Archival Report

Biological Psychiatry

Amplification of Positivity Treatment for Anxiety and Depression: A Randomized Experimental Therapeutics Trial Targeting Social Reward Sensitivity to Enhance Social Connectedness

Charles T. Taylor, Murray B. Stein, Alan N. Simmons, Feng He, Christopher Oveis, Holly B. Shakya, William J. Sieber, James H. Fowler, and Sonia Jain

ABSTRACT

BACKGROUND: Social disconnection is common and causes significant impairment in anxiety and depressive disorders, and it does not respond sufficiently to available treatments. The positive valence system supports social bond formation and maintenance but is often hyporesponsive in people with anxiety or depression. We conducted an experimental therapeutics trial to test the hypothesis that targeting positive valence processes through cognitive and behavioral strategies would enhance responsivity to social rewards, a core mechanism underlying social connectedness.

METHODS: Sixty-eight adults who endorsed clinically elevated anxiety and/or depression with social impairment were randomized 1:1:1 to 5 (n = 23) or 10 (n = 22) sessions of amplification of positivity (AMP) treatment or waitlist (n = 23). Pre- to posttreatment change in striatal activity (primary outcome) during social reward anticipation was measured using functional magnetic resonance imaging, and reactivity to a social affiliation task (secondary) and self-reported social connectedness (exploratory) were examined. Primary analyses compared AMP (doses combined) versus waitlist. A second aim was to compare the effects of different doses.

RESULTS: AMP engaged the hypothesized treatment target, leading to greater striatal activation during anticipation of social rewards versus waitlist (d = 1.01 [95% CI = 0.42–1.61]; largest striatal volume). AMP yielded larger improvements in positive affect and approach behavior during the affiliation task (but not other outcomes) and social connectedness. Larger striatal and social connectedness increases were observed for 5-session versus 10-session AMP (d range = 0.08–1.03).

CONCLUSIONS: Teaching people with anxiety or depression strategies to increase positive thoughts, behaviors, and emotions enhances activity in brain regions that govern social reward processing and promotes social connectedness. Social reward sensitivity may be a transdiagnostic target for remediating social disconnection.

https://doi.org/10.1016/j.biopsych.2023.07.024

Social disconnection—both perceived and actual—is a common and disabling feature of anxiety and depressive disorders (1) that diminishes quality of life (2,3). It consists of several facets, including structure (e.g., small social network), function (e.g., perceived lack of support), and quality (e.g., dissatisfaction with social relationships or roles). First-line psychosocial treatments produce modest improvements in social functioning, falling short of much larger changes observed for symptoms (4–6), with social disconnection persisting after symptoms remit (1). These findings suggest that current treatments do not sufficiently engage the mechanisms that support positive connections with others.

How are social connections established and maintained? Humans are fundamentally motivated by cues (incentives) that signal the possibility of reward (i.e., desired or valued outcomes) or punishment (i.e., aversive outcomes) (7). Social contexts yield cues that signal the potential for establishing a social connection. Positive valence cues from others (e.g., signs of affiliation such as smiling or social initiation) activate approach motivation, including anticipation of valued social outcomes (8). Social approach motivation is characterized by the desire to connect with others; it encourages behavior that is directed toward pursuing social connection (7), including displays of active engagement and responsiveness (9) [e.g., self-disclosure (10), positive emotional expressions (11)]. Approach behaviors elicit affiliative reactions from others (9,12,13) that generate positive emotions and increase motivation to connect with others in the future (14,15). These positive valence processes create a cycle that supports the initiation and strengthening of social bonds regulated by the mesolimbic reward circuit that activates in response to cues that signal potential social reward (16–18) (i.e., incentive

ISSN: 0006-3223

salience or "wanting") (19). The striatum is centrally involved in this circuit; it reliably engages across various social cues and contexts [e.g., the opportunity to engage in self-disclosure (20); sharing experiences with others (21); and anticipating viewing a smiling face (18)], suggesting its central role in supporting social connection (8).

Hyporesponsivity of the positive valence system characterizes depression (22) and some forms of anxiety (23) [most notably social anxiety disorder (24) and posttraumatic stress disorder (25)] as evidenced by low positive affect (24), diminished approach motivation and behavior (26,27), and reduced activation in mesolimbic circuits during reward processing (25,28), including anticipation of social rewards (29,30). People who are diagnosed with these conditions also experience social anhedonia—loss of interest in pursuing or pleasure in response to social activities—which is associated with greater social impairments (31) even controlling for anxiety and depression severity (32). Notably, research in individuals with anxiety and depression has demonstrated that positive valence processes (e.g., approach motivation, positive affect) are robustly associated with social connectedness beyond any effects of negative valence processes (e.g., avoidance motivation, negative affect) (3,14). Therefore, diminished sensitivity to social rewards may be a transdiagnostic target for improving social connectedness in anxiety and depression. Although it is not the only candidate mechanism underpinning social disconnection (cf. heightened social threat reactivity and avoidance) (3,33), and not everyone who experiences anxiety or depression is characterized by diminished sensitivity to social rewards, it is an underexplored target with the potential to address an unmet treatment need.

First-line treatments do not sufficiently repair positive valence deficits in individuals with anxiety or depression (34–36), which may explain in part why social impairments persist following established treatments (4,5). Amplification of positivity (AMP) was developed to address this gap (37). It comprises cognitive and behavioral strategies (i.e., positive activity interventions) (38–40) that target positive valence thoughts, behaviors, and emotions through repeated practices, including noticing and amplifying responsivity to positive events (e.g., savoring, reminiscing, disclosing positive events to others), promoting the experience and expression of gratitude, and engaging in acts of kindness. These strategies are not routinely part of existing evidence-based treatments for anxiety or depression. In nonclinical samples, they have been shown to engage processes that are believed to facilitate social connections. For example, engaging in kind acts (charitable donation) (41) and savoring positive memories (42) activate the striatum. Gratitude has positive dyadic effects: its experience induces social approach behaviors toward one's benefactor (43), while its expression elicits approach behaviors from the benefactor toward the expresser (44). Initial evidence from studies of individuals seeking treatment for anxiety or depression has revealed large increases in positive affect (37) and social connectedness (45) following 10 sessions of AMP compared to those in the waitlist (WL) condition, with session-by-session improvements in connectedness accounted for by increases in positive affect beyond any effects of reductions in negative affect (45). The current trial builds on this work to establish whether AMP enhances sensitivity to social rewards, a mechanism hypothesized to underpin social connectedness (8).

Current Study

This study was grounded within the National Institute of Mental Health experimental therapeutics pipeline (46) in which engagement of an identified treatment target (i.e., hypothesized mechanism underlying a clinical or functional outcome of interest) must be established before pursuing tests of clinical efficacy. Therefore, we conducted a mechanism-focused, 3-arm, parallel randomized controlled trial of 2 doses of AMP (5 or 10 sessions) versus WL in patients who endorsed clinically elevated anxiety or depression with at least moderate social impairment to test the primary hypothesis that AMP (both doses combined) would be superior to WL in increasing striatal activation during social reward anticipation (the primary measure of target engagement and outcome upon which decisions to further evaluate AMP in future trials was based). Anticipatory processing of social reward cues was chosen as the primary outcome because it sets into motion the chain of events that support the pursuit of valued outcomes, including social connection. Secondary measures of target engagement were evaluated from psychophysiological, behavioral, and subjective responses that were obtained during a standardized social affiliation task (47).

A second aim was to compare effect size differences for 5- versus 10-session regimens to determine whether the treatment target could be engaged more efficiently (46). A small-to-medium (d = 0.4) effect size advantage of the 10-session protocol was identified a priori as the criterion to support its future evaluation over the 5-session protocol, pending support for the primary hypothesis. Finally, although this mechanism-focused trial was not powered to test clinical efficacy, we nevertheless explored AMP effects on measures of social functioning, symptoms, and well-being to inform future trials.

METHODS AND MATERIALS

Participants

A total of 68 participants were enrolled between April 2018 and July 2019. Inclusion criteria were age 18 to 55 years (inclusive); Overall Anxiety Severity and Impairment Scale score ≥8 (reflective of a probable anxiety disorder diagnosis) (48) or Patient Health Questionnaire score ≥10 (reflective of a probable diagnosis of major depression) (49); evidence of social disconnection [Social Connectedness Scale Revised (SCSR) score <90 (50)]; and moderate or greater social impairments (Sheehan Disability Scale—social domain score ≥5) (51). Participants were recruited through primary care clinics and announcements in community and online settings. Exclusion criteria were concurrent psychotropic use; concurrent psychotherapy (unless 12-week stability criteria were met); suicidal ideation with intent; history of major neurologic disorder or moderate to severe traumatic brain injury; severe or unstable medical conditions; past-year moderate to severe alcohol or cannabis use disorder or mild to severe substance use disorder (all other drugs); bipolar I or psychotic disorders; and magnetic resonance imaging (MRI) contraindications. Diagnostic interviews for sample characterization and exclusion

criteria were conducted using the Mini International Neuropsychiatric Interview for DSM-5 (version 7.0.2). See Table 1 for demographic and baseline clinical characteristics of the study participants.

All procedures involving human participants were performed in accordance with the ethical standards of the University of California San Diego Human Research Protection Program and with the Code of Ethics of the World Medical Association (Declaration of Helsinki).

Table 1. Patient Demographic and Baseline Clinical Characteristics

Variable	AMP, *n* = 45	WL, *n* = 23
Gender Identity		
Female	31 (68.9%)	16 (69.6%)
Male	13 (28.9%)	7 (30.4%)
Other	1 (2.2%)	0 (0%)
Age, Years	30.2 (9.8)	29.7 (8.4)
Years of Education	15.5 (2.1)	16.5 (2.6)
Race		
Asian	13 (28.9%)	4 (17.4%)
Black	2 (4.4%)	1 (4.4%)
More than one race	1 (2.2%)	2 (8.7%)
Pacific Islander	1 (2.2%)	0 (0%)
White	26 (57.8%)	16 (69.6%)
Unknown/declined to respond	2 (4.4%)	0 (0%)
Hispanic Ethnicity	8 (17.8%)	8 (34.8%)
Relationship		
Single	24 (53.3%)	13 (56.5%)
Married	9 (20%)	6 (26.1%)
Cohabitating	7 (15.6%)	2 (8.7%)
Divorced or separated	5 (11.1%)	1 (4.4%)
Other	0 (0%)	1 (4.4%)
Employment		
Not working	11 (24.4%)	5 (21.7%)
Working part-time	7 (15.6%)	1 (4.4%)
Working full-time	19 (42.2%)	11 (47.8%)
Student	8 (17.8%)	6 (26.1%)
Diagnoses[a]		
Major depressive disorder	39 (87%)	16 (70%)
Social anxiety disorder	26 (58%)	16 (70%)
Generalized anxiety disorder	23 (51%)	15 (65%)
Posttraumatic stress disorder	3 (7%)	2 (9%)
Panic disorder	4 (9%)	3 (13%)
Agoraphobia	7 (16%)	3 (13%)
Obsessive-compulsive disorder	4 (9%)	2 (9%)
Eating disorder	3 (7%)	1 (4%)
Mild alcohol use disorder	6 (13%)	1 (4%)
Mild cannabis use disorder	3 (7%)	2 (9%)
Suicidal ideation (past month)	16 (36%)	7 (30%)
PHQ-9[b]	13.0 (4.4)	10.5 (4.9)
OASIS	10.1 (3.3)	10.7 (3.1)
SCSR	60.8 (15.3)	62.0 (11.9)
SDS	17.2 (5.8)	15.5 (5.3)

Values are presented as mean (SD) or *n* (%).

AMP, amplification of positivity; OASIS, Overall Anxiety Severity and Impairment Scale; PHQ-9, Patient Health Questionnaire; SCSR, Social Connectedness Scale Revised; SDS, Sheehan Disability Scale; WL, waitlist.

[a]Percentages sum to >100% given high comorbidity across the sample.

[b]Groups significantly differed ($p < .05$).

Outcome Measures

Primary Outcome—Social Incentive Delay Task. The social incentive delay task (52) reliably activates the striatum (primary region of interest) when people anticipate obtaining social rewards (e.g., viewing a smiling face) (18).[1] During reward blocks, distinct cues indicated whether to anticipate social reward or a neutral outcome. Participants gained social reward if their reaction to the target was on time. The primary outcome was a change in striatal activation during social reward anticipation trials compared with implicit baseline. This contrast was used (cf. anticipation of social reward minus neutral outcomes) because the reliability of difference scores is always lower than the reliability of their individual parts (53), which has been shown to diminish the reliability of task-based functional MRI (54) [see also (55)] and hinder mechanistic research (56). See Supplemental Methods for a full description.

Secondary Outcomes—Social Affiliation Task. Participants completed an 18-minute conversation with a trained same-sex experimental assistant (confederate). Conversation partners alternated responding to questions gradually increasing in intimacy. Different questions and same-sex confederates were used at baseline and posttest. This task reliably induces connectedness between unacquainted partners (47). See Supplemental Methods for details. Secondary measures of social reward sensitivity were participant-reported positive affect following the conversation (57); positive facial expressions during the perceiver (listening) role of the task (11); affiliative (social approach) behavior (13,47); future approach motivation (desire for future interaction) (11,14,58); and respiratory sinus arrhythmia (59) reactivity (difference between the beginning and end of the task) (60,61). See the Supplement for details.

Key Exploratory Outcomes—Social Connectedness. The primary clinical end point prespecified in this program of work was measured using the NIH Toolbox Friendship and Loneliness surveys (62) and the SCSR (50).

Other Exploratory Outcomes. Surveys assessing different facets of social functioning, positive and negative valence symptoms, functioning, and well-being were used (see Supplemental Methods).

Nonspecific Treatment Measures

Treatment credibility/expectancy, working alliance, and homework completion data were collected for AMP participants. See the Supplement.

[1]We use the term "reward" to be consistent with the literature on the social incentive delay (18) and social reward processing (8) in which smiling faces are commonly used as incentive cues. Prior work demonstrates that people are willing to forgo money or exert significant amounts of effort to view smiling faces (73), suggesting that such cues are perceived as rewarding [see (8)].

Treatment

Amplification of Positivity. AMP is a manualized, clinician-delivered intervention comprising 3 core elements: 1) increasing exposure and responsiveness to positive events; 2) practicing gratitude; and 3) engaging in kind or generous acts toward others. The 5- and 10-session protocols were identical in the initial 4 treatment strategies (noticing and amplifying positive events; gratitude reflection; acts of kindness; scheduling pleasurable, engaging, and meaningful activities); the 10-session AMP included additional activities targeting the core domains (active/constructive responding; gratitude expression; make someone else happier; live this month like it is your last in your current city). See Table S1 for treatment modules by arm. Following their final in-person session, 5-session AMP participants reviewed their treatment plan with their clinician by phone (30 minutes; week 6) and received weekly e-mails to encourage their continued engagement in treatment activities (weeks 7–10). See Supplemental Methods for treatment adherence scale descriptions.

Waitlist. WL participants completed pre- and postassessments at a 10-week interval. They were offered AMP following the postassessment; however, their treatment data were not included in the analyses.

Procedure

Participants provided informed written consent prior to eligibility screening. Those who met inclusion criteria and agreed to participate completed baseline assessments comprising self-report surveys and the social affiliation task followed by a separate functional MRI session involving the social incentive delay task. Participants were then randomized 1:1:1 to AMP (5 or 10 sessions) or WL using a randomly permuted block design. Stratification factors were sex assigned at birth and social connectedness (SCSR $\geq$ 60 vs. $\leq$59). Experimental personnel (e.g., confederates, functional MRI operators) were blinded to treatment assignment, but participants and clinicians were not. Participants completed postassessments after finishing their assigned treatment protocol or approximately 10 weeks after the baseline MRI (WL). Participants received monetary compensation for assessment sessions. Procedures were approved by the university's Human Research Protections Program.

Statistical Analyses

All randomized participants who had baseline data and at least one postbaseline measurement were included in the analysis (modified intent-to-treat).

Analysis of Primary Outcome. Voxelwise activation data within the striatum (Harvard-Oxford anatomical mask including the caudate, putamen, and nucleus accumbens) were entered into a generalized linear model (AFNI: 3dLME) (63) comparing activation across groups (AMP vs. WL) over time (pre, post) to social reward cues during anticipation (any reward vs. implicit baseline). Permutation testing within AFNI's 3dClustSim (63) was used to minimize identification of false-positive activations within the striatum mask (voxelwise a priori probability of .005 with corrected clusterwise activation probability of .05). Parameter estimates were extracted from significant clusters that emerged from the group $\times$ time analysis within the striatum mask for effect size computation and to visualize treatment-related effects. Data from 4 participants were removed (blinded to treatment assignment) due to poor quality (see the Supplement).

Analysis of Secondary Target Engagement Outcomes. Social affiliation task outcomes were analyzed using a linear mixed-effects model. Independent variables included treatment arm (AMP vs. WL), visit (pre, post), and treatment by visit interaction.

Analysis of Exploratory Social Connectedness, Symptom, and Functioning Outcomes. Our key exploratory outcome was social connectedness, measured using the NIH Toolbox Friendship and Loneliness scales and the SCSR. Additional social functioning, symptom, and well-being outcomes are presented in the Supplement. Exploratory outcomes were analyzed as described above for secondary outcomes. There was no correction for multiple comparisons for secondary or exploratory outcomes because this early-phase trial was intended to inform measurement decisions in future work.

Analyses of secondary and exploratory outcomes and effect size computations were conducted using R version 3.6.1 (https://www.r-project.org/). Baseline demographic variables that were both unbalanced at baseline ($p < .10$) and associated with the outcome ($p < .15$) of clinical interest were included in the model as covariates. Because all baseline demographic variables were balanced between the arms, they were not included in the model.

Effect Size Computation. Cohen's *d* differences for the change score from baseline were computed for: 1) AMP (both doses combined) versus WL (primary aim), and 2) 5- versus 10-session AMP (secondary aim). Effect size and 95% CIs are presented for primary and secondary outcomes and for key social connectedness outcomes.

Baseline Group Comparison. Categorical variables were evaluated using Fisher's exact tests. Continuous variables were analyzed with Wilcoxon's rank-sum tests.

Sample Size Determination. The study was a priori powered assuming 15% attrition and alpha = .05 using a two-sided, 2-sample *t* test. Therefore, we planned to enroll 71 subjects to have an evaluable sample size of 60 (40 AMP, 20 WL), which would provide 80% power to detect a standardized change between groups of 78%.

RESULTS

Preliminary Analyses

Participant progress throughout the trial is summarized in Figure 1 (CONSORT [Consolidated Standards for Reporting Trials] diagram). Groups did not differ on baseline characteristics (all $ps > .10$), except that AMP participants had higher Patient Health Questionnaire scores versus WL participants ($p = .037$) (see Table 1). There were 37 out of 45 (82%) AMP

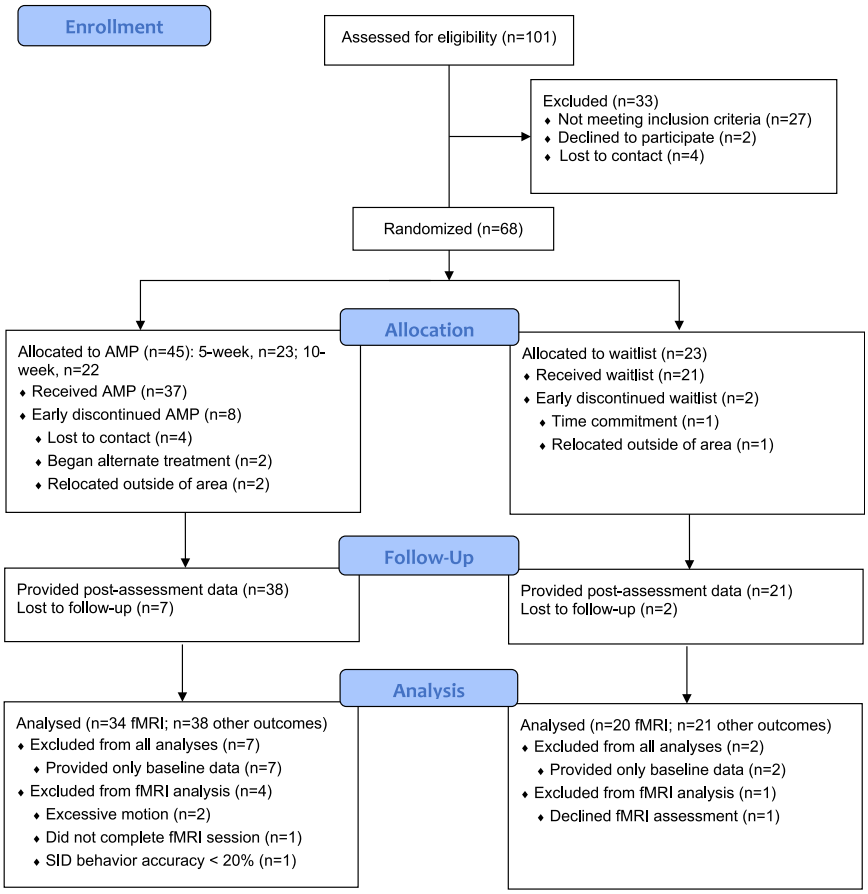

Figure 1. CONSORT (Consolidated Standards for Reporting Trials) flow diagram summarizing participants' progress throughout the study. AMP, amplification of positivity; fMRI, functional magnetic resonance imaging; SID, social incentive delay.

completers and 21 out of 23 (91%) WL completers. Post-assessment data were obtained from 1 AMP participant who discontinued early; all other early discontinuations were lost to follow-up. The 5- and 10-session AMP arms did not differ significantly on treatment credibility (p = .60) or expectancy (p = .81), homework compliance from weeks 2 to 5 ($p > .05$), or working alliance (all $p > .05$). Treatment adherence was uniformly high (see the Supplement). Two adverse events—both mild (presence of suicidal ideation without intent)—occurred in the 10-session AMP group and were deemed to be unrelated to the intervention.

Primary Target Engagement Outcomes

The group × time linear mixed-effects analysis revealed that AMP participants displayed significantly greater pre- to post-treatment activation increases in several striatal regions, including the left nucleus accumbens, bilateral caudate, and bilateral putamen, compared with WL participants (see Table 2). Figure 2 illustrates activation differences by group within the largest cluster that emerged from this analysis (right putamen, 86 voxels, x = 25, y = 1, z = 2). These results demonstrate that AMP engaged the hypothesized treatment target, i.e., striatal activation during anticipation of social rewards.

Secondary Target Engagement Outcomes

Group × time interaction terms revealed significantly larger pre- to posttreatment increases in postconversation positive affect (d = 0.62, 95% CI = 0.06–1.18) and social approach behaviors (d = 0.65, 95% CI = 0.09–1.21) for AMP versus WL participants. The group × time interaction was not significant for respiratory sinus arrhythmia reactivity (d = 0.50, 95% CI = −0.06 to 1.05), positive facial expressions (d = −0.07, 95% CI = −0.62 to 0.47), or future approach motivation (d = −0.23, 95% CI = −0.78 to 0.31) (see Table 3).

Key Exploratory Outcomes: Social Connectedness

Group × time interactions revealed significantly larger AMP versus WL increases in social connectedness (National Institutes of Health Friendship: b = 3.22, SE = 1.09, t_{57} = 2.95, p = .005, d = 0.80, 95% CI = 0.24–1.37; SCSR: b = 12.24, SE = 2.98, t_{57} = 4.11, $p < .001$, d = 1.12, 95% CI = 0.54–1.70) and decreases in loneliness (b = −4.02, SE = 1.00, t_{57} = −4.03, $p <$ .001, d = −1.10, 95% CI = −1.68 to −0.52) (see Table S2).

Other Exploratory Outcomes

Significantly larger AMP versus WL improvements were observed on measures of anxiety, depression, positive and negative affect, functional interference, satisfaction with social

Table 2. Regions of Interest Analysis Within the Striatum Showing Significant Group × Time Effects During Social Reward Anticipation

Region	Cluster Size, No. of Voxels	MNI Coordinates Center of Mass			*t* Statistic	Change, Mean (SD)		Effect Size (95% CI)
		x	y	z		Waitlist, *n* = 20	AMP, *n* = 34	
Right Putamen	86	25	1	2	3.73	0.17 (0.19)	0.35 (0.21)	1.01 (0.42–1.61)
Left Caudate	30	−11	1	17	3.53	−0.19 (0.32)	0.15 (0.48)	0.80 (0.21–1.38)
Left Putamen	27	−28	−1	1	3.26	−0.11 (0.23)	0.07 (0.23)	0.76 (0.18–1.35)
Left Putamen	25	−18	10	7	3.58	−0.03 (0.25)	0.16 (0.29)	0.68 (0.10–1.26)
Right Caudate	12	14	0	22	3.85	−0.17 (0.32)	0.13 (0.46)	0.73 (0.15–1.31)
Left NAc	7	−10	7	−12	3.97	−0.94 (1.38)	0.45 (1.05)	1.18 (0.57–1.79)

Results shown from the linear mixed-effects analysis of group (AMP vs. waitlist) × time (pretreatment, posttreatment) on percent signal change for social reward anticipation trials of the social incentive delay task. Change reflects the difference from baseline (posttreatment activation minus pretreatment activation). Effect size (Cohen's *d*) was computed from the pre- to posttreatment change difference between groups (AMP > waitlist).

AMP, amplification of positivity; MNI, Montreal Neurological Institute; NAc, nucleus accumbens.

roles and activities, and meaning and purpose (all *p*s < .05). Groups did not differ significantly on changes in satisfaction with discretionary social activities, perceived emotional support, or general life satisfaction (*p* > .05), although mean improvements favored AMP versus WL participants (see Tables S3 and S4).

AMP Dose Comparison

Brain activation data (% signal change) were extracted from significant group × time clusters within the striatum (Table 2), and pre- to posttreatment effect size differences were compared by AMP dose. The 5- versus 10-session protocol evidenced larger increases across all striatal regions (*d* range = 0.08−1.03; 5 > 10-session) (Table S5). Similar dose effects were observed across social affiliation task outcomes and key social connectedness exploratory outcomes (see Tables S6 and S7).

DISCUSSION

This mechanism-focused experimental therapeutics trial examined whether AMP increases responsivity to social reward in individuals seeking treatment for anxiety or depression. In support of our primary hypothesis, AMP increased striatal activation during social reward anticipation compared with WL, demonstrating a large group difference on average. To our knowledge, this study is the first to demonstrate psychosocial treatment enhancement of the striatum during social reward processing in people with anxiety or depression, suggesting engagement of a mechanism that supports the drive of humans to connect with others (8). Therefore, AMP may offer a new approach for remediating social disconnection in anxiety and depressive disorders and possibly other psychiatric conditions that are characterized by diminished social reward responsivity (31).

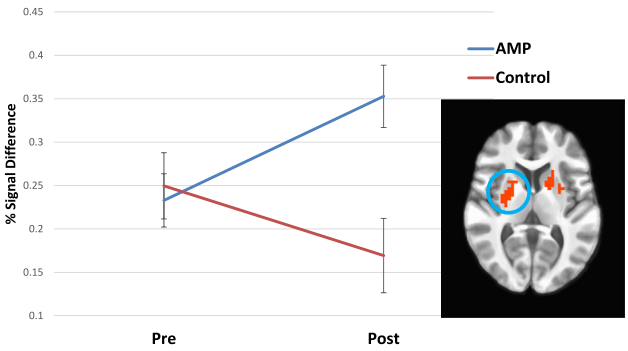

Figure 2. Change in striatal activation (right putamen, x = 25, y = 1, z = 2) from pre- to posttreatment during social reward anticipation trials of the social incentive delay task. Data reflect parameter estimates extracted from the largest cluster emerging from the group × time linear mixed-effects model (voxelwise a priori probability of .005 with corrected clusterwise activation probability of .05 within the striatum mask). AMP, amplification of positivity.

AMP engaged several distinct regions within the striatum, including the left nucleus accumbens, bilateral caudate, and bilateral putamen, consistent with meta-analyses suggesting broad striatal engagement when people anticipate possible social rewards (18,64). Subdivisions within the striatum serve distinct but complementary functions in reward processing: the ventral striatum (comprising the nucleus accumbens) codes the value of stimuli, shaping reward expectations and approach motivation, whereas the dorsal striatum (comprising the caudate and putamen) is involved in action selection and motor behavior, guiding pursuit of anticipated reward outcomes (16). Functions served by each subdivision are engaged during the social incentive delay task as well as social connection opportunities more broadly. Although the experimental therapeutics framework necessitates focus on a central treatment target to inform next-step decisions about treatment evaluation (46), social reward processing is multifaceted, involving different phases (e.g., anticipation, responsiveness) and brain regions outside the striatum (e.g., anterior insula, anterior cingulate cortex) (18). Therefore, future work should examine broader neural effects of AMP across different phases of social incentive cue processing.

AMP also increased positive affect and social approach behavior during a dyadic affiliation task—factors that have been consistently linked to positive social outcomes, including partner liking and desire for future interaction—compared with the WL (medium-to-large effect size differences) (9,13–15). However, groups did not differ on change in respiratory sinus arrhythmia reactivity (medium-sized group difference; AMP >

Table 3. Descriptive Summaries of Treatment Outcome Measures From the Social Affiliation Task at Baseline and Postassessment for the Waitlist (n = 21) and AMP (n = 38) Groups

Measure	Baseline, Mean (SD)	Postassessment, Mean (SD)	Change, Mean (SD)	Results (Group × Time) β	SE	*df*	*t*	*p*
Positive Affect—Postconversation								
Waitlist	34.95 (11.20)	33.24 (11.2)	−1.71 (11.56)	6.63	2.91	57	2.28	.026
AMP	32.37 (12.25)	37.29 (13.1)	4.92 (10.19)					
Positive Facial Expressions								
Waitlist, n = 21	0.44 (0.22)	0.45 (0.16)	0.02 (0.14)	−0.02	0.06	56	−0.27	.79
AMP, n = 37	0.47 (0.22)	0.47 (0.31)	0.00 (0.23)					
Social Approach Behavior								
Waitlist	28.43 (5.50)	26.29 (6.37)	−2.14 (5.24)	4.17	1.74	57	2.39	.020
AMP	25.79 (7.35)	27.82 (5.94)	2.03 (6.95)					
Respiratory Sinus Arrhythmia Reactivity								
Waitlist, n = 21	0.03 (0.53)	−0.11 (0.40)	−0.14 (0.65)	0.36	0.19	56	1.85	.07
AMP, n = 37	−0.04 (0.43)	0.18 (0.60)	0.21 (0.76)					
Desire for Future Interaction								
Waitlist	40.48 (9.57)	41.24 (8.57)	0.76 (11.3)	−2.60	3.04	57	−0.86	.40
AMP	38.79 (10.46)	36.95 (11.16)	−1.84 (11.13)					

Change reflects the difference from baseline (posttreatment minus pretreatment).
AMP, amplification of positivity.

WL), positive facial expressions, or desire for future interaction (small group differences). It is unclear whether AMP strategies impact those outcomes less directly or whether features of the paradigm (e.g., different conversation partners at pre- and postassessment) reduced sensitivity to detecting change. For example, desire for future interaction is likely influenced by multiple factors, including general motivational tendencies (e.g., approach vs. avoidance), specific qualities of the interaction partner, subjective affect following the encounter (14), and the future social context being considered (e.g., asking for advice vs. becoming friends). Nevertheless, dyadic task outcomes suggest that AMP may influence some positive valence affective and behavioral processes that have been shown to support social connections—complementing the primary neural outcomes.

Psychosocial treatment development rarely evaluates the requisite dose or duration of treatment that is needed to exert its effects (46); however, doing so could help develop interventions with potential for maximal clinical impact (65). Accordingly, a second aim of this study involved comparing target engagement across 2 doses of AMP that contained the same core strategies intended to engage positive valence responses. The 5-session protocol showed larger striatal engagement, suggesting that additional treatment exercises included in the 10-session protocol were not needed to further engage the hypothesized treatment target compared with allowing time for participants to practice the core AMP skills. Parsimony may have facilitated deeper learning of core skills with greater opportunities to personalize them (65). It is also possible that the 5-session program induced time scarcity, thereby motivating participants to maximize engagement in the few treatment sessions that they had. Including a measure of target engagement immediately after the core AMP strategies were administered would be necessary to confirm this possibility. Regardless, current findings underscore the value of designing psychosocial trials with dose effects in mind (46,65). Of course, this trial is limited by the way that dose was conceptualized and by comparing only 2 dosing levels. It also remains unknown whether there are patients for whom the 5- versus 10-session doses may be optimal.

This mechanism-focused trial was not powered to test clinical efficacy; however, we evaluated efficacy to inform future trials. Consistent with previous findings (45), AMP led to large improvements in social connectedness compared with WL as reflected on measures of perceived friendship, belongingness, and loneliness. Converging with the primary striatal outcomes, 5-session AMP led to nominally larger improvements in connectedness compared with the 10-session protocol. Extending the assessment battery to capture different aspects of social functioning more fully, we found that AMP produced larger increases in satisfaction with social roles and activities and similar but less robust increases in perceived emotional support and satisfaction with discretionary social activities. AMP-related improvements were also observed across measures of positive and negative affect, anxiety and depression symptoms, functional interference, and psychological well-being. Those outcomes converge with earlier findings (37) and other emerging positive affect-targeted approaches in anxiety and depressive disorder samples (66,67), further supporting the value of explicitly targeting positive valence processes in treating these conditions. These findings are encouraging because improvements in positive affect and well-being tend to be smaller and lag behind reductions in clinical symptoms following established treatments (4,5,34). Generalization of AMP effects to negative affect and symptom outcomes is consistent with work demonstrating that positive emotions mitigate negative responses to stressors that often fuel anxiety and depression (38,68,69).

The current findings should be interpreted alongside several caveats. This trial was conducted in a small sample in which the primary AMP comparison was against WL. The next step will be to replicate target engagement in a larger sample compared with a control condition accounting for common therapeutic effects. Such sufficiently powered trials should also examine whether changes in responsivity to social reward covary with improvements in social connectedness and for whom AMP may be most useful. The transdiagnostic sample raises the question of whether response to AMP varies by principal diagnosis (e.g., depression vs. anxiety) or symptom dimension (e.g., anhedonia vs. anxious arousal). AMP may be especially efficacious for patients who are experiencing elevated anhedonia [e.g., primarily those diagnosed with major depression but also many patients with social anxiety disorder (70) or posttraumatic stress disorder (71)] compared with those primarily characterized by anxious arousal (e.g., panic disorder). Relatedly, although all participants reported at least moderate social functioning impairments at intake, the mechanisms that underlie those impairments likely vary across individuals. Diminished sensitivity to social rewards may be the central mechanism underpinning social disconnection in some patients, whereas heightened sensitivity to aversive social outcomes may be a more influential mechanism in others (3). Idiographic approaches that consider which target(s), for whom, and under what set of conditions (72) may be fruitful to address the multiply determined nature of social disconnection.

The majority female sample composition is consistent with the epidemiology of anxiety and depressive disorders; however, evidence pointing to sex differences in sensitivity to social reward cues (52) suggests that future research with larger samples should examine whether AMP response varies by gender identity or sex. Measures of the treatment target in the current study were limited by presenting static images of smiling faces (cf. dynamic social cues encountered in real-life) to participants and having them engage in a contrived, circumscribed social context. Measurement of social connectedness and symptom outcomes relied on self-report. It would be valuable to establish whether AMP results in changes in real-world connectedness that translate into improved quality of life and which specific facets of connectedness change or do not change (e.g., network size, participation in social activities and groups, perceived quality).

Conclusions

Social disconnection is common in anxiety and depressive disorders and does not improve sufficiently following first-line treatments. The current findings offer initial support for AMP in enhancing responses to positive valence social cues and contexts. To our knowledge, this study is the first to show psychosocial treatment enhancement of the striatum during social reward processing in people with anxiety or depression, thus suggesting engagement of a mechanism that supports the drive to connect with others. The dose effect finding suggests that core activities focused on increasing exposure and responsivity to positive events, practicing gratitude, and engaging in kind acts may be sufficient to enhance striatal sensitivity to social rewards and connectedness. Replicating these effects in larger samples and determining whether striatal engagement accounts for improvements in social connectedness is needed now.

ACKNOWLEDGMENTS AND DISCLOSURES

This work was supported by National Institute of Mental Health (Grant Nos. R61MH113769 and R33MH113769 [to CTT]). The project described was partially supported by National Institutes of Health (Grant No. ULTR001442) Clinical and Translational Science Award funding beginning August 13, 2015 and beyond. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

We thank the many individuals who helped make this research possible: Michelle Behrooznia, Sarah Pearlstein, Matthew Boland, and Maria Kryza-Lacombe for serving as study therapists; Sarah Pearlstein and Taylor Smith for conducting diagnostic interviews; Taylor Smith, Kimberley Potter, Thomas Tsai, and Alison Sweet for overseeing project management; Margaret Satchwell for assisting with preparing results tables; and the many student and postbaccalaureate volunteers for their help with recruitment, screening, data collection and management.

CTT played a lead role in conceptualization, funding acquisition, investigation, methodology, data curation, and project administration, supervision, and writing (original draft; review and editing) and played a supporting role in formal analysis. MBS played a supporting role in conceptualization, funding acquisition, investigation, methodology, project administration, supervision, and writing (review and editing). ANS played a lead role in data curation and formal analysis and a supporting role in writing (review and editing). FH played a lead role in data curation and formal analysis and a supporting role in writing (review and editing). CO played a supporting role in funding acquisition, methodology, data curation, and writing (review and editing). HBS played a supporting role in funding acquisition and writing (review and editing). WJS played a supporting role in funding acquisition, resources, project administration, and writing (review and editing). JF played a supporting role in funding acquisition and writing (review and editing). SJ played a lead role in formal analysis and a supporting role in funding acquisition, data curation, and writing (review and editing).

Requests for deidentified data, analysis code, and research materials should be made to Charles T. Taylor, c1taylor@health.ucsd.edu.

CTT declares that in the past 3 years he has been a paid consultant for Bionomics and has received payment for editorial work for UpToDate, Inc. and the journal *Depression and Anxiety*. MBS declares that in the past 3 years he has been a paid consultant for Acadia Pharmaceuticals, Aptinyx, Bionomics, Clexio, EmpowerPharm, Genentech/Roche, GW Pharma, Janssen, Nobilis Therapeutics, and Oxeia Biopharmaceuticals, and has received payment for editorial work for UpToDate, Inc. and the journals *Biological Psychiatry* and *Depression and Anxiety*. All other authors report no biomedical financial interests or potential conflicts of interest.

ClinicalTrials.gov: Novel Behavioral Intervention to Enhance Social Connections in Anxiety and Depression; https://clinicaltrials.gov/ct2/show/NCT03196544; NCT03196544.

ARTICLE INFORMATION

From the Department of Psychiatry, University of California San Diego, San Diego, California (CTT, MBS, ANS); Herbert Wertheim School of Public Health & Human Longevity Science, University of California San Diego, San Diego, California (FH, HBS, SJ); VA San Diego Healthcare System, San Diego, California (MBS, ANS); Rady School of Management, University of California San Diego, San Diego, California (CO); Department of Family Medicine, University of California San Diego, San Diego, California (WJS); and Department of Political Science, University of California San Diego, San Diego, California (JHF).

Address correspondence to Charles T. Taylor, Ph.D., at c1taylor@health.ucsd.edu.

Received Feb 28, 2023; revised Jul 4, 2023; accepted Jul 25, 2023.

Supplementary material cited in this article is available online at https://doi.org/10.1016/j.biopsych.2023.07.024.

REFERENCES

1. Saris IMJ, Aghajani M, van der Werff SJA, van der Wee NJA, Penninx BWJH (2017): Social functioning in patients with depressive and anxiety disorders. Acta Psychiatr Scand 136:352–361.
2. Olatunji BO, Cisler JM, Tolin DF (2007): Quality of life in the anxiety disorders: A meta-analytic review. Clin Psychol Rev 27:572–581.
3. Taylor CT, Pearlstein SL, Stein MB (2020): A tale of two systems: Testing a positive and negative valence systems framework to understand social disconnection across anxiety and depressive disorders. J Affect Disord 266:207–214.
4. Hofmann SG, Wu JQ, Boettcher H (2014): Effect of cognitive-behavioral therapy for anxiety disorders on quality of life: A meta-analysis. J Consult Clin Psychol 82:375–391.
5. McKnight PE, Kashdan TB (2009): The importance of functional impairment to mental health outcomes: A case for reassessing our goals in depression treatment research. Clin Psychol Rev 29:243–259.
6. Renner F, Cuijpers P, Huibers MJ (2014): The effect of psychotherapy for depression on improvements in social functioning: A meta-analysis. Psychol Med 44:2913–2926.
7. Gable SL, Berkman ET (2008): Making connections and avoiding loneliness: Approach and avoidance social motives and goals. In: Elliot AJ, editor. Handbook of Approach and Avoidance Motivation. New York, NY: Psychology Press, 203–216.
8. Tamir DI, Hughes BL (2018): Social rewards: From basic social building blocks to complex social behavior. Perspect Psychol Sci 13:700–717.
9. Laurenceau JP, Barrett LF, Pietromonaco PR (1998): Intimacy as an interpersonal process: The importance of self-disclosure, partner disclosure, and perceived partner responsiveness in interpersonal exchanges. J Pers Soc Psychol 74:1238–1251.
10. Aron A, Melinat E, Aron EN, Vallone RD, Bator RJ (1997): The experimental generation of interpersonal closeness: A procedure and some preliminary findings. Pers Soc Psychol Bull 23:363–377.
11. Pearlstein SL, Taylor CT, Stein MB (2019): Facial affect and interpersonal affiliation: Displays of emotion during relationship formation in social anxiety disorder. Clin Psychol Sci 7:826–839.
12. Maisel NC, Gable SL, Strachman A (2008): Responsive behaviors in good times and in bad. Pers Relationship 15:317–338.
13. Taylor CT, Alden LE (2011): To see ourselves as others see us: An experimental integration of the intra and interpersonal consequences of self-protection in social anxiety disorder. J Abnorm Psychol 120:129–141.
14. Taylor CT, Pearlstein SL, Stein MB (2017): The affective tie that binds: Examining the contribution of positive emotions and anxiety to relationship formation in social anxiety disorder. J Anxiety Disord 49:21–30.
15. Vittengl JR, Holt CS (2000): Getting acquainted: The relationship of self-disclosure and social attraction to positive affect. J Soc Pers Relat 17:53–66.
16. Bhanji JP, Delgado MR (2014): The social brain and reward: Social information processing in the human striatum. Wiley Interdiscip Rev Cogn Sci 5:61–73.
17. Fareri DS, Delgado MR (2014): Social rewards and social networks in the human brain. Neuroscientist 20:387–402.
18. Martins D, Rademacher L, Gabay AS, Taylor R, Richey JA, Smith DV, *et al.* (2021): Mapping social reward and punishment processing in the human brain: A voxel-based meta-analysis of neuroimaging findings using the social incentive delay task. Neurosci Biobehav Rev 122:1–17.
19. Olney JJ, Warlow SM, Naffziger EE, Berridge KC (2018): Current perspectives on incentive salience and applications to clinical disorders. Curr Opin Behav Sci 22:59–69.
20. Tamir DI, Mitchell JP (2012): Disclosing information about the self is intrinsically rewarding. Proc Natl Acad Sci USA 109:8038–8043.
21. Wagner U, Galli L, Schott BH, Wold A, van der Schalk J, Manstead AS, *et al.* (2015): Beautiful friendship: Social sharing of emotions improves subjective feelings and activates the neural reward circuitry. Soc Cogn Affect Neurosci 10:801–808.
22. Whitton AE, Pizzagalli DA (2022): Anhedonia in depression and bipolar disorder. Curr Top Behav Neurosci 58:111–127.
23. Taylor CT, Hoffman SN, Khan AJ (2022): Anhedonia in anxiety disorders. Curr Top Behav Neurosci 58:201–218.
24. Kashdan TB (2007): Social anxiety spectrum and diminished positive experiences: Theoretical synthesis and meta-analysis. Clin Psychol Rev 27:348–365.
25. Nawijn L, van Zuiden M, Frijling JL, Koch SBJ, Veltman DJ, Olff M (2015): Reward functioning in PTSD: A systematic review exploring the mechanisms underlying anhedonia. Neurosci Biobehav Rev 51:189–204.
26. Richey JA, Brewer JA, Sullivan-Toole H, Strege MV, Kim-Spoon J, White SW, Ollendick TH (2019): Sensitivity shift theory: A developmental model of positive affect and motivational deficits in social anxiety disorder. Clin Psychol Rev 72:101756.
27. Trew JL (2011): Exploring the roles of approach and avoidance in depression: An integrative model. Clin Psychol Rev 31:1156–1168.
28. Keren H, O'Callaghan G, Vidal-Ribas P, Buzzell GA, Brotman MA, Leibenluft E, *et al.* (2018): Reward processing in depression: A conceptual and meta-analytic review across fMRI and EEG studies. Am J Psychiatry 175:1111–1120.
29. Cremers HR, Veer IM, Spinhoven P, Rombouts SA, Roelofs K (2014): Neural sensitivity to social reward and punishment anticipation in social anxiety disorder. Front Behav Neurosci 8:439.
30. A Richey JA, Ghane M, Valdespino A, Coffman MC, Strege MV, White SW, Ollendick TH (2017): Spatiotemporal dissociation of brain activity underlying threat and reward in social anxiety disorder. Soc Cogn Affect Neurosci 12:81–94.
31. Barkus E, Badcock JC (2019): A transdiagnostic perspective on social anhedonia. Front Psychiatry 10:216.
32. Olson EA, Pizzagalli DA, Rosso IM (2021): Social anhedonia is associated with low social network diversity in trauma-exposed adults. J Trauma Stress 34:241–247.
33. Cacioppo S, Grippo AJ, London S, Goossens L, Cacioppo JT (2015): Loneliness: Clinical import and interventions. Perspect Psychol Sci 10:238–249.
34. Dunn BD, German RE, Khazanov G, Xu C, Hollon SD, DeRubeis RJ (2020): Changes in positive and negative affect during pharmacological treatment and cognitive therapy for major depressive disorder: A secondary analysis of two randomized controlled trials. Clin Psychol Sci 8:36–51.
35. Kring AM, Persons JB, Thomas C (2007): Changes in affect during treatment for depression and anxiety. Behav Res Ther 45:1753–1764.
36. Sewart AR, Niles AN, Burklund LJ, Saxbe DE, Lieberman MD, Craske MG (2019): Examining positive and negative affect as outcomes and moderators of cognitive-behavioral therapy and acceptance and commitment therapy for social anxiety disorder. Behav Ther 50:1112–1124.
37. Taylor CT, Lyubomirsky S, Stein MB (2017): Upregulating the positive affect system in anxiety and depression: Outcomes of a positive activity intervention. Depress Anxiety 34:267–280.
38. Layous K, Chancellor J, Lyubomirsky S (2014): Positive activities as protective factors against mental health conditions. J Abnorm Psychol 123:3–12.
39. Lyubomirsky S, Layous K (2013): How do simple positive activities increase well-being? Curr Dir Psychol Sci 22:57–62.
40. Seligman MEP, Steen TA, Park N, Peterson C (2005): Positive psychology progress: Empirical validation of interventions. Am Psychol 60:410–421.
41. Moll J, Krueger F, Zahn R, Pardini M, de Oliveira-Souza R, Grafman J (2006): Human fronto-mesolimbic networks guide decisions about charitable donation. Proc Natl Acad Sci USA 103:15623–15628.
42. Speer ME, Bhanji JP, Delgado MR (2014): Savoring the past: Positive memories evoke value representations in the striatum. Neuron 84:847–856.
43. Bartlett MY, Condon P, Cruz J, Baumann J, Desteno D (2012): Gratitude: Prompting behaviours that build relationships. Cogn Emot 26:2–13.
44. Williams LA, Bartlett MY (2015): Warm thanks: Gratitude expression facilitates social affiliation in new relationships via perceived warmth. Emotion 15:1–5.

45. Taylor CT, Pearlstein SL, Kakaria S, Lyubomirsky S, Stein MB (2020): Enhancing social connectedness in anxiety and depression through amplification of positivity: Preliminary treatment outcomes and process of change. Cognit Ther Res 44:788–800.
46. Insel TR (2015): The NIMH experimental medicine initiative. World Psychiatry 14:151–153.
47. Hoffman SN, Thomas ML, Pearlstein SL, Kakaria S, Oveis C, Stein MB, Taylor CT (2021): Psychometric evaluation of a controlled social affiliation paradigm: Findings from anxiety, depressive disorder, and healthy samples. Behav Ther 52:1464–1476.
48. Campbell-Sills L, Norman SB, Craske MG, Sullivan G, Lang AJ, Chavira DA, *et al.* (2009): Validation of a brief measure of anxiety-related severity and impairment: The Overall Anxiety Severity and Impairment Scale (OASIS). J Affect Disord 112:92–101.
49. Kroenke K, Spitzer RL, Williams JB (2001): The PHQ-9: Validity of a brief depression severity measure. J Gen Intern Med 16:606–613.
50. Lee RM, Draper M, Lee S (2001): Social connectedness, dysfunctional interpersonal behaviors, and psychological distress: Testing a mediator model. J Couns Psychol 48:310–318.
51. Leon AC, Olfson M, Portera L, Farber L, Sheehan DV (1997): Assessing psychiatric impairment in primary care with the Sheehan Disability Scale. Int J Psychiatry Med 27:93–105.
52. Spreckelmeyer KN, Krach S, Kohls G, Rademacher L, Irmak A, Konrad K, *et al.* (2009): Anticipation of monetary and social reward differently activates mesolimbic brain structures in men and women. Soc Cogn Affect Neurosci 4:158–165.
53. Hedge C, Powell G, Sumner P (2018): The reliability paradox: Why robust cognitive tasks do not produce reliable individual differences. Behav Res Methods 50:1166–1186.
54. Infantolino ZP, Luking KR, Sauder CL, Curtin JJ, Hajcak G (2018): Robust is not necessarily reliable: From within-subjects fMRI contrasts to between-subjects comparisons. NeuroImage 173:146–152.
55. Elliott ML, Knodt AR, Ireland D, Morris ML, Poulton R, Ramrakha S, *et al.* (2020): What is the test–retest reliability of common task-functional MRI measures? New empirical evidence and a meta-analysis. Psychol Sci 31:792–806.
56. Rodebaugh TL, Scullin RB, Langer JK, Dixon DJ, Huppert JD, Bernstein A, *et al.* (2016): Unreliability as a threat to understanding psychopathology: The cautionary tale of attentional bias. J Abnorm Psychol 125:840–851.
57. Watson D, Clark LA, Tellegen A (1988): Development and validation of brief measures of positive and negative affect: The Panas scales. J Pers Soc Psychol 54:1063–1070.
58. Coyne JC (1976): Depression and the response of others. J Abnorm Psychol 85:186–193.
59. Porges SW (2007): The polyvagal perspective. Biol Psychol 74:116–143.
60. Kok BE, Fredrickson BL (2010): Upward spirals of the heart: Autonomic flexibility, as indexed by vagal tone, reciprocally and prospectively predicts positive emotions and social connectedness. Biol Psychol 85:432–436.
61. Stellar JE, Cohen A, Oveis C, Keltner D (2015): Affective and physiological responses to the suffering of others: Compassion and vagal activity. J Pers Soc Psychol 108:572–585.
62. Cyranowski JM, Zill N, Bode R, Butt Z, Kelly MA, Pilkonis PA, *et al.* (2013): Assessing social support, companionship, and distress: National Institute of Health (NIH) Toolbox Adult Social Relationship Scales. Health Psychol 32:293–301.
63. Cox RW (1996): AFNI: Software for analysis and visualization of functional magnetic resonance neuroimages. Comput Biomed Res 29:162–173.
64. Gu R, Huang W, Camilleri J, Xu P, Wei P, Eickhoff SB, Feng C (2019): Love is analogous to money in human brain: Coordinate-based and functional connectivity meta-analyses of social and monetary reward anticipation. Neurosci Biobehav Rev 100:108–128.
65. Cougle JR (2012): What makes a quality therapy? A consideration of parsimony, ease, and efficiency. Behav Ther 43:468–481.
66. Dunn BD, Widnall E, Reed N, Owens C, Campbell J, Kuyken W (2019): Bringing light into darkness: A multiple baseline mixed methods case series evaluation of Augmented Depression Therapy (ADepT). Behav Res Ther 120:103418.
67. Craske MG, Meuret AE, Ritz T, Treanor M, Dour H, Rosenfield D (2019): Positive affect treatment for depression and anxiety: A randomized clinical trial for a core feature of anhedonia. J Consult Clin Psychol 87:457–471.
68. Fredrickson BL, Mancuso RA, Branigan C, Tugade MM (2000): The undoing effect of positive emotions. Motiv Emot 24:237–258.
69. Fredrickson BL, Tugade MM, Waugh CE, Larkin GR (2003): What good are positive emotions in crises? A prospective study of resilience and emotions following the terrorist attacks on the United States on September 11th, 2001. J Pers Soc Psychol 84:365–376.
70. Tung ES, Brown TA (2020): Distinct risk profiles in social anxiety disorder. Clin Psychol Sci 8:477–490.
71. Vinograd M, Stout DM, Risbrough VB (2022): Anhedonia in post-traumatic stress disorder: Prevalence, phenotypes, and neural circuitry. Curr Top Behav Neurosci 58:185–199.
72. Hofmann SG, Hayes SC (2019): The future of intervention science: Process-based therapy. Clin Psychol Sci 7:37–50.
73. Hayden BY, Parikh PC, Deaner RO, Platt ML (2007): Economic principles motivating social attention in humans. Proc Biol Sci 274:1751–1756.