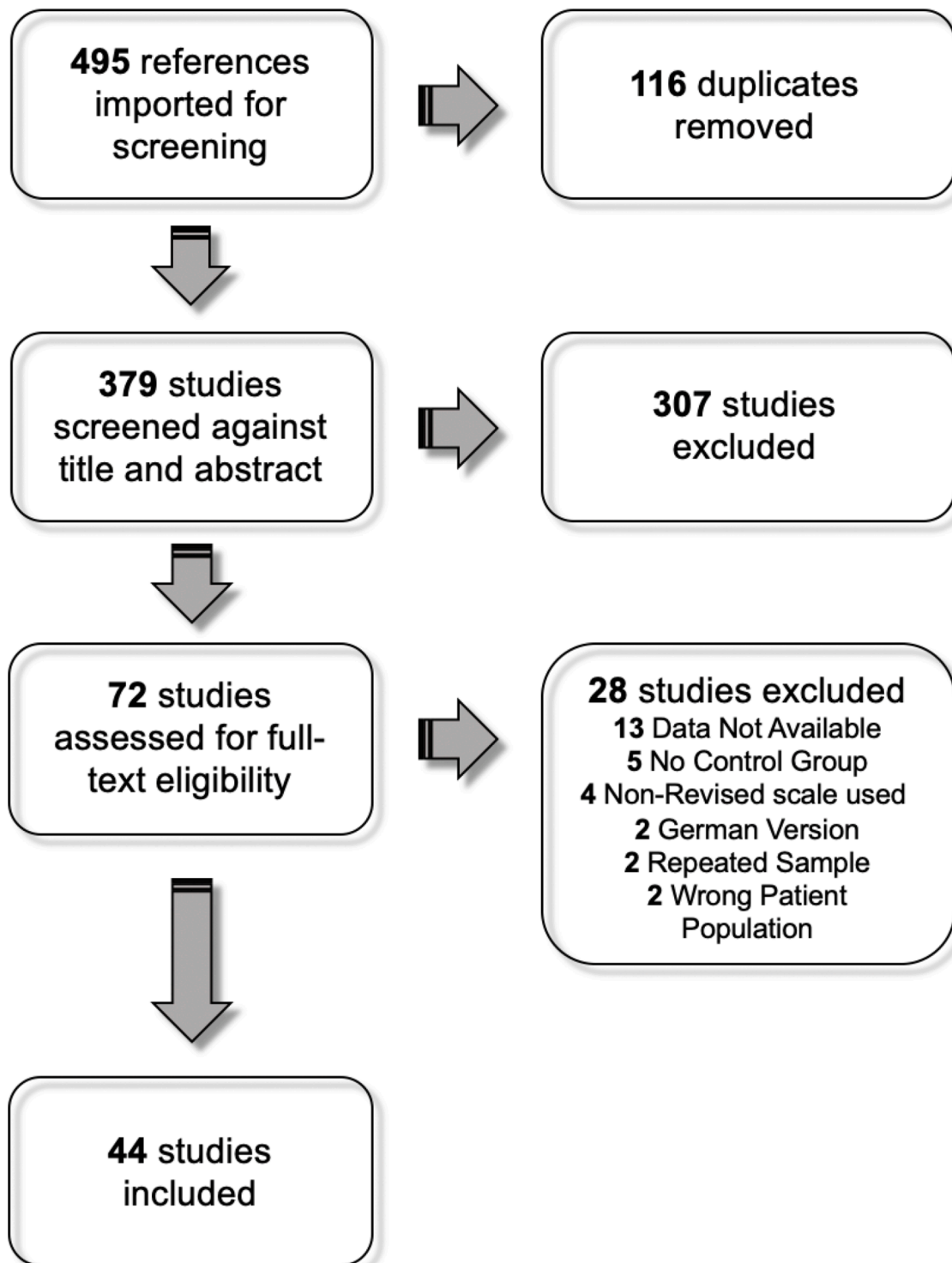
## Schizophrenia Spectrum Disorders Studies Chart



**Fig. 1.** Study Selection Process SSDs.

psychosis; (6) studies with no control group. Co-occurring substance use was excluded from the analysis as substance use may artificially impact ventral striatal and other motivation and reward pathways relevant for anhedonia. Furthermore, we only included studies using Chapman Scales with a factor structure that is congruent with the English version.

### 2.2. Data extraction & coding

We uploaded search results into the Covidence systematic review website (https://www.covidence.org). Two separate reviewers—the first-author and a trained research assistant—reviewed the abstract and full-text for both searches. Any discrepancies were discussed by both reviewers until a consensus was reached. Reviewers extracted means, standard deviations, and sample sizes for each scale across both clinical and control groups. When means or standard deviations were not available, authors were contacted for either raw data or summary scores. Reviewers extracted the following covariates: (1) mean age, (2) percentage of white participants, (3) percentage of men, (4) percentage of atypical antipsychotics prescribed to SSD samples, (5) percentage of antidepressants prescribed to MDD samples, (6) mean and standard deviation of depressive symptom severity, (7) MDE vs. MDD status, and (8) mean and standard deviation for negative symptoms within SSD samples only. To allow for pooling across various scales, negative symptom and depression scores were transformed to proportions by dividing the mean values by the total points possible on each measure, and then multiplying by 100. Authors were contacted when data were not available.

We conducted the searches in April 2020 (See Fig. 1 for flowchart of study selection and exclusion process). The SSD search yielded 495 results, of which 116 duplicates were removed, leaving 379 for review. After abstract screening and full-text review, a total of 44 studies with 44 individual effects were included for review. See Tables 1.a-b for details of study characteristics. The MDD search yielded 283 results, of which 36 duplicates were removed, leaving 247 for review. See Fig. 2 for flowchart of study selection and exclusion process. Following abstract screening and full-text review, a total of 8 studies were included for review. See Table 2 for details of study characteristics.

Finally, we used Web of Science to search for studies that cited the Chapman scales. We found 308 studies including MDD groups, and 612 studies including SSD groups. After comparing these results from our

previous PsycInfo and PubMed searches, and eliminating duplicates, we identified 117 SSD articles that were not previously screened, and 25 MDD articles that were not previously screened. After screening the title and abstract, none of these additional articles met inclusion criteria.

### 2.3. Statistical analysis

We calculated Hedge's g for differences between clinical and control groups across PAS and SAS scales by dividing the difference of clinical and control means by the pooled standard deviation. To assess the magnitude of difference between PAS and SAS within each clinical group, we calculated Cohen's d for repeated measures using the following formula (Cohen's $d_{rm}$; Cohen, 1988; Lakens, 2013):

$$\text{Cohen's } d_m = \frac{M\text{diff}}{\sqrt{SD1 \wedge 2 + SD2 \wedge 2 - 2 \times r \, SD1 \, X \, SD2}} \; X \; \sqrt{2(1-r)}$$

The numerator represents the difference of the standardized PAS and SAS means, while the denominator represents the standard deviation of differences. We used $r = 0.40$ to represent the correlation between PAS and SAS measures, as this correlation was established in several prior studies (e.g., Chapman et al., 1995; Fonesca-Pedrero et al., 2008; Kwapil et al., 2008). We then converted these values to Hedge's g to account for small sample bias. We conducted all analyses using the *dmetar* package (Harrer, Cuijpers, Furukawa, and Ebert, 2019) on R software (R Core Team, 2021). To determine model heterogeneity, we used Q and $I^2$ (Higgins et al., 2003). $I^2$ values 25% or below represent low heterogeneity, while values above 25% represent moderate to high heterogeneity (Higgins et al., 2003). When $I^2$ was greater than 25%, we ran random effects models, and for $I^2$ values below 25%, we ran fixed effects models.

For each effect, we detected outliers by searching for studies with confidence intervals (CIs) that did not overlap with the CI of the pooled effect. In other words, we defined a study as an outlier if 1) the upper bound of the 95% CI was lower than the lower bound of the pooled effect CI, or 2) if the lower bound of the 95% CI was higher than the upper bound of the pooled effect CI (Viechtbauer and Cheung, 2010). We report effects both with and without outliers included in overall pooling.

We examined the influence of negative symptoms, depressive symptoms, age, sex, race and medication status on between-group and within-group differences across both scales, by conducting mixed-effects linear regression models. For the MDD studies, we did not assess the

**Table 1a**
Study and SSD participant characteristics.

| Citation | Sample Size | | % Male | | Mean Age | | Dep. Sx. Severity | Neg. Sx. Severity |
|---|---|---|---|---|---|---|---|---|
| | SZ | CN | SZ | CN | SZ | CN | SZ | SZ |
| Barch et al., (2008) | 66 | 44 | 65 | 66 | 37.1 | 36.8 | 23.65 | 36.0 |
| Barch et al. (2014) | 59 | 39 | 57.6 | 48.7 | 39.3 | 37.4 | 18.10 | 40.0 |
| Baslet et al. (2009) | 21 | 20 | 76.2 | 60 | 36.86 | 34.4 | | |
| Berlin et al. (2011) | 38 | 32 | 66 | 66 | 36.66 | 39.19 | | 36.80 |
| Berlin et al. (1998) | 20 | 20 | 52 | 35 | 30 | 47 | 15 | 61.22 |
| Blanchard et al. (1998) | 43 | 15 | 63 | 53 | 36.14 | 36.4 | | |
| Bodapati et al. (2019) | 38 | 43 | 53 | 45 | 39.47 | 38.26 | 52.64 | |
| Burbridge and Barch (2007) | 49 | 47 | 63 | 49 | 39.4 | 37.4 | | |
| Catalano et al., (2019) | 41 | 29 | 68.3 | 69 | 38 | 39.1 | 9.30 | 35.71 |
| Cohen et al., (2005) | 73 | 22 | 75 | 68 | 41.67 | 38.95 | | |
| Ceaser and Barch (2016) | 22 | 20 | 75 | 55 | 40.41 | 33.65 | | |
| Cicero et al. (2014) | 54 | 32 | 87 | 91 | 41.46 | 43 | 47.33 | 40.62 |
| Choi et al. (2014) | 15 | 17 | 66.7 | 58.8 | 29.1 | 29.1 | | 33.47 |
| Culbreth et al. (2016) | 58 | 40 | 66.7 | 52.8 | 37 | 36.36 | 17.67 | 24.00 |
| Dowd et al. (2012) | 29 | 22 | 72 | 70 | 31.44 | 33.2 | | 59.00 |
| Fortunati et al. (2015) | 53 | 46 | 60.4 | 50 | 40.1 | 38.3 | 9.92 | 44.65 |
| Gard et al. (2007) | 28 | 29 | 64.7 | 50 | 48.06 | 46.62 | | |
| Gruzelier et al., (1993) | 7 | 11 | 42.9 | 54.5 | | | | |
| Herbener et al. (2007) | 33 | 28 | 57 | 46.4 | 37.55 | 40.82 | | 30.61 |
| Horan et al. (2006) | 30 | 31 | 83 | 68 | 46.2 | 40.5 | | |
| Horan et al. (2010) | 38 | 36 | 81.6 | 74.3 | 44.5 | 38.5 | 35.71 | 46.00 |
| Kim et al. (2019) | 23 | 23 | 47.8 | 47.8 | 33.35 | 34.04 | | 40.02 |
| Kiwanuka et al. (2014) | 100 | 78 | 68 | 70 | 41.14 | 40.68 | | 57.20 |

Note: SSD = Schizophrenia Spectrum Disorder, CN = Control, Prop. Dep. Sx. Severity = Depression Symptom Severity, Neg. Sx. Severity = Negative Symptom Severity.

**Table 1.b**
Study and SSD participant characteristics.

| Citation | Sample Size | | % Male | | Mean Age | | Dep. Sx. Severity | Neg. Sx. Severity |
|---|---|---|---|---|---|---|---|---|
| | SZ | CN | SZ | CN | SZ | CN | SZ | SZ |
| Kuha et al. (2011) | 91 | 67 | 51.6 | 49.2 | 44.5 | 47 | | |
| Lee et al. (2012) | 14 | 16 | 64.2 | 43.8 | 29.9 | 30.3 | | 39.18 |
| Lee et al. (2016) | 20 | 25 | 70 | 64 | 43.9 | 41.5 | | 34.08 |
| Li et al. (2018) | 26 | 26 | 57.7 | 57.7 | 22.77 | 24.6 | | 32.90 |
| Makowski et al. (2016) | 15 | 15 | 73.3 | 60 | 33.1 | 35.2 | 4.074 | 17.91 |
| Mann et al. (2013) | 54 | 39 | 59.3 | 48.7 | 38.85 | 36.5 | | 31.60 |
| Martin et al. (2013) | 48 | 28 | 83.3 | 92.8 | 40.78 | 43.6 | | |
| Martin et al. (2019) | 15 | 23 | 53 | 47.8 | 41.47 | 31.67 | 21.698 | |
| Olsen et al. (2015) | 25 | 33 | 0 | 0 | 41.56 | 38.39 | 16.846 | 28.25 |
| Park et al. (2015) | 20 | 20 | 50 | 40 | 28.6 | 26.1 | | 35.10 |
| Park et al. (2009) | 27 | 27 | 51.9 | 51.9 | 28.5 | 26.5 | | 41.43 |
| Trémeau et al. (2009) | 64 | 32 | 84.4 | 81.3 | 37 | 37 | 8.167 | 9.091 |
| Tremeau et al. (2014)) | 120 | 62 | 80 | 74 | 38.1 | 34.5 | 6.5 | 41.63 |
| Tso et al. (2010) | 33 | 33 | 66.7 | 69 | 38.5 | 38.2 | | 40.48 |
| Tso et al. (2014) | 39 | 36 | 58.9 | 63.8 | 41.5 | 40.4 | 18.10 | 21.63 |
| Umesh et al. (2018) | 20 | 20 | 100 | 100 | 29.8 | 29.8 | 18.095 | 42.86 |
| Waltz et al. (2015) | 42 | 44 | 76.2 | 61.4 | 37.9 | 38.6 | | 30.00 |
| Waltz et al. (2013) | 29 | 21 | 82.8 | 71.46 | 39.6 | 39.6 | | 29.00 |
| Wang et al. (2020) | 41 | 43 | 43.9 | 44.2 | 27.3 | 27.9 | 25.846 | 27.10 |
| Wynn et al. (2010) | 34 | 36 | 78.09 | 66.7 | 44.6 | 38.6 | | 47.60 |
| Zhorinitsky et al., (2012) | 23 | 25 | 60.9 | 64 | 39.9 | 40. | | 31.83 |
| Zou et al. (2018) | 146 | 73 | 56.2 | 49.3 | 36.29 | 36.0 | 25.302 | 38.14 |

Note: SSD = Schizophrenia Spectrum Disorder, CN = Control, Dep. Sx. Severity = Depression Symptom Severity, Neg. Sx. Severity = Negative Symptom Severity

influence of remitted MDD vs. current MDD, race, or antidepressants as a moderator as there was not a sufficient number of studies providing the necessary data. Finally, we examined publication bias through funnel plots and Egger's tests for each pooled effect. When interpreting funnel plots, we examined the extent to which the studies evenly scattered on either side of the overall effect. An uneven scattering of studies may suggest the presence of publication bias.

## 3. Results

### 3.1. Schizophrenia spectrum disorders

Analyses of PAS between SSD and controls yielded a large effect ($g = 0.89$; 95% CI: 0.78–1.00; $t = 16.12$; $p < 0.0001$), indicating that individuals with SSD self-reported greater physical anhedonia than controls. This effect increased to 0.97 (95% CI: 0.88 – 1.10; $t = 22.94$; $p < 0.0001$) after removing four outliers (Baslet et al., 2009; Burbridge et al., 2007; Makowski et al., 2016; Zou et al., 2018). Group differences on SAS yielded a similarly large effect ($g = 0.93$; 95% CI: 0.67 – 1.18; $t = 7.32$, $p < 0.0001$), demonstrating that individuals with SSD self-reported greater social anhedonia than controls. We detected one extreme outlier (Umesh et al., 2018), which yielded an effect size of 8.7. Upon further investigation of this study, we found that standard deviations for both controls and SSDs were extremely low (i.e., 0.97 and 1.40, respectively). Nevertheless, after removing this outlier, the group difference in SAS remained large ($g = 0.84$; 95% CI: 0.76 – 0.92; $t = 20.80$; $p < 0.0001$). The Q statistic was significant for each effect suggesting heterogeneity beyond sampling error. $I^2$ values indicate that less than 60% of the observed variance in each effect was due to sampling error (see Table 3). Mixed effects meta-regression models did not indicate any significant moderators across either effect.

Within-group differences between PAS and SAS in the SSD studies were pooled through a fixed effects model. This model yielded a moderate effect ($g = -0.50$; 95% CI: −0.83 – −0.17; $z = -3.00$; $p < 0.0001$). This suggests that SAS scores were significantly higher than PAS scores across SSD studies. The Q statistic was not significant, and $I^2 = 0.0\%$, suggesting no heterogeneity in the effects beyond sampling error (see Table 3). The effect size increased to −0.58 (95% CI: −0.92 – −0.25; $z = -3.45$; $p < 0.001$) after removing one outlier (Makowski et al., 2016). Mixed effects models indicated a significant effect of age on within-group differences between SAS and PAS, such that older

individuals demonstrated smaller discrepancies between physical and social anhedonia compared to younger individuals. The remaining moderators were not significant. Of note, the average standardized score (0–100) for negative symptoms was 36.52.

Fig. 3-8

Visual inspection of funnel plots and Egger's test revealed asymmetry and publication bias for the between-group effect of social anhedonia ($t(44) = 2.60$, $p = 0.01$), and the within-group difference between PAS and SAS ($t(41) = -2.09$, $p = 0.04$). For the between group effect, asymmetry appeared to be primarily driven by the Umesh et al. (2018) study. For the within-group effect, asymmetry appeared to be driven by the Makowski et al. (2016) study. We did not find asymmetry or publication bias for the between-group effect of physical anhedonia ($t(41) = 1.42$, $p = 0.16$) (see Figs. 9-11).

### 3.2. Major depressive disorder

Group differences in PAS scores between MDD and controls yielded a large effect of 1.18 (95% CI: 0.91 – 1.44; $z = 8.68$; $p < 0.0001$), indicating that individuals with MDD report greater physical anhedonia than controls. There was also a large aggregated group difference in SAS scores ($g = 1.32$; 95% CI: 1.04 –1.61; $z = 9.04$; $p < 0.0001$), indicating greater self-reported social anhedonia in MDD compared to controls. Finally, the mean difference between PAS and SAS within MDD was not significant ($g = -0.72$; 95% CI: −2.9 – 1.47; $z = -0.64$; $p < 0.52$), indicating that individuals with MDD report similar social and physical anhedonia. No outliers were detected for any of these effects. Q statistics suggested no heterogeneity beyond sampling error. $I^2$ values indicated that less than 40% of the observed variance in each effect was due to sampling error (see Table 4).

Mixed effects models indicated significant effect of depressive symptoms, such that greater symptoms were associated with a larger difference in PAS scores between those with MDD and controls. The remaining moderators of the MDD-Control group differences and within-group MDD differences were not significant. Funnel plots and Egger's tests did not indicate asymmetry or publication bias for any effect. However, because the sample size is small (8 studies), the Egger's test may lack statistical power to detect significance (see Figs. 12-14).

Although underpowered (five studies each), we conducted two final analyses of studies that compared the Chapman Scales between MDD and SSDs directly (Berlin, Givry-Steiner, Lecrubier and Puech, 1998;

## Major Depression Disorder Studies Chart
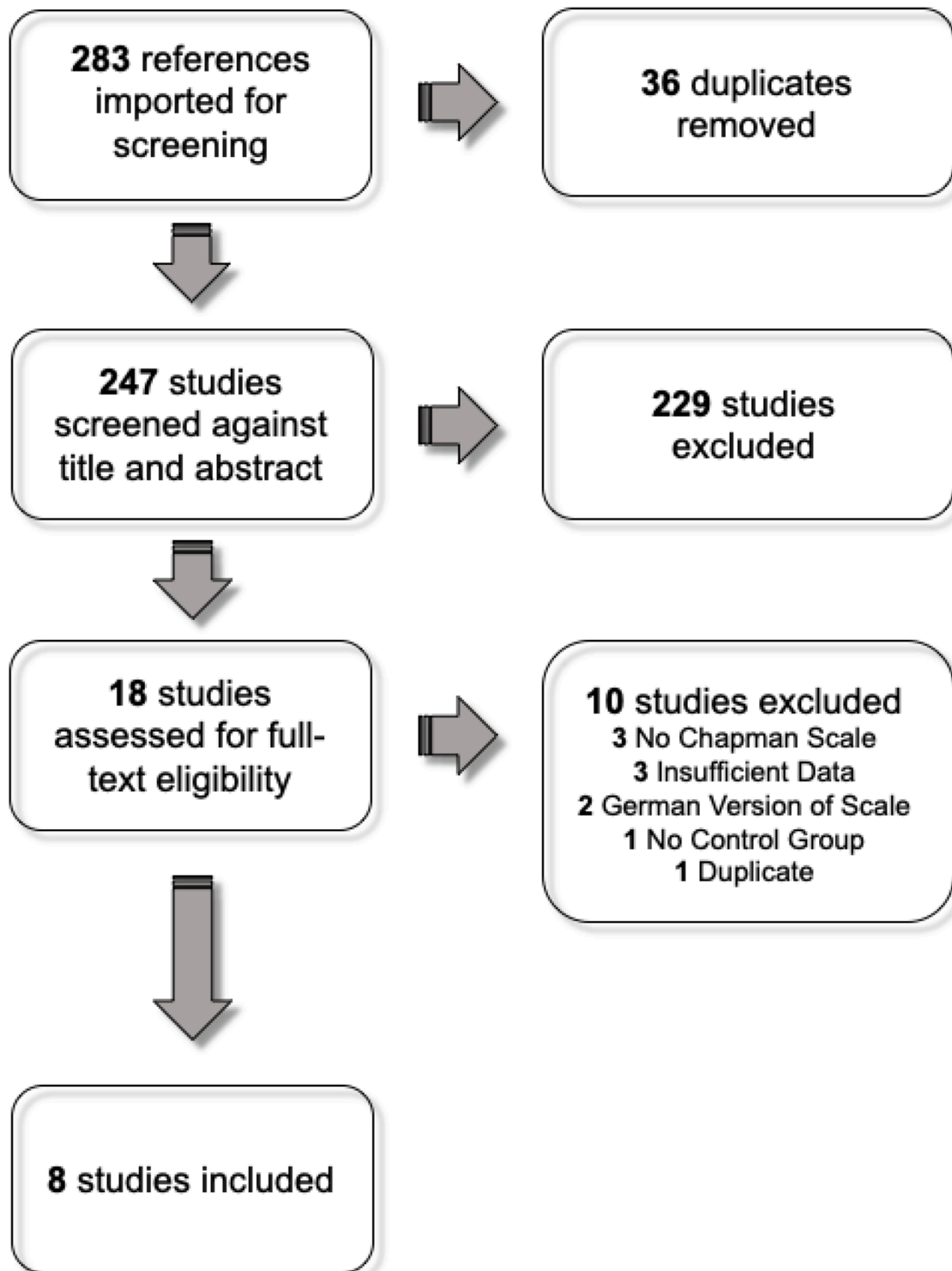


**Fig. 2.** Study Selection Process MDD.

Chuang et al., 2014; Olsen, Bjorkquist, Bodapati, Shankman and Herbener, 2015; Pelizza & Ferarri, 2009; Wang et al., 2020) Group differences indicate nonsignificantly elevated scores among those with MDD compared to those with SSDs on both PAS ($g = 0.15$, $p = 0.17$) and SAS ($g = 0.03$, $p = 0.78$). Across both meta-analyses, two studies showed negligible differences between the groups (Berlin et al. 1998; Wang et al., 2020) two other studies showed moderate negative differences (i. e., SSDs elevated relative to MDD; Chuang et al., 2014; Olsen et al., 2015), and one study showed a large positive effect (i.e., MDD elevated relative to SSDs; Pelizza and Ferarri, 2009). The overall positive SMDs were likely driven by the one moderate positive effect, suggesting that the analysis is too underpowered to yield a meaningful interpretation.

**Table 2**
Study and MDD participant characteristics.

| Citation | Sample Size | | % Male | | Mean Age | | Dep. Sx. Severity |
|---|---|---|---|---|---|---|---|
| | MDD | CN | MDD | CN | MDD | CN | MDD |
| Berlin et al. (1998) | 20 | 20 | 35 | 35 | 47 | 47 | 42 |
| Chuang et al. (2014) | 24 | 20 | 70 | 80 | 33.08 | 34.3 | 5.96 |
| Naudin et al. (2014) | 22 | 41 | 27 | 41 | 33.2 | 34 | 37.1 |
| Naudin et al. (2015) | 20 | 24 | 20 | 38 | 64.9 | 67.4 | 29.12 |
| Savitz et al. (2013) | 10 | 26 | 20 | 42.3 | 38.2 | 34.5 | 24.3 |
| Olsen et al. (2015) | 18 | 33 | 0 | 0 | 35.94 | 38.39 | 15 |
| Sczepanik et al. (2017) | 21 | 23 | 61.9 | 56.5 | 35.5 | 31.8 | 32.7 |
| Wang et al. (2020) | 44 | 43 | 34 | 44.1 | 28.66 | 27.93 | 15.11 |

Note: MDD = Major Depression Disorder, CN = Control, Dep. Sx. Severity = Depression Symptom Severity.

**Table 3**
Results of meta-analyses and meta-regression analyses: SSDs .

| SSD | Hedge's G | 95%CI | Q | $I^2$ | p |
|---|---|---|---|---|---|
| Physical Anhedonia | 0.89 *** | 0.78 \| 1.0 | 83.9 | 51.2% | <0.0001 |
| Social Anhedonia | 0.93*** | 0.67 \| 1.19 | 101.3 | 56.6% | <0.0001 |

| Moderators (Phys\|Soc) | β | SE | 95%CI Low | 95%CI Hi | p |
|---|---|---|---|---|---|
| Age | <0.01\|− | 0.01\|0.03 | −0.02\|− | 0.03\| | 0.72\| |
| %Male | 0.01 | <0.01\| | 0.07 | 0.04 | 0.57 |
| %white | <0.01\| | 0.01 | −0.01\|− | 0.01\| | 0.74\| |
| %Atypical AP | 0.01 | <0.01\|< | 0.01 | 0.03 | 0.18 |
| NegSx | −0.01\|− | 0.01 | −0.01\|− | 0.004\| | 0.30\| |
| DepSx | 0.01 | 0.004\| | 0.01 | 0.01 | 0.80 |
|  | −0.01\|− | 0.02 | −0.01\|− | 0.002\| | 0.12\| |
|  | 0.02 | 0.01\|0.02 | 0.06 | 0.01 | 0.20 |
|  | 0.01\|− | 0.01\|0.03 | −0.01\|− | 0.02\| | 0.15\| |
|  | 0.02 |  | 0.05 | 0.02 | 0.39 |
|  | −0.01\| |  | −0.02\|− | 0.01\| | 0.37\| |
|  | 0.04 |  | 0.02 | 0.09 | 0.21 |

| | Hedge's G | 95%CI | Q | $I^2$ | p |
|---|---|---|---|---|---|
| Phys. vs. Soc. within-group | −0.50** | −0.83\|−0.17 | 26.0 | 0.0% | <0.01 |

| Moderators | β | SE | 95%CI | p |
|---|---|---|---|---|
| Age* | −0.08 | 0.03 | −0.14\|−0.02 | <0.05 |
| %Male | <−0.01 | 0.01 | 0.02 | 0.66 |
| %White | 0.005 | 0.02 | −0.03\|0.03 | 0.74 |
| % Atypical AP | <0.01 | 0.01 | 0.02 | 0.60 |
| NegSx | <0.001 | 0.01 | −0.03\|0.03 | 0.97 |
| DepSx | −0.03 | 0.02 | 0.04 | 0.15 |
|  |  |  | −0.02\|0.03 |  |
|  |  |  | −0.03\|0.03 |  |
|  |  |  | −0.08\|0.01 |  |

Note: Atypical AP= Atypical Antipsychotic, NegSx= Negative Symptom Severity, DepSx=Depressive Symptom Severity.

For more information, please see our OSF page https://osf.io/m7fhw/)

## 4. Discussion

We conducted a meta-analytic review of trait-level, self-reported physical and social anhedonia across MDD and SSDs. We quantified the magnitude of physical and social anhedonia between controls and clinical groups, as well as between facets of anhedonia within groups, and determined the extent to which clinical and demographic characteristics moderated effects. As predicted, people with SSDs and MDD reported significantly higher trait-level physical and social anhedonia than controls, reflected in large between-group effect sizes. These findings demonstrate a high degree of self-reported physical and social anhedonia across the published literature in clinical populations characterized by deficits in the experience of trait-based pleasure.

Elevated Chapman Scale scores across MDD and SSDs indicate that although anhedonia severity in MDD may be more state-like than in SSDs, both groups experience at least some degree of anhedonia in stable manner. In MDD, anhedonia may be more pronounced during periods of low mood, while in SSDs anhedonia may be elevated regardless of mood state; this suggests that although both groups experience elevated anhedonia, it may be of a different nature. Our results further demonstrated that trait-based social anhedonia may be more severe than physical anhedonia within SSDs, but similar across the social and physical domains within MDD. This suggests that people with SSDs endorse greater challenges experiencing pleasure from social rewards as opposed to non-social rewards, and thus provides preliminary support for treating trait-based physical and social anhedonia as distinct constructs. Elevated social anhedonia relative to physical anhedonia in SSDs aligns with previous lab-based elicitation tasks, demonstrating blunted in-the moment hedonic responses to social relative to nonsocial stimuli in SSD (Bjorkquist & Herbener, 2012; Catalano et al., 2018; Lee, Jimenez, Reavis, Horan, Wynn, & Green, 2018). In contrast, these studies are inconsistent with EMA reported findings of elevated positive affect in social contexts (Mote and Fulford, 2020). Future studies should re-examine potential differences in social and nonsocial (i.e., physical or monetary) stimuli across psychopathology to determine whether elevated social anhedonia (relative to nonsocial anhedonia) is specific to psychotic illnesses or is a transdiagnostic phenomenon.

Several challenges limit how we interpret the above findings. Within SSDs, elevated SAS relative to PAS may be driven by limited opportunities for social contact, rather than limited social hedonic capacity. Items such as "I don't really feel very close to my friends" and "I like to make long distance phone calls to friends and relatives" often depend on the presence of recent interactions and ongoing relationships, which are consistently low in people with SSDs (Gayer-Anderson & Morgan, 2013) and MDD (Kupferberg, 2016). Indeed, reduced social network size, and a lower likelihood of being in romantic partnerships (Gayer-Anderson & Morgan, 2013; Kupferberg, 2016) is common in both these populations. Elevated trait-level social anhedonia may reflect impoverished social environments rather than dampened capacity to experience social pleasure. This may further clarify why EMA studies, which probe participants to consider in-the-moment experiences, indicate intact social pleasure (Mote and Fulford, 2020). A critical future direction is to examine the extent to which frequency of social contact is related to trait-based reports of social anhedonia.

In this study, limited social opportunities may also explain the relatively higher social anhedonia in the SSDs group but not in the MDD group. Although typically reduced in both groups, limited social opportunities may be even more pronounced in those with SSDs compared to those with MDD, since SSDs symptoms are more chronic and enduring. It is also important to note that many items in the SAS tap into asociality (i.e., another negative symptom describing the reduction of social initiation due to decreased interest in forming close relationships with others), which is a related, but distinct construct that typically captures attitudes and behavior arising from anhedonia, rather than the experience of anhedonia itself. Similarly—in-line with burgeoning research—elevated SAS may arise from challenges associated poor social cognition, and challenges with translating the desire for social affiliation to actual social behavior (Barch et al., 2015; Fulford et al., 2018).

Furthermore, consistent with most trait-level self-report scales, the Chapman Scales require individuals to reflect on past experiences that, due to retrospective recall biases, may lead people to report
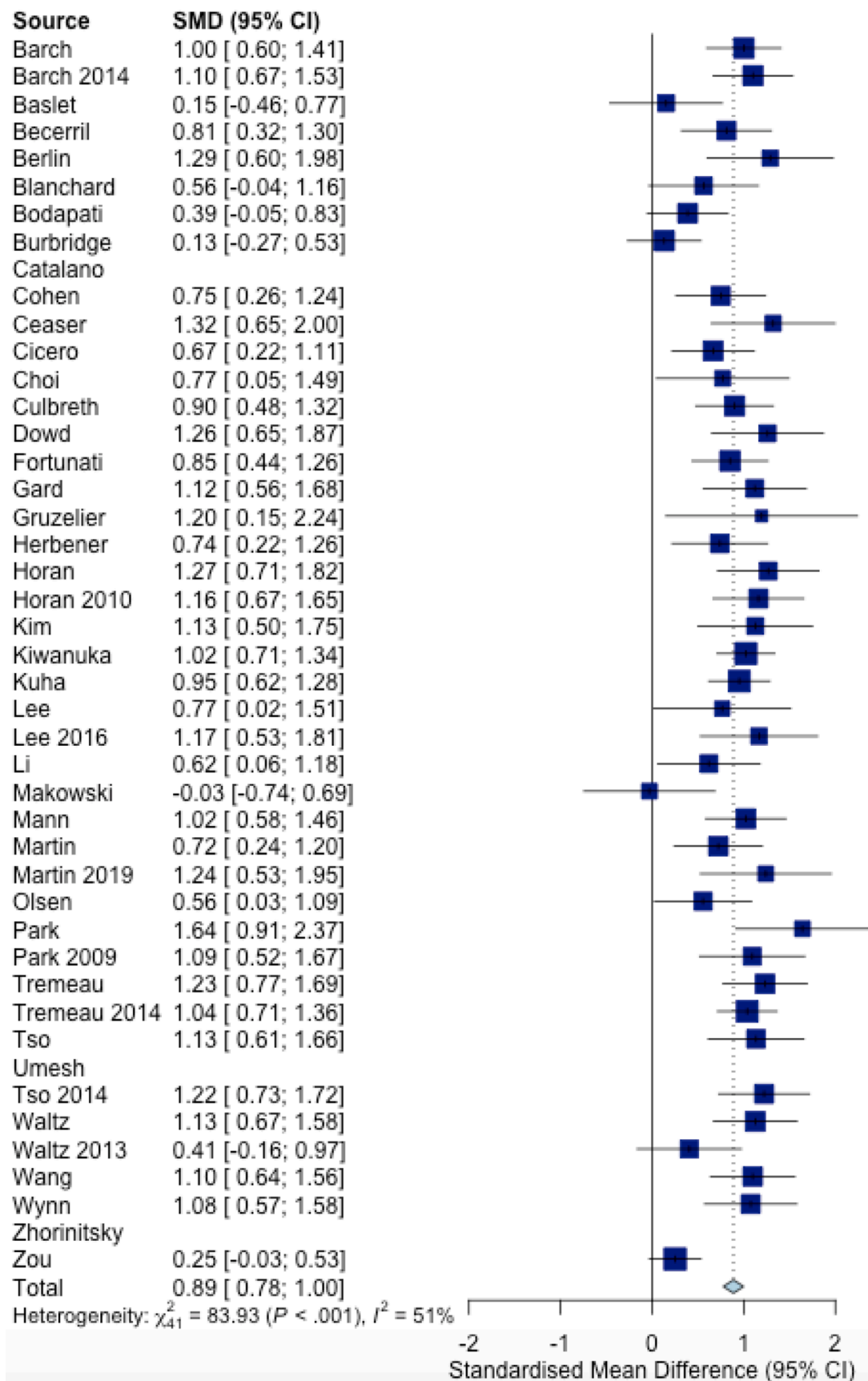
| Source | SMD (95% CI) |
|---|---|
| Barch | 1.00 [ 0.60; 1.41] |
| Barch 2014 | 1.10 [ 0.67; 1.53] |
| Baslet | 0.15 [-0.46; 0.77] |
| Becerril | 0.81 [ 0.32; 1.30] |
| Berlin | 1.29 [ 0.60; 1.98] |
| Blanchard | 0.56 [-0.04; 1.16] |
| Bodapati | 0.39 [-0.05; 0.83] |
| Burbridge | 0.13 [-0.27; 0.53] |
| Catalano | |
| Cohen | 0.75 [ 0.26; 1.24] |
| Ceaser | 1.32 [ 0.65; 2.00] |
| Cicero | 0.67 [ 0.22; 1.11] |
| Choi | 0.77 [ 0.05; 1.49] |
| Culbreth | 0.90 [ 0.48; 1.32] |
| Dowd | 1.26 [ 0.65; 1.87] |
| Fortunati | 0.85 [ 0.44; 1.26] |
| Gard | 1.12 [ 0.56; 1.68] |
| Gruzelier | 1.20 [ 0.15; 2.24] |
| Herbener | 0.74 [ 0.22; 1.26] |
| Horan | 1.27 [ 0.71; 1.82] |
| Horan 2010 | 1.16 [ 0.67; 1.65] |
| Kim | 1.13 [ 0.50; 1.75] |
| Kiwanuka | 1.02 [ 0.71; 1.34] |
| Kuha | 0.95 [ 0.62; 1.28] |
| Lee | 0.77 [ 0.02; 1.51] |
| Lee 2016 | 1.17 [ 0.53; 1.81] |
| Li | 0.62 [ 0.06; 1.18] |
| Makowski | -0.03 [-0.74; 0.69] |
| Mann | 1.02 [ 0.58; 1.46] |
| Martin | 0.72 [ 0.24; 1.20] |
| Martin 2019 | 1.24 [ 0.53; 1.95] |
| Olsen | 0.56 [ 0.03; 1.09] |
| Park | 1.64 [ 0.91; 2.37] |
| Park 2009 | 1.09 [ 0.52; 1.67] |
| Tremeau | 1.23 [ 0.77; 1.69] |
| Tremeau 2014 | 1.04 [ 0.71; 1.36] |
| Tso | 1.13 [ 0.61; 1.66] |
| Umesh | |
| Tso 2014 | 1.22 [ 0.73; 1.72] |
| Waltz | 1.13 [ 0.67; 1.58] |
| Waltz 2013 | 0.41 [-0.16; 0.97] |
| Wang | 1.10 [ 0.64; 1.56] |
| Wynn | 1.08 [ 0.57; 1.58] |
| Zhorinitsky | |
| Zou | 0.25 [-0.03; 0.53] |
| Total | 0.89 [ 0.78; 1.00] |

Heterogeneity: $\chi^2_{41} = 83.93$ ($P < .001$), $I^2 = 51\%$

Standardised Mean Difference (95% CI)

**Fig. 3.** Forest Plot of differences in physical anhedonia between SZ & controls.

| Source | SMD (95% CI) |
|---|---|
| Barch | 0.66 [ 0.27; 1.05] |
| Barch 2014 | 0.89 [ 0.47; 1.32] |
| Baslet | 0.54 [-0.09; 1.16] |
| Becerril | 1.18 [ 0.67; 1.70] |
| Berlin | 0.87 [ 0.21; 1.52] |
| Blanchard | 1.04 [ 0.42; 1.66] |
| Bodapati | 0.47 [ 0.03; 0.92] |
| Burbridge | 0.37 [-0.03; 0.77] |
| Catalano | 0.75 [ 0.26; 1.24] |
| Cohen | 0.61 [ 0.12; 1.09] |
| Ceaser | 1.30 [ 0.63; 1.97] |
| Cicero | 0.72 [ 0.27; 1.17] |
| Choi | 0.32 [-0.38; 1.02] |
| Culbreth | 1.01 [ 0.58; 1.43] |
| Dowd | 0.68 [ 0.11; 1.25] |
| Fortunati | 0.85 [ 0.43; 1.26] |
| Gard | 1.18 [ 0.61; 1.74] |
| Gruzelier | 1.02 [ 0.00; 2.04] |
| Herbener | 0.86 [ 0.34; 1.39] |
| Horan | 1.07 [ 0.53; 1.61] |
| Horan 2010 | 1.23 [ 0.73; 1.73] |
| Kim | 0.95 [ 0.34; 1.56] |
| Kiwanuka | 0.77 [ 0.46; 1.07] |
| Kuha | 0.98 [ 0.65; 1.32] |
| Lee | 0.40 [-0.33; 1.12] |
| Lee 2016 | 0.93 [ 0.31; 1.56] |
| Li | 0.62 [ 0.07; 1.18] |
| Makowski | 0.60 [-0.13; 1.34] |
| Mann | 0.61 [ 0.19; 1.03] |
| Martin | 0.76 [ 0.27; 1.24] |
| Martin 2019 | 1.27 [ 0.55; 1.98] |
| Olsen | 0.62 [ 0.09; 1.15] |
| Park | 0.88 [ 0.23; 1.54] |
| Park 2009 | 1.10 [ 0.53; 1.68] |
| Tremeau | 1.03 [ 0.58; 1.48] |
| Tremeau 2014 | 0.59 [ 0.28; 0.90] |
| Tso | 1.14 [ 0.61; 1.66] |
| Tso 2014 | 1.06 [ 0.57; 1.54] |
| Waltz | 0.63 [ 0.20; 1.07] |
| Waltz 2013 | 0.30 [-0.27; 0.86] |
| Wang | 1.32 [ 0.85; 1.79] |
| Wynn | 1.32 [ 0.80; 1.84] |
| Zhorinitsky | 0.83 [ 0.24; 1.43] |
| Zou | 0.97 [ 0.68; 1.27] |
| Total | 0.84 [ 0.76; 0.92] |

Heterogeneity: $\chi^2_{43} = 47.56$ ($P = .29$), $I^2 = 10\%$

Standardised Mean Difference (95% CI)



**Fig. 4.** Forest Plot of differences in social anhedonia between SZ & controls. One outlier (Umesh et al.) removed from plot. .

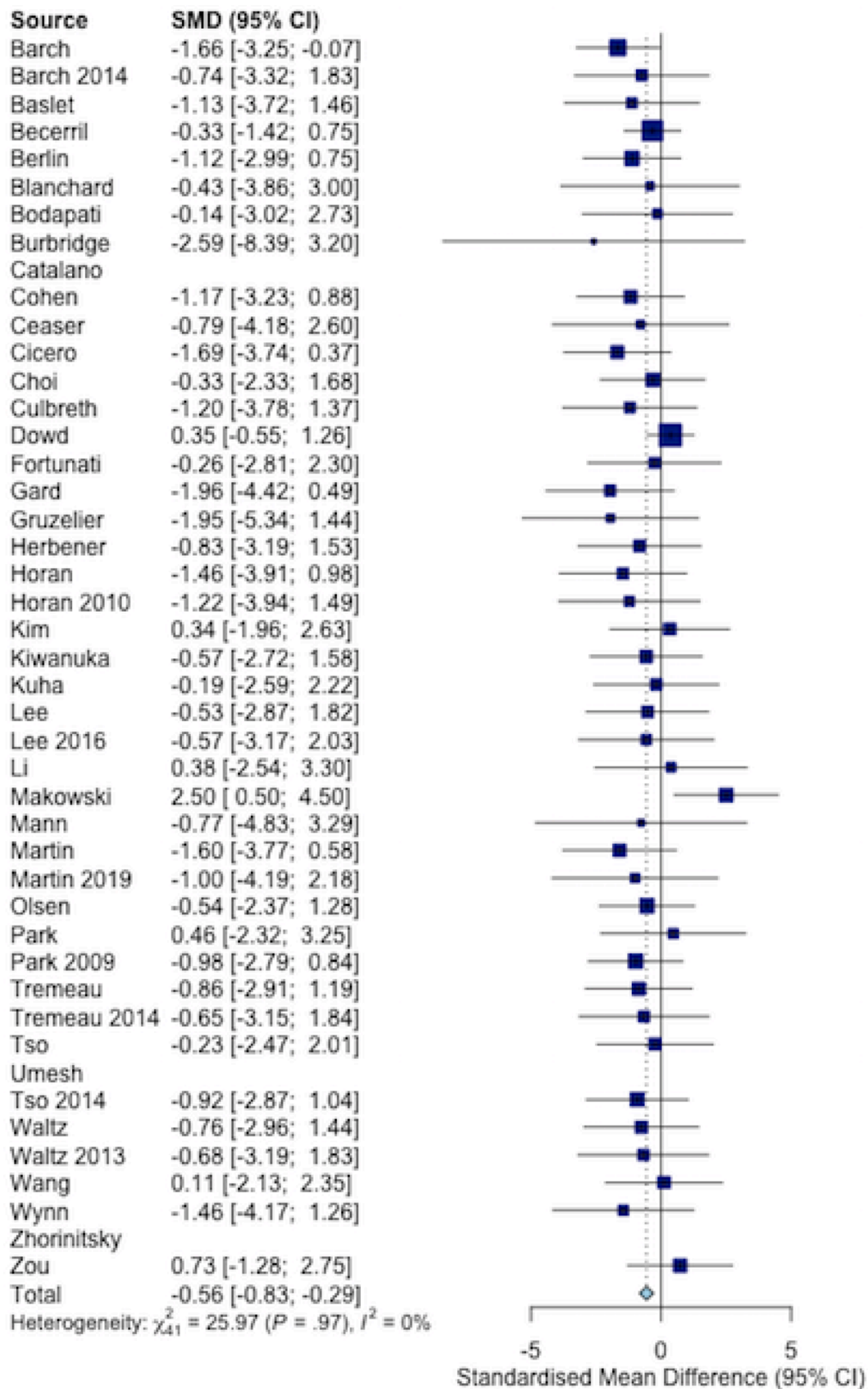| Source | SMD (95% CI) |
|---|---|
| Barch | -1.66 [-3.25; -0.07] |
| Barch 2014 | -0.74 [-3.32; 1.83] |
| Baslet | -1.13 [-3.72; 1.46] |
| Becerril | -0.33 [-1.42; 0.75] |
| Berlin | -1.12 [-2.99; 0.75] |
| Blanchard | -0.43 [-3.86; 3.00] |
| Bodapati | -0.14 [-3.02; 2.73] |
| Burbridge | -2.59 [-8.39; 3.20] |
| Catalano | |
| Cohen | -1.17 [-3.23; 0.88] |
| Ceaser | -0.79 [-4.18; 2.60] |
| Cicero | -1.69 [-3.74; 0.37] |
| Choi | -0.33 [-2.33; 1.68] |
| Culbreth | -1.20 [-3.78; 1.37] |
| Dowd | 0.35 [-0.55; 1.26] |
| Fortunati | -0.26 [-2.81; 2.30] |
| Gard | -1.96 [-4.42; 0.49] |
| Gruzelier | -1.95 [-5.34; 1.44] |
| Herbener | -0.83 [-3.19; 1.53] |
| Horan | -1.46 [-3.91; 0.98] |
| Horan 2010 | -1.22 [-3.94; 1.49] |
| Kim | 0.34 [-1.96; 2.63] |
| Kiwanuka | -0.57 [-2.72; 1.58] |
| Kuha | -0.19 [-2.59; 2.22] |
| Lee | -0.53 [-2.87; 1.82] |
| Lee 2016 | -0.57 [-3.17; 2.03] |
| Li | 0.38 [-2.54; 3.30] |
| Makowski | 2.50 [0.50; 4.50] |
| Mann | -0.77 [-4.83; 3.29] |
| Martin | -1.60 [-3.77; 0.58] |
| Martin 2019 | -1.00 [-4.19; 2.18] |
| Olsen | -0.54 [-2.37; 1.28] |
| Park | 0.46 [-2.32; 3.25] |
| Park 2009 | -0.98 [-2.79; 0.84] |
| Tremeau | -0.86 [-2.91; 1.19] |
| Tremeau 2014 | -0.65 [-3.15; 1.84] |
| Tso | -0.23 [-2.47; 2.01] |
| Umesh | |
| Tso 2014 | -0.92 [-2.87; 1.04] |
| Waltz | -0.76 [-2.96; 1.44] |
| Waltz 2013 | -0.68 [-3.19; 1.83] |
| Wang | 0.11 [-2.13; 2.35] |
| Wynn | -1.46 [-4.17; 1.26] |
| Zhorinitsky | |
| Zou | 0.73 [-1.28; 2.75] |
| Total | -0.56 [-0.83; -0.29] |

Heterogeneity: $\chi^2_{41} = 25.97$ ($P = .97$), $I^2 = 0\%$

**Fig. 5.** Forest Plot of differences between social and physical anhedonia within SZ.

| Source | SMD (95% CI) |
|---|---|
| Berlin et al | 1.75 [ 1.01; 2.49] |
| Chuang et al | 1.38 [ 0.71; 2.05] |
| Naudin et al | 1.28 [ 0.71; 1.85] |
| Naudin et al | 0.93 [ 0.30; 1.55] |
| Savitz et al | 1.69 [ 0.85; 2.53] |
| Olsen et al | 0.52 [-0.07; 1.10] |
| Szczepanik et al | 1.28 [ 0.62; 1.93] |
| Wang et al | 1.04 [ 0.59; 1.48] |
| Total | 1.18 [ 0.91; 1.44] |
| 95% PI | [ 0.56; 1.79] |

Heterogeneity: $\chi^2_7 = 10.18$ ($P = .18$), $I^2 = 31\%$

Standardised Mean Difference (95% CI)
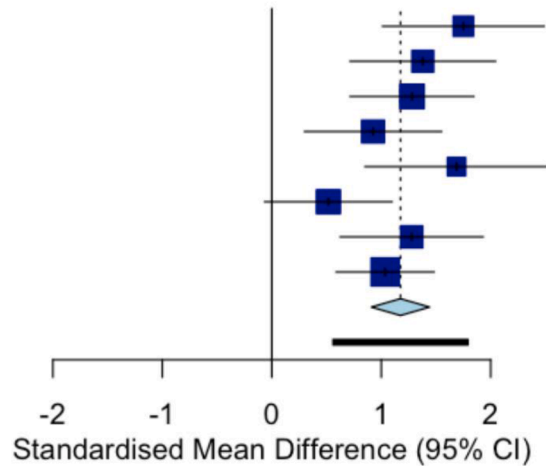
**Fig. 6.** Forest Plot of Differences in Physical Anhedonia Between MDD and Controls.

| Source | SMD (95% CI) |
|---|---|
| Berlin et al | 0.87 [0.21; 1.52] |
| Chuang et al | 1.11 [0.47; 1.75] |
| Naudin et al | 1.73 [1.12; 2.33] |
| Naudin et al | 1.20 [0.55; 1.85] |
| Savitz et al | 2.13 [1.23; 3.02] |
| Olsen et al | 1.17 [0.55; 1.79] |
| Szczepanik et al | 1.87 [1.15; 2.59] |
| Wang et al | 1.01 [0.57; 1.46] |
| Total | 1.32 [1.04; 1.61] |
| 95% PI | [0.60; 2.05] |

Heterogeneity: $\chi^2_7 = 11.45$ ($P = .12$), $I^2 = 39\%$

Standardised Mean Difference (95% CI)
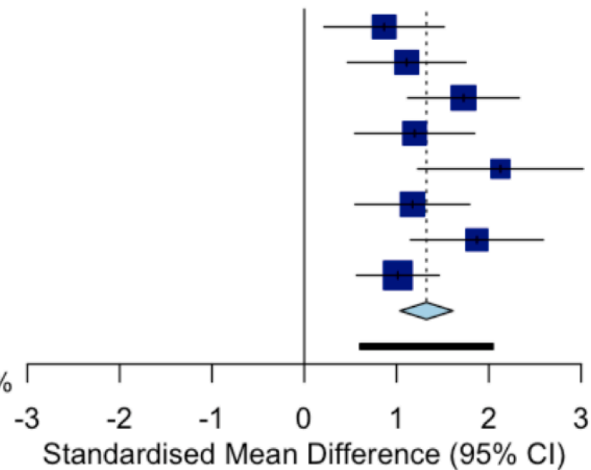
**Fig. 7.** Forest plot of differences on Social Anhedonia between MDD and HC.

| Source | SMD (95% CI) |
|---|---|
| Berlin et al | 0.13 [-5.24; 5.51] |
| Chuang et al | -0.66 [-9.46; 8.15] |
| Naudin et al | -0.71 [-6.55; 5.13] |
| Naudin et al | -0.96 [-6.86; 4.93] |
| Savitz et al | -1.23 [-8.03; 5.57] |
| Olsen et al | -1.46 [-6.77; 3.84] |
| Szczepanik et al | -1.33 [-9.15; 6.49] |
| Wang et al | 0.10 [-5.84; 6.04] |
| Total | -0.72 [-2.92; 1.47] |
| 95% PI | [-3.46; 2.02] |

Heterogeneity: $\chi^2_7 = 0.30$ ($P > .99$), $I^2 = 0\%$

Standardised Mean Difference (95% CI)
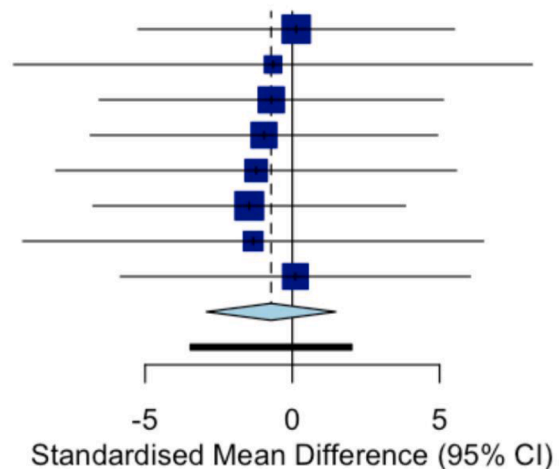
**Fig. 8.** Forest plot of Differences Between Physical & Social Anhedonia within MDD.
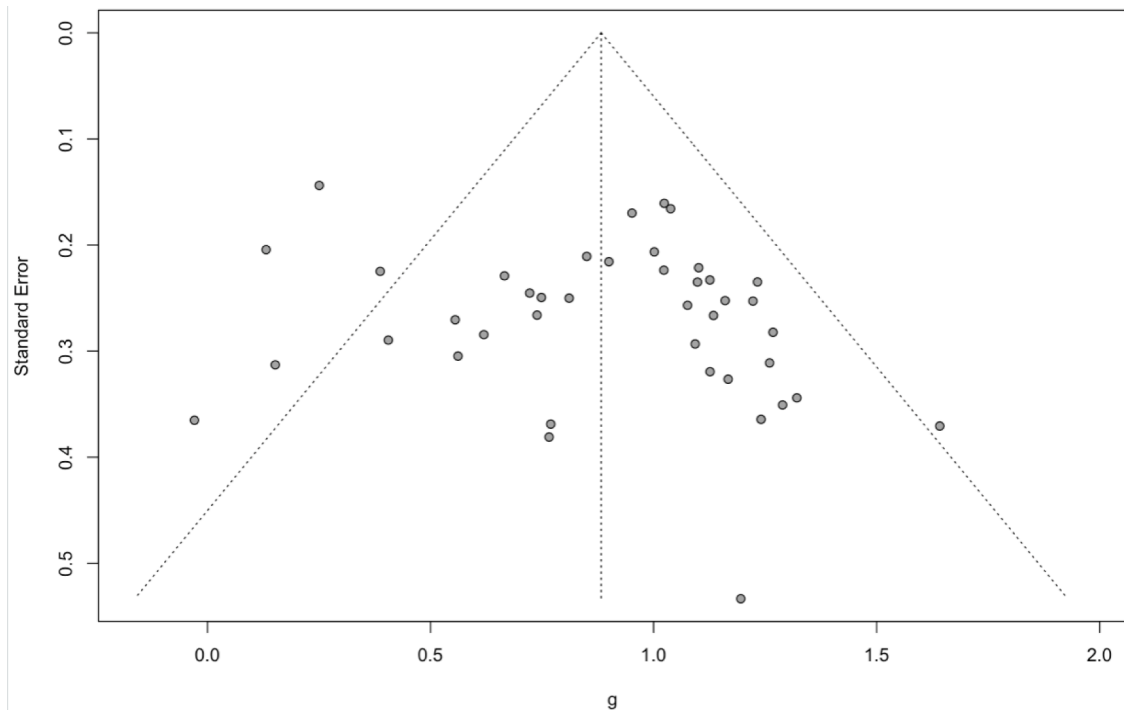
**Fig. 9.** Funnel Plot of Differences in Physical Anhedonia Between SSDs and Controls.
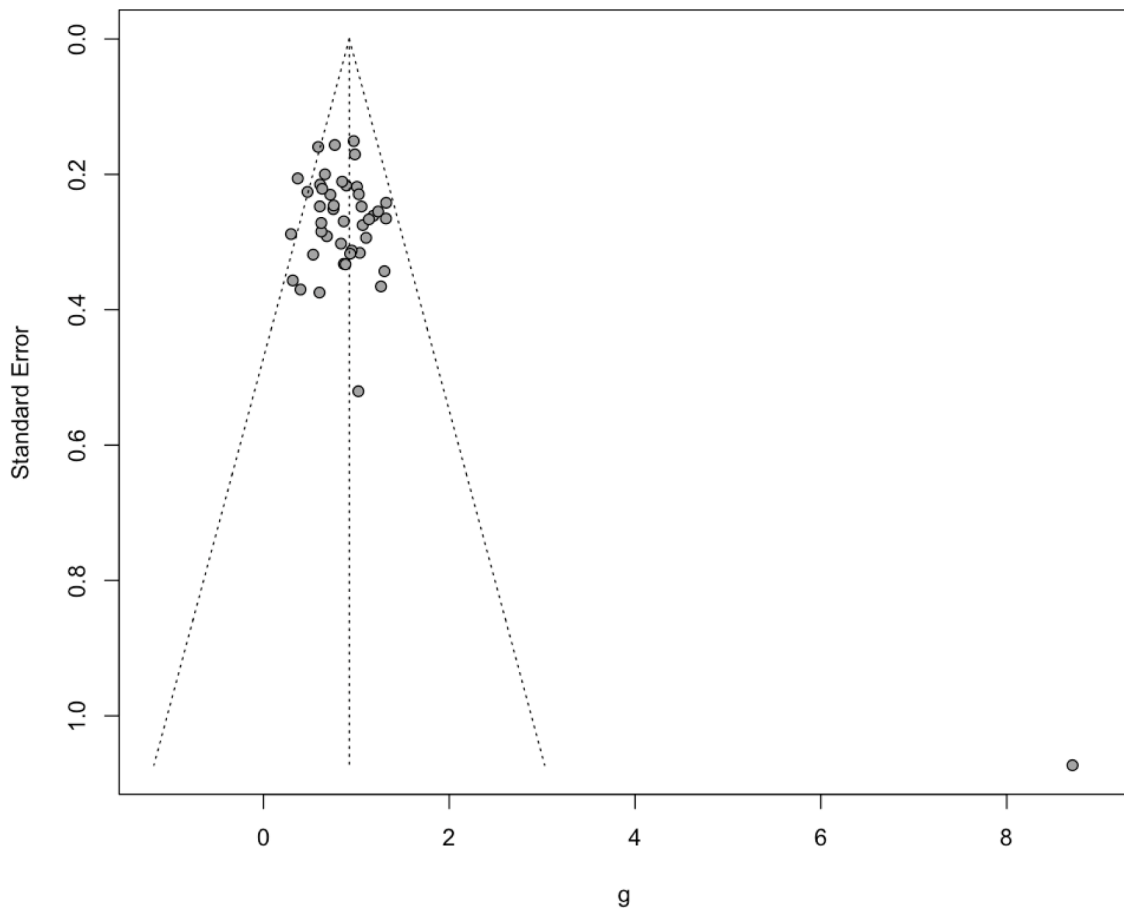


**Fig. 10.** Funnel Plot of Differences in Social Anhedonia Between SSDs and Controls.
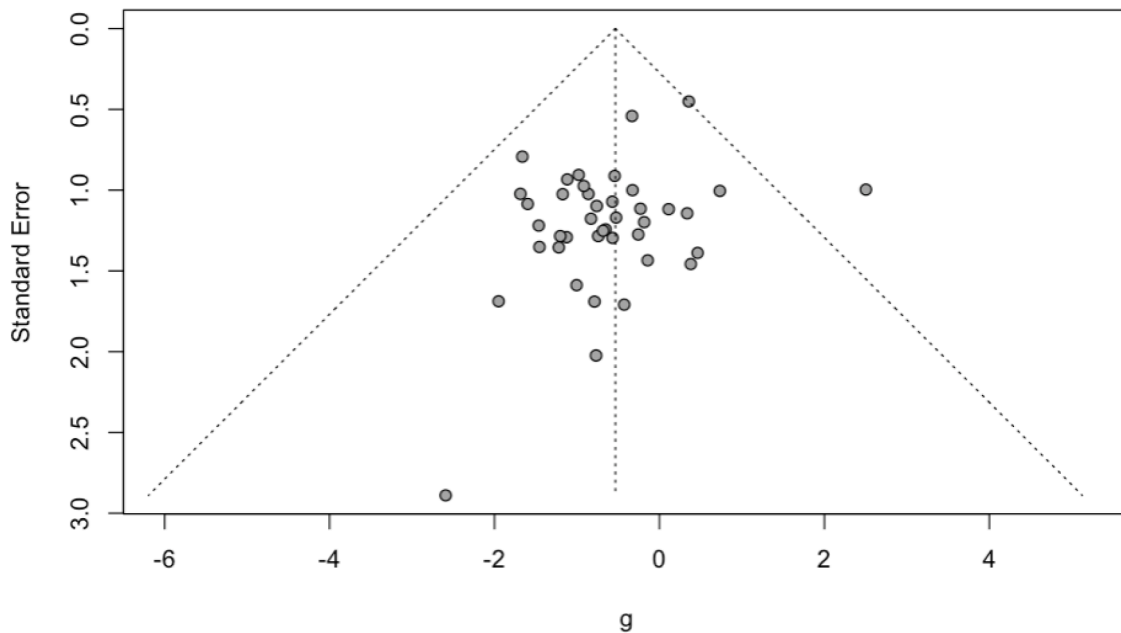
**Fig. 11.** Funnel Plot of Differences Between Physical and Social Anhedonia within SSDs.

**Table 4**
Results of meta-analyses and meta-regression analyses: Major Depressive Disorder.

| Major Depressive Disorder | Hedge's G | 95%CI | Q | I² | p |
|---|---|---|---|---|---|
| Physical Anhedonia | 1.15** | 0.94\|1.37 | 10.18 | 31.3% | <0.0001 |
| Social Anhedonia | 1.29** | 1.07\|1.51 | 11.45 | 38.9% | <0.0001 |
| Moderators (Phys\|Soc) | β | SE | 95%CI Low | 95% Hi | p |
| Age | −0.00\|− 0.01 | 0.01\|0.01 | −0.03\|− 0.01 | 0.03\| 0.02 | 0.94\| 0.70 |
| %Male | 0.01\| 0.001 | 0.01\|0.01 | −0.00\|− 0.01 | 0.02\| 0.02 | 0.10\| 0.94 |
| DepSx | 0.02*\| 0.00 | | 0.001\|− 0.02 | 0.03\| 0.02 | 0.03\| 0.79 |
| Phys. vs. Soc. within-group | Hedge's G | 95%CI | Q | I² | p |
| | −0.72 | −2.92\|− 1.47 | 0.30 | 0.0% | 0.52 |
| Moderators | β | SE | 95% CI | p | |
| Age | −0.003 | 0.1 | −0.20\| 0.13 | 0.98 | |
| %Male | 0.01 | 0.06 | 0.20 | 0.83 | |
| DepSx | 0.02 | 0.07 | −0.10\| 0.16 | 0.83 | |

Note: Atypical AP= Atypical Antipsychotic, DepSx=Depression.

experiencing less pleasure than they actually experienced when in-the-moment. Previous findings suggest that self-reported or clinician-rated anhedonia is more reflective of anticipatory than consummatory deficits, as individuals engage in similar memory recall when anticipating future events (Kring and Elis, 2013). Anticipatory pleasure deficits may also explain why findings of elevated trait-based anhedonia in SSD vs. controls, along with previous self-report findings, are inconsistent with laboratory-based tasks assessing in-the-moment responses among people with SSDs. Given that negative memory biases have been observed in both groups, future work should focus on comparing anhedonia across different methods to more directly examine the impact of recall bias on hedonic deficits.

Regarding our second aim, we were surprised to find that interview-

rated negative symptom severity did not moderate group differences in physical or social anhedonia between those with SSDs and controls. Though unexpected, this finding is consistent with those of Visser et al. (2020), who found that negative symptoms did not moderate self-reported trait anhedonia in people with SSDs. On one hand, the lack of moderation from negative symptoms may be due to an insufficient number of SSD studies included in this review, leading to a lack of power in the analysis. On the other hand, these results may indicate that negative symptoms do not influence the variance of self-reported trait anhedonia. Unlike clinical interviews, self-report measures only capture the experiential component of negative symptoms (i.e., anhedonia, avolition, asociality) but not the expressive component (i.e., affective flattening, alogia). Thus, it is possible we were not able to detect moderation because the Chapman Scales do not capture the full scope of negative symptoms. Furthermore, responses on trait-based assessments may be more directly impacted by cognitive difficulties and negative memory biases than are those elicited by clinical interviews of negative symptoms. Finally, the lack of moderation may also reflect a lack of coherence between clinical interviews (i.e., observer-rated assessment) and self-reports, as this has been detected in previous studies (Durand et al., 2015; Gould et al., 2015).

Furthermore, we found that depressive symptom severity moderated group differences in physical anhedonia, but not social anhedonia, in people with MDD. These findings suggest that physical anhedonia, relative to social anhedonia, is more likely to covary with depressive symptoms, and therefore worsen as a function of depression. Further, these findings suggest that in regards to assessment and treatment, physical anhedonia may be a stronger marker for the presence of a depressive episode, while social anhedonia may persist as a trait-like deficit regardless of depressive symptom severity. Overall, it is possible that physical anhedonia covaries more strongly with the experience of depression than does social anhedonia. Nonetheless, given the relatively low magnitude of these effects, findings should be further examined in future studies.

Sample characteristics (i.e., age, sex, race, medication status) did not moderate within- or between-group differences in social or physical anhedonia in MDD. Within SSDs, age was a source of heterogeneity for the within-group discrepancy between physical and social anhedonia, such that older age was associated with smaller differences between the two forms of anhedonia. This finding is consistent with a recent
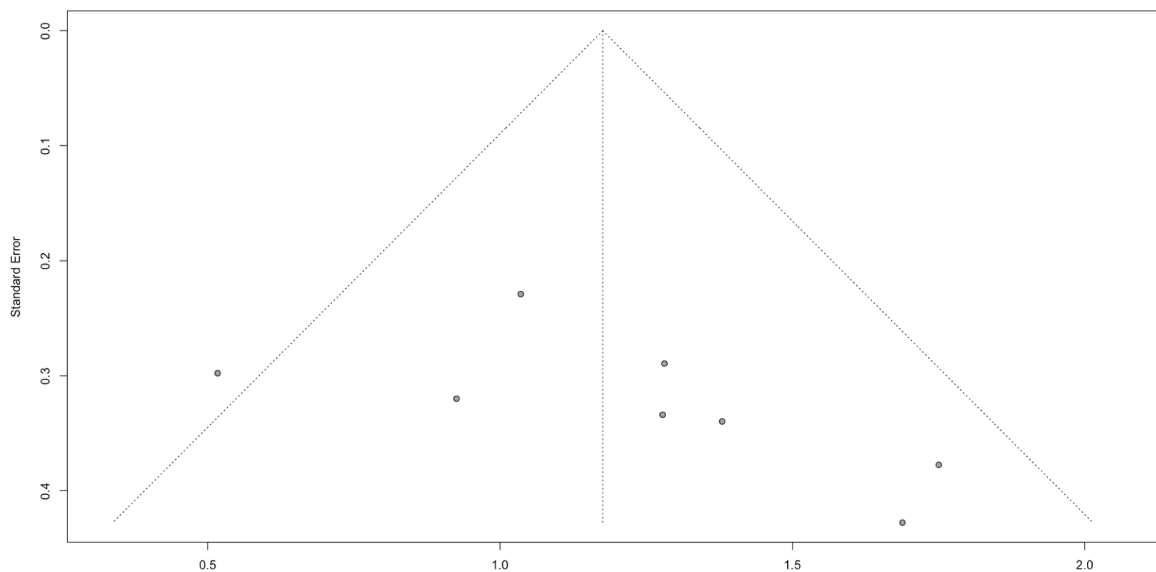
**Fig. 12.** Funnel Plot of Differences in Physical Anhedonia Between MDD and Controls.
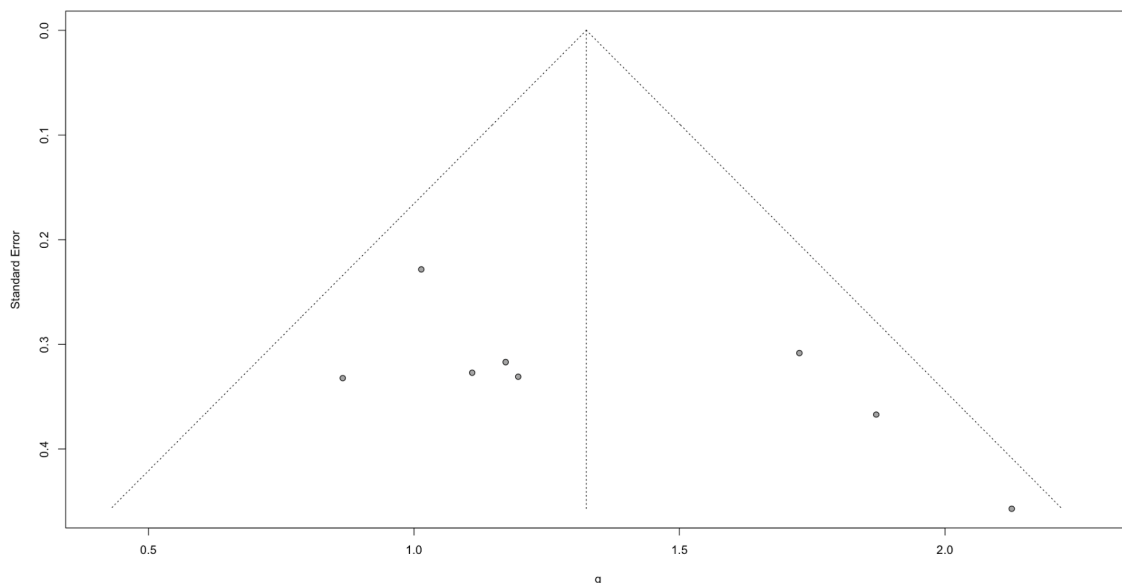


**Fig. 13.** Funnel Plot for Differences in Social Anhedonia Between MDD & Controls.

epidemiological investigation which found lower social anhedonia in healthy older adults compared to middle-aged adults (Dodell-Feder and Germine, 2018). Accordingly, smaller differences between the two subtypes may reflect social anhedonia following a normative decline within SSD, with physical anhedonia persisting at relatively the same level. Indeed, if self-reported social anhedonia reflects limited social opportunities, as discussed above, it may be that higher social anhedonia in older age reflects reduced social interactions over time.

Finally, it is important to note potential for publication bias based on exclusion of two influential studies in our analyses. Umesh et al. (2018) was the only study included in this review that was conducted in India, while participants in Makowski et al. (2016) were all male. It is possible these study details contributed to extreme effect sizes. Nevertheless, the results did not drastically change with the exclusion of these outliers, and therefore does not skew our interpretation of the results.

### 4.1. Limitations & conclusions

Several limitations of this meta-analysis must be considered. First, the small number of MDD studies raises concerns for these analyses lacking statistical power to detect differences. It is unclear whether the null findings for the moderators and the SAS/PAS within-group differences reflect a lack of statistical power, or true null effects. Future studies should directly compare social and physical anhedonia in MDD to further clarify whether anhedonia generalizes across domains or is specific to the social domain. Second, we could only include a limited number of moderators, due to limited reporting of data. Consequently, it remains unclear how racialized experiences, active vs. recovered MDD status, or antidepressants may impact the effects of anhedonia in MDD. Third, the large degree of heterogeneity within the SSD studies limits the precision of the overall effects and challenges the generalizability of these findings. Fourth, due to the limited availability of data, we were unable to directly compare the effects of anhedonia between MDD and SSD. Finally, MDD findings may not generalize to those in remission, as
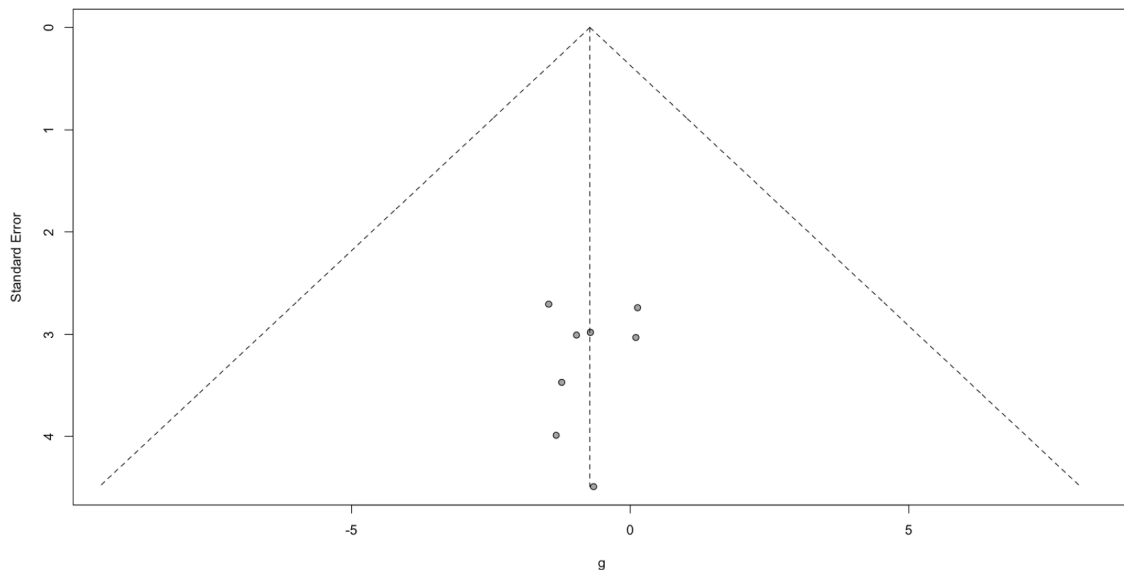
**Fig. 14.** Funnel Plot for Differences between Social and Physical Anhedonia within MDD.

the majority of our sample consisted of those experiencing a MDE at time of data collection.

In summary, this meta-analysis found that individuals with SSD and MDD reported greater trait social and physical anhedonia compared to controls. Importantly, only the SSD group demonstrated significant differences between social and physical anhedonia, while differences were not significant in MDD. Within MDD, depressive symptoms appear more strongly associated with physical anhedonia compared social anhedonia, and thus may be more predictive of a current episode. This is the first study to examine the relative difference between social and physical anhedonia using one of the most widely used scales in psychopathology literature. Our results add to the literature by suggesting that the extent to which trait-level, self-reported anhedonia is primarily physical, social, or generalized in nature may differ across clinical groups. Specifically, for people with SSDs, the experience of anhedonia may be more social in nature, while for people with MDD, the experience of anhedonia is more generalized. These results provide evidence for differing phenomenology and etiology of anhedonia across mood and psychotic disorders, and may inform more refined treatment targets.

## CRediT authorship contribution statement

**Arti Gandhi:** Conceptualization, Methodology, Formal analysis, Writing – original draft, Visualization. **Jasmine Mote:** Conceptualization, Writing – review & editing. **Daniel Fulford:** Conceptualization, Writing – original draft, Writing – review & editing, Supervision.

## Declaration of Competing Interest

None.

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