

Expectancy Effects of Placebo Neurofeedback in ADHD Treatment Seekers: A Neuropsychological Investigation

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Objective: Though there is evidence to suggest that expectancies can impact outcomes of various medical and psychological treatments, little is known about the role of expectancy effects in neurocognitive interventions, such as neurofeedback for attention-deficit/hyperactivity disorder (ADHD). The present study investigated the effects of treatment expectancies on ADHD symptom report and neuropsychological performance by using an expectancy manipulation in the context of placebo neurofeedback. **Method:** Eighty-five young adults seeking treatment for ADHD were administered 1 session of placebo neurofeedback and randomly assigned to positive or negative expectancy groups. Primary outcome measures include ADHD symptom self-report questionnaires and neuropsychological tests. **Results:** Consistent with hypotheses, participants in the positive expectancy group who received positive false feedback reported fewer ADHD symptoms at postfeedback ($p < .001$, $\eta_p^2 = .41$), whereas participants in the negative expectancy group who received negative false feedback reported more symptoms at postfeedback ($p = .01$, $\eta_p^2 = .15$). As expected, individuals who received positive expectancies also significantly improved their performance on a working memory test ($p = .002$, $\eta_p^2 = .22$); no other neuropsychological test performance was impacted by expectancies. Beliefs about neurofeedback effectiveness did not moderate or mediate expectancy effects. **Conclusion:** Results indicate that treatment expectancies impact ADHD symptom report and some neuropsychological test performance. Therefore, expectancy effects should be considered in the evaluation of outcomes for neurocognitive interventions, such as neurofeedback for ADHD.

Key Points

Question: The present study examined expectancy effects in ADHD symptom report and neuropsychological performance among young adults seeking treatment for ADHD by using an expectancy manipulation in the context of placebo neurofeedback. **Findings:** The study found evidence for expectancy effects on ADHD symptom report and working memory performance following the expectancy manipulation; however, there were no expectancy effects on sustained attention performance. **Importance:** These findings indicate that ADHD symptom report and some neuropsychological tests are susceptible to expectancy effects and therefore expectancy should be considered in the evaluation of treatment outcomes. **Next Steps:** Moving forward, it is recommended that future research investigate potential mechanisms of treatment expectancy effects and systematically compare clinically diagnosed individuals with treatment seekers.

Keywords: expectancy effects, placebo, attention-deficit/hyperactivity disorder, neurofeedback

Accumulating evidence to date highlights the relevance of assessing patients' expectancies when interpreting treatment-related outcomes. Expectancies, broadly defined as anticipatory cognitions regarding the likelihood of future behavior and experiences, influence patient responses to clinical contexts (Laferton, Kube, Salzmann, Auer, & Shedden-Mora, 2017; Peerdeman, van Laarhoven, Peters, & Evers, 2016). For example, expectancies are

associated with the progression and outcome of treatments for patients with pain (e.g., Linde et al., 2007), substance use (e.g., Kelemen, 2008), depression (e.g., Rutherford, Wager, & Roose, 2010), and Parkinson's disease (e.g., McRae et al., 2004). Further, expectancy has been identified as a key cognitive process underlying placebo and nocebo effects, phenomena in which subjective and physiological changes result from administration of an inert (placebo) treatment within a psychosocial context (Benedetti, 2009; Brown, 2015; Colloca & Miller, 2011; Price, Finniss, & Benedetti, 2008). Expecting symptom reduction after treatment (i.e., positive expectations) can lead to a genuine improvement in health status, even if the treatment has no active properties (i.e., placebo effect; Benedetti, 2009). In contrast, the nocebo effect pertains to the negative effect resulting from negative expectations.

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Two distinct types of treatment-related expectancies shape patient behavior (Kirsch, 1999; Laferton et al., 2017; Peerdeman et al., 2016). According to Kirsch's (1985) response expectancy theory, stimulus expectancies are expectancies of the occurrence of external events including beliefs that one has (or has not) received a specific treatment. Alternatively, response expectancies refer to expectancies of one's own automatic responses to the external environment, including beliefs about the likelihood that one will encounter symptom change upon receiving treatment. Among patients undergoing treatment, those who hold expectancies that they have actually received active treatment (i.e., stimulus expectancy) and who expect the treatment will lead to symptom reduction (i.e., response expectancy) can have positive health changes that mirror anticipated outcomes, irrespective of whether they receive the active or placebo treatment. These expectancies can be learned from the environment and social observations, as well as personal experiences (Colloca & Miller, 2011).

Despite the evidence for treatment-related expectancies in some health conditions, there are relatively fewer studies on expectancy effects within nonpharmacological interventions for neurocognitive dysfunctions. One such intervention is neurofeedback for attention-deficit/hyperactivity disorder (ADHD), which is purported to train individuals to self-regulate their brain activity patterns by providing real-time feedback of electrophysiological activity (Holtmann, Sonuga-Barke, Cortese, & Brandeis, 2014). While ADHD symptom improvements have been found in several neurofeedback clinical trials, a substantial number of studies show that neurofeedback was equivalent to or less effective than control treatment as assessed by symptom rating scales and neuropsychological tests (Arnold et al., 2013; Arns, Heinrich, & Strehl, 2014; Cortese et al., 2016; Lansbergen, van Dongen-Boomsma, Buitelaar, & Slaats-Willemse, 2011; Perreau-Linck, Lessard, Levesque, & Beaugard, 2010; van Dongen-Boomsma, Vollebregt, Slaats-Willemse, & Buitelaar, 2013; Vollebregt, van Dongen-Boomsma, Buitelaar, & Slaats-Willemse, 2014). Interestingly, one study of neurofeedback for ADHD found a significant correlation ($r = .44$) between the average of treatment expectancies measured across every fifth neurofeedback session and reduction in self-reported ADHD symptoms (Mayer, Blume, Wyckoff, Brokmeier, & Strehl, 2016). Further, among participants who showed generalization of their newly acquired regulation skills to trials without feedback, there was a large relationship ($r = .63$) between expectancy and self-reported inattentive symptoms. These results suggest that effects of neurofeedback, to some extent, can be due to expectancies rather than the active ingredients of the treatment itself. However, research has yet to examine treatment expectancy as a primary predictor of outcomes in neurofeedback for ADHD. If expectancies are not assessed, the interpretation of symptom changes may be difficult or, at times, incorrect.

A recent pilot study (Lee & Suhr, 2019) investigating treatment expectancies in placebo neurofeedback found evidence of expectancy effects on self-reported ADHD symptomatology for young adults seeking treatment for ADHD. Similar to designs of biofeedback studies (e.g., Shahidi & Powell, 1988; Strunk, Sutton, & Burns, 2009), participants in the study expected to receive active neurofeedback (positive stimulus expectancy), but instead received a placebo with prerecordings of feedback from another individual indicating noncontingent changes in electrophysiological activity (manipulated response expectancy). Positive false feedback (i.e.,

feedback indicating above average attention) led to decreased report of overall ADHD symptoms and negative false feedback (i.e., feedback indicating below average attention) led to increased report of overall ADHD symptoms from pre- to postexpectancy manipulation, both with large effect sizes. A similar pattern of results was found for inattentive symptoms specifically, but reliable declines in hyperactive/impulsive symptoms were only present for those given positive false feedback. These findings provide preliminary evidence that expectancy is an important psychological factor to consider when neurofeedback outcomes are measured by changes in symptom self-report, which is consistent with robust expectancy effects that have been found on self-report measures of pain, depression, and anxiety in the context of interventions (Stewart-Williams & Podd, 2004). Therefore, neurofeedback for ADHD appears to be a good model for continuing to investigate the power of expectancy effects in neurocognitive interventions.

There is also some evidence of expectancy effects on neuropsychological test performance (Fresson, Dardenne, Geurten, Anzaldi, & Meulemans, 2017; Kvavilashvili & Ellis, 1999; Magalhães De Saldanha da Gama, Slama, Caspar, Gevers, & Cleeremans, 2013; Smith & Sullivan, 2003). A review by Suhr and Wei (2013) reported average between-groups effect sizes of 0.39 for attention tasks, 0.31 for memory tests, 0.32 for motor speed tasks, and 0.68 for intellectual and executive functioning tasks across studies that examined the effect of expectancy-related cognitions on neuropsychological performance in various psychological conditions. In contrast, a study that examined expectancy effects of placebo stimulants in college students did not find significant effects on neuropsychological tests of working memory and attention, although they did show expectancy effects on self-reported symptoms (Lookatch, Fivecoat, & Moore, 2017). Given that no published studies to date have examined expectancy effects on neuropsychological test performance in the context of pre- and postoutcome evaluations for neurocognitive interventions, further research is warranted to clarify the magnitude of treatment expectancy effects on neuropsychological performance.

The purpose of the present study was to enhance understanding of the effect of treatment expectancies on both self-report and neuropsychological outcomes in neurofeedback for ADHD. To address the study's objectives, expectancies were experimentally manipulated in the context of a placebo version of neurofeedback. Young adults seeking treatment for ADHD believed they were receiving one session of active neurofeedback (positive stimulus expectancy), but instead of being presented with their real neurofeedback results, participants heard a prerecorded session with false feedback indicating above average or below average performance (positive and negative response expectancies, respectively). We hypothesized a decreased self-report of ADHD symptoms from pre- to postexpectancy manipulation for those receiving positive false feedback and increased self-report of ADHD symptoms for those receiving negative false feedback (Hypothesis 1). In addition, we hypothesized an improved performance on attention tests from pre- to postexpectancy manipulation for those receiving positive false feedback and decline in performance on attention tests for those receiving negative false feedback (Hypothesis 2). Finally, exploratory analyses were conducted to examine self-reported beliefs about neurofeedback effectiveness as moderators and mediators of hypothesized response expectancy effects.

Method

Participants

Participants were undergraduate students enrolled in introductory-level psychology courses at a Midwestern public university. Participants responded to an online study recruitment advertisement seeking individuals who were concerned that they had ADHD and offering to provide information about an alternative treatment to stimulant medication. Exclusionary criteria included self-report of: (a) diagnostic history of neurological disorders; (b) visual or auditory impairments; (c) limited fluency in English reading and writing; (d) past experience receiving neurofeedback; and (e) use of substances, including caffeine, alcohol, marijuana, and other drugs, within 12 hr before participating in the study. Individuals taking stimulant medication ($n = 1$ in positive expectancy group; $n = 7$ in negative expectancy group) were not excluded from the study, but were asked to discontinue use for at least 24 hr prior to their study session in order to avoid potential interference effects. Participants completed the study on an individual basis, provided they followed the preparatory requirements and met inclusionary criteria. Informed consent was obtained prior to participation, and course credit was provided as compensation according to the ethical standards of the university's institutional review board.

Of the 88 participants who completed the study, three participants were excluded from analyses because they did not correctly recall the false feedback they had received during the experiment as part of a manipulation check. All further data are reported on the remaining sample of 85 participants. The mean age of the sample was 19.08 ($SD = 1.07$, range = 18–22 years). Fifty participants (58.8%) identified as female, 54 participants (63.5%) were in their first year of college, and 70 participants (82.4%) reported right-hand dominance. Sixty-six of the participants identified as Caucasian/White (77.6%), whereas seven identified as African American/Black (8.2%), four identified as Hispanic/Latino (4.7%), three identified as Asian American/Pacific Islander (3.5%), and five identified as multiracial or other (5.9%).

Apparatus

A brain sensing headband called the Muse (2014 edition, Interaxon, Toronto, Ontario, Canada) was used as a placebo neurofeedback device. Muse is advertised as a training tool that helps improve cognitive function, attention, concentration, and decreases stress. The Muse headband contains seven calibrated sensors—two on the forehead, two behind the ears, and three reference sensors—that detect and measure brain activity primarily in the frontal lobe. In addition to the Muse headband, an accompanying Muse app on an iPad tablet was used to calibrate the headband. The Muse electroencephalography software, MuseLab, installed onto a desktop computer was used to record electrophysiological activity while playing prerecorded false feedback during a focused-attention meditation task. Although the duration of an actual neurofeedback session is longer than 5 min, the aim of the present study was to elicit an expectancy effect on outcomes within a neurofeedback treatment protocol. A 5-min session was selected based on prior research on biofeedback relaxation therapy, which demonstrated that even receiving biofeedback for five minutes yielded significant differences pre- and postbiofeedback irrespec-

tive of whether participants received real or false feedback (Strunk et al., 2009).

Measures

Demographics and health history. Participants provided information about their age, educational history, sex at birth, gender identity, racial/ethnic background, and dominant handedness. Participants were also inquired about their level of concern that they may have ADHD. Subsequent questions asked participants about diagnosis and treatment for ADHD symptoms, diagnosis and treatment for other psychological conditions, history of head injury, concussion, seizures, neurological disorders, and medical conditions. Participants were also asked questions about their sleep and any substance use.

ADHD symptom self-report. The Adult ADHD Self-Report Scale (ASRS) was administered as a screener for ADHD symptoms to characterize the sample at baseline (Adler et al., 2006; Kessler et al., 2005). Respondents were asked to indicate the frequency of the occurrence of symptoms over the past 6 months on a 5-point Likert-type scale ranging from *never* to *very often*. The first six items are used as an ADHD screener, while the remaining 12 items are used to serve as further probes into the individual's symptoms. The ASRS six-item screener has outperformed the 18-item scale in sensitivity (68.7% and 56.3%, respectively), specificity (99.5% and 98.3%, respectively), and total classification accuracy (97.9% and 96.2%, respectively; Kessler et al., 2005) in adults. The ASRS screener has also demonstrated internal consistency in the range of .63–.72 and test–retest reliability in the range of .58–.77 for adult samples (Kessler et al., 2007). A clinical cutoff of 14 or greater (0–13 vs. 14–24) for the ASRS screener was predictive of ADHD diagnoses (area under the curve = .79; Kessler et al., 2007). With this scoring approach, the internal consistency of the six-item ASRS screener was .75 in the present study.

In addition, the State Attention and Arousal Scale (SAAS), adapted from the Barkley Adult ADHD Rating Scale-IV Self-Report Current Symptoms Scale (BAARS-IV; Barkley, 2011), was developed and used as a measure of self-reported current ADHD symptoms at pre- and postexpectancy manipulation. The intention of the SAAS was to serve as a state measure of ADHD symptoms, rather than a clinical scale of ADHD symptoms experienced during the past 6 months. The SAAS consists of 18 BAARS-IV self-report items describing attention and arousal states. For example, the BAARS-IV item, “fidgeting with hands or feet or squirm in seat” was modified to the SAAS item, “I feel fidgety.” Respondents were asked to indicate to what extent each statement best describes their current experience of symptoms by responding on a 4-point Likert scale, where 1 = *not at all*, 2 = *mildly*, 3 = *moderately*, and 4 = *severely*. A total ADHD symptom score and subscale scores for inattentive and hyperactive/impulsive symptoms were calculated to test the study's main hypotheses. The SAAS has demonstrated high internal consistency in the pilot study conducted by Lee and Suhr (2019; $\alpha = .85-.91$). Internal consistencies in the present study were .91 and .89 for the total score pre- and postexpectancy manipulation, respectively.

Neurofeedback beliefs questionnaire. Following an explanation of the neurofeedback treatment at baseline, participants were instructed to rate their beliefs about the effectiveness of

neurofeedback for improving symptoms related to ADHD on a 4-point Likert scale, where 0 = *not effective*, 1 = *slightly effective*, 2 = *effective*, and 3 = *very effective*. Participants were also inquired of their beliefs about neurofeedback effectiveness following the placebo administration.

Neuropsychological tests. The AX Continuous Performance Test (AX-CPT; [Marcora, Staiano, & Manning, 2009](#)) was administered to assess sustained and divided attention, as well as impulsivity. The AX-CPT is an experimental version of the continuous performance test in which sequences of letters are visually presented one at a time in a continuous fashion on a computer screen. Participants are instructed to respond differently to target and nontarget trials. Target trials are defined as a cue-probe sequence in which the letter A appears as the cue and the letter X appears as the probe. The remaining letters of the alphabet serve as invalid cues and nontarget probes. Two white distractor letters (which could be any letter but A, K, X, or Y) are also presented between the red cue and red probe. All letters are presented centrally on a black background for a duration of 100 ms and each letter is followed by a 1,000-ms interval. Thus, one trial involves presentation of a cue, followed by two distractors, which are followed by a probe. Any missed or incorrect response elicits a bleep sound. The AX-CPT has demonstrated moderate test-retest reliabilities ranging from .65 to .74 for hits, misses, and hit response time in children and adolescents ([Halperin, Sharma, Greenblatt, & Schwartz, 1991](#)). In the present study, AX-CPT performance at pre- and postexpectancy manipulation were measured as overall percentage of correct responses and overall mean hit response time for correct responses over 300 trials.

The Auditory Consonant Trigram (ACT) test, also known as the Brown-Peterson technique ([Peterson & Peterson, 1959](#)), was administered to measure working memory ([Lezak, Howieson, Bigler, & Tranel, 2012](#)). Several versions of the test have been adapted for research and clinical use, but the test typically consists of an auditory presentation of three consonants (e.g., X-C-P) followed by a number (e.g., 194). Upon hearing the three consonants and a number, participants are instructed to count aloud backward by 3s as a distractor task (e.g., 194, 191, 188, etc.) for varying lengths of time, ranging 0-, 3-, 9-, 18-, or 36-s intervals. Once the participant is told to stop counting, they are asked to recall the three consonants. When using the ACT with 9-, 18-, and 36-s distraction durations, previous studies have found small practice effects when using alternate forms and no statistically significant differences in performance according to sex, age, or education level (e.g., [Stuss, Stethem, & Poirier, 1987](#)). The ACT has also demonstrated good test-retest reliability ($r = .63$) among individuals with mean age of 20.83 ([Mertens, Gagnon, Coulombe, & Messier, 2006](#)), as well as adequate internal consistency ($\alpha = .79$; [Shura, Rowland, & Miskew, 2016](#)). In the present study, alternate forms consisting of 15 different trials with 9-, 18-, and 36-s distraction durations were administered at pre- and postexpectancy manipulation, with performance measured as a total score across trials.

Procedure

Upon providing informed consent and compliance with the study's preparatory instructions, each participant completed a series of baseline self-report questionnaires that assessed demographic and health history, as well as both "trait" (ASRS) and

"state" (SAAS) ADHD symptoms. Participants subsequently were administered the AX-CPT and ACT. Prior to administration of the placebo neurofeedback (Muse), participants received standardized information about neurofeedback, including the sequence of events they would experience during the neurofeedback session and the different types of feedback they might hear that was contingent on their brain activity during the neurofeedback task. Participants were instructed that if their attention was truly concentrated on their breathing, they would hear calm winds and birds chirping. However, if their attention was fluctuating, the winds would pick up and no bird sounds would be heard. Participants were informed that their goal was to quiet the wind and hear bird sounds. Immediately after receiving these standardized instructions, participants responded to questions regarding their beliefs about neurofeedback. To increase the salience of their expectancies, participants observed the calibration of the neurofeedback device and completed a 1-min "practice" session.

Participants were randomly assigned to one of two experimental conditions: (a) the positive expectancy group, for which participants heard a prerecorded session indicating high neurofeedback success (i.e., audio feedback of calm winds and many birds chirping) and received verbal feedback from the experimenter that they were concentrating very well, or (b) the negative expectancy group, for which participants heard a prerecorded session indicating low neurofeedback success (i.e., audio feedback of loud winds and no bird sounds) and received verbal feedback that they were not concentrating well. Following the calibration and practice session, participants were administered a 5-min session of placebo neurofeedback during which they received either positive or negative false feedback according to the experimental condition. Participants then completed the second set of self-report questionnaires that assessed ADHD symptoms (SAAS) and postmanipulation beliefs about neurofeedback effectiveness. Subsequently, participants were administered neuropsychological tests (AX-CPT, ACT), followed by the manipulation check and debriefing. Participation lasted 2 hr.

Data Analytic Plan

Data were analyzed using SPSS Statistics (Version 25). Demographic and psychological characteristics of participants were compared by experimental group using chi-square and analysis of variance (ANOVA) tests. Outcome measures were screened for missing data, presence of outliers, normality of the distributions, sphericity, and homogeneity of variances and covariances. Tests for violations of assumptions were conducted and are reported when significant. Mixed ANOVAs, with experimental group (positive vs. negative) as a between-subjects factor and time of measurement (Time 1 vs. Time 2) as a within-subject factor, were used to evaluate effects of the expectancy manipulation on SAAS total score, SAAS subscale scores, AX-CPT overall percent correct, AX-CPT mean hit response times, and ACT total score. The interaction between experimental group and time of measurement was the critical statistical analysis to test the study hypotheses. Effect sizes were calculated and interpreted using partial eta squared to indicate the magnitude of group differences ([Cohen, 1973](#)). Exploratory analyses examining beliefs about neurofeedback effectiveness in relation to type of expectancy were tested using mixed ANOVA. To determine whether baseline neurofeed-

back beliefs moderate the effect of expectancy group on changes in symptom report and neuropsychological test performance, as well as whether postmanipulation neurofeedback beliefs mediate the effect of expectancy group on those outcomes, moderated mediation was conducted using Model 5 of the PROCESS macro (Hayes, 2018). Separate models were tested to predict changes in SAAS total score, AX-CPT scores, and ACT total score.

Results

Descriptive Statistics

Groups did not differ in age, $F(1, 83) = .01, p = .94$, sex at birth, $\chi^2(1) = 1.62, p = .20$, gender identity, $\chi^2(2) = 2.28, p = .32$, White/non-White race/ethnicity, $\chi^2(1) = .19, p = .66$, current year in college, $\chi^2(3) = 1.30, p = .73$, average GPA, $F(1, 76) = .08, p = .78$, or dominant handedness, $\chi^2(1) = .19, p = .66$. Groups differed in history of ADHD diagnosis, $\chi^2(1) = 4.52, p = .03$, with the negative expectancy group including more individuals with ADHD diagnostic history ($n = 7$) than the positive expectancy group ($n = 1$). Those who reported history of ADHD diagnosis were the same individuals who reported stimulant medication use. There were no significant differences in scores on the ADHD screener, $F(1, 83) = 2.24, p = .09$, baseline ADHD symptom report, $F(1, 83) = .33, p = .57$, and baseline neuropsychological performance, $ps > .05$, between participants who reported ADHD diagnostic history and those without ADHD diagnosis. The groups did not differ in their level of concern about having ADHD, $F(1, 75) = .74, p = .39$, history of diagnosis of other psychological disorders, $\chi^2(1) = .04, p = .84$, or number of individuals with scores above the clinical cutoff on the ADHD screener, $\chi^2(1) = .53, p = .47$. Twenty-six participants fell above

the ASRS clinical cutoff, with 11 in the positive expectancy group and 15 in the negative expectancy group.

H_1 : Expectancy Effects on Self-Report of ADHD Symptoms

As hypothesized, there was a significant Group \times Time interaction for SAAS total score, $F(1, 83) = 30.67, p < .001, \eta_p^2 = .27$ (Table 1). As expected, groups did not differ in total ADHD symptoms at preexpectancy manipulation, $F(1, 83) = .14, p = .70, \eta_p^2 = .002$, but differed at postexpectancy manipulation, $F(1, 83) = 18.95, p < .001, \eta_p^2 = .19$, with the negative expectancy group reporting higher ADHD symptoms than the positive expectancy group at postexpectancy manipulation. Furthermore, consistent with hypotheses, participants in the positive expectancy group who received positive false feedback reported significant decreases in total ADHD symptoms from pre- to postexpectancy manipulation, $F(1, 40) = 28.04, p < .001, \eta_p^2 = .41$, whereas participants in the negative expectancy group who received negative false feedback reported significant increases in total ADHD symptoms from pre- to postexpectancy manipulation, $F(1, 43) = 7.34, p = .01, \eta_p^2 = .15$.

Supplemental analyses of SAAS subscales scores were conducted to investigate expectancy effects on ADHD symptom dimensions (i.e., inattention, hyperactivity/impulsivity). The Inattention subscale score showed a similar pattern to the SAAS total score findings, with a significant Group \times Time interaction, $F(1, 83) = 28.25, p < .001, \eta_p^2 = .25$. As with total symptoms, groups did not differ in inattentive symptoms at preexpectancy manipulation, $F(1, 83) = .20, p = .65, \eta_p^2 = .002$, but differed at postexpectancy manipulation, $F(1, 83) = 24.19, p < .001, \eta_p^2 = .23$, with the negative expectancy group reporting significantly

Table 1
Means (Standard Deviations) of ADHD Symptoms and Neuropsychological Test Scores

Measure	Group	
	Positive expectancy ($n = 41$), M (SD)	Negative expectancy ($n = 44$), M (SD)
SAAS total score		
Time 1	33.56 (8.64)	32.84 (8.81)
Time 2	28.61 (5.72)	35.64 (8.74)
SAAS Inattention score		
Time 1	13.19 (4.31)	12.75 (4.74)
Time 2	10.63 (3.06)	15.02 (4.89)
SAAS Hyperactivity/Impulsivity score		
Time 1	5.29 (1.99)	5.48 (2.34)
Time 2	4.00 (1.04)	4.93 (1.26)
AX-CPT percent correct		
Time 1	79.48 (21.57)	78.67 (21.29)
Time 2	86.21 (18.61)	85.73 (14.69)
AX-CPT mean hit response time		
Time 1	426.55 (64.68)	404.93 (85.36)
Time 2	411.18 (78.74)	382.08 (52.53)
ACT total score		
Time 1	30.56 (6.59)	31.25 (7.30)
Time 2	33.80 (7.23)	30.18 (9.16)

Note. Percent correct in percentage values. Hit mean response time in milliseconds. ADHD = attention-deficit/hyperactivity disorder; Time 1 = preexpectancy manipulation; Time 2 = postexpectancy manipulation; SAAS = State Attention and Arousal Scale; AX-CPT = AX Continuous Performance Test; ACT = Auditory Consonant Trigram test.

higher inattentive symptoms at postexpectancy manipulation relative to the positive expectancy group. Further, consistent with hypotheses, participants in the positive expectancy group who received positive false feedback reported significant decreases in inattentive symptoms from pre- to postexpectancy manipulation, $F(1, 40) = 21.77, p < .001, \eta_p^2 = .35$, whereas participants in the negative expectancy group who received negative false feedback reported significant increases in inattentive symptoms from pre- to postexpectancy manipulation, $F(1, 43) = 10.15, p = .003, \eta_p^2 = .19$. There was no significant Group \times Time interaction, $F(1, 83) = 3.24, p = .07, \eta_p^2 = .04$, or main effect of group, $F(1, 83) = 2.05, p = .16, \eta_p^2 = .02$, for the Hyperactivity/Impulsivity subscale score. However, there was a significant main effect of time, $F(1, 83) = 19.62, p < .001, \eta_p^2 = .19$, such that, collapsed across groups, participants reported less hyperactivity/impulsivity symptoms at postexpectancy manipulation.

H₂: Expectancy Effects on Neuropsychological Test Performance

Group distributions for overall percentage of correct responses on the AX-CPT were negatively skewed and kurtotic across both time points. A log base 10 transformation improved the normality of the group distributions and was used in analyses of AX-CPT scores. No other test assumptions were violated. Contrary to expectations, there was no significant Group \times Time interaction, $F(1, 83) = .42, p = .52, \eta_p^2 = .005$, or main effect of group, $F(1, 83) = 1.50, p = .22, \eta_p^2 = .018$. However, there was a significant main effect of time, $F(1, 83) = 27.64, p < .001, \eta_p^2 = .25$, such that, collapsed across groups, the percentage of correct responses increased from pre- to postexpectancy manipulation. In addition, there was no significant Group \times Time interaction, $F(1, 83) = .28, p = .60, \eta_p^2 = .003$, or main effect of group, $F(1, 83) = 2.87, p = .09, \eta_p^2 = .03$, for mean hit response times on the AX-CPT. However, there was a significant main effect of time, $F(1, 83) = 7.20, p = .009, \eta_p^2 = .080$, such that, collapsed across groups, there were faster mean hit response times from pre- to postexpectancy manipulation.

With regard to the ACT, as hypothesized, there was a significant Group \times Time interaction, $F(1, 83) = 12.99, p = .001, \eta_p^2 = .13$ (see Table 1). Groups did not differ in ACT total score at preexpectancy manipulation, $F(1, 83) = .21, p = .65, \eta_p^2 = .002$, but differed at postexpectancy manipulation, $F(1, 83) = 4.06, p = .04, \eta_p^2 = .05$. Postexpectancy manipulation ACT performance was significantly lower for the negative expectancy group relative to the positive expectancy group, $p = .05$. Furthermore, consistent with hypotheses, participants in the positive expectancy group demonstrated significant improvements in ACT performance from pre- to postexpectancy manipulation, $F(1, 40) = 11.28, p = .002, \eta_p^2 = .22$. Participants in the negative expectancy group who received negative false feedback demonstrated nonsignificant, but moderately sized decline in ACT performance from pre- to postexpectancy manipulation, $F(1, 43) = 2.17, p = .15, \eta_p^2 = .05$.

Exploratory Analyses: Effects of Beliefs About Neurofeedback Effectiveness

There was a significant Group \times Time interaction, $F(1, 83) = 6.03, p = .02, \eta_p^2 = .07$, for beliefs about neurofeedback effec-

tiveness. Simple effect analyses revealed groups did not differ in self-reported beliefs about neurofeedback effectiveness at preexpectancy manipulation, $F(1, 83) = 1.17, p = .28, \eta_p^2 = .01$, or postexpectancy manipulation, $F(1, 83) = 2.94, p = .09, \eta_p^2 = .03$. While participants in the positive expectancy group who received positive false feedback did not report changes in beliefs about neurofeedback effectiveness, $F(1, 40) = .12, p = .73, \eta_p^2 = .003$, participants in the negative expectancy group who received negative false feedback reported significantly lower beliefs about neurofeedback effectiveness from pre- to postexpectancy manipulation, $F(1, 44) = 8.80, p = .005, \eta_p^2 = .17$.

Moderated mediation models revealed no significant interactions between expectancy type and baseline neurofeedback beliefs for all outcome measures, $ps > .05$. There was a significant relationship between expectancy group and postmanipulation neurofeedback beliefs, $b = -.48, t(83) = -2.39, p = .02$. There was also a significant effect of postmanipulation neurofeedback beliefs on change in SAAS total score, $b = -3.08, t(83) = -3.37, p = .001$, but not for other outcome measures, $ps > .05$. The indirect effects of expectancy type on change in SAAS total score was .69 (95% CI [-.10, 1.89]), change in AX-CPT percent correct was -.33 (95% CI [-2.88, 2.67]), change in AX-CPT mean response times was -.36 (95% CI [-8.56, 6.68]), and change in ACT total score was -.11 (95% CI [-.84, .49]). Thus, the indirect effects were not statistically significant.

Discussion

Previous research has demonstrated the importance of expectancy effects on treatment-related outcomes, though only one prior study has examined the role of expectancy effects in nonpharmacological interventions for neurocognitive dysfunctions, such as neurofeedback for ADHD (Lee & Suhr, 2019). The goal of the present study was to investigate the effects of treatment expectancies on self-report and neuropsychological outcomes in neurofeedback for ADHD treatment seekers. Results support the hypothesis that treatment expectancies influence self-report of current ADHD symptoms. All participants self-reported similar levels of overall ADHD symptoms at baseline, but changed their self-report of symptoms after the response expectancy manipulation. As predicted, participants who received false feedback inducing negative expectancies increased their self-report of overall ADHD symptoms, whereas participants who received false feedback inducing positive expectancies decreased their self-report of overall ADHD symptoms. These findings are in accordance with prior research demonstrating that expectancies can significantly influence symptom reports in positive and negative directions (Benedetti, 2009; Lee & Suhr, 2019). The fact that robust expectancy effects on symptom self-reports were obtained over a short time interval is notable and suggests that treatment expectancies should be considered when evaluating treatment outcomes using symptom report measures.

Differential effects of treatment expectancies on ADHD symptom dimensions also emerged. Participants who received false feedback inducing negative expectancies increased their self-report of inattentive symptoms, whereas those who received false feedback inducing positive expectancies decreased self-report of inattentive symptoms. Given that the placebo neurofeedback involved an attention meditation task, it is not surprising that there

were large effects of response expectancies on inattentive symptoms. However, treatment expectancies did not influence self-report of hyperactive/impulsive symptoms. Although these results contrast with findings reported by Lee and Suhr (2019) of expectancy effects on self-report of both ADHD symptom dimensions, Mayer and colleagues (2016) found that self-reported expectancy correlated with improvements in only inattentive symptoms among individuals who successfully learned the neurofeedback task. Taken together, it appears that expectancies have a particularly robust effect on inattentive symptom report. Future studies are necessary to clarify why response expectancies in neurofeedback have a stronger effect on reports of inattentive symptoms of ADHD.

Relative to the large effects of expectancies found on self-reported ADHD symptoms, there were mixed results of expectancy effects on neuropsychological test performance. All participants improved on the sustained attention test over time, regardless of type of expectancy. In contrast, there were effects of treatment expectancies on working memory performance. Participants who received false feedback inducing positive expectancies improved their performance on the working memory test over time. Although not significant, participants who received false feedback inducing negative expectancies demonstrated moderately sized decline in performance on the working memory test. It is possible that the continuous performance test was not difficult enough to assess change, as there were ceiling effects across both groups. In addition, it may be that expectancies have a sizable effect on certain types of neuropsychological functions as opposed to others. For example, in a study that manipulated expectancies regarding the properties of a placebo EEG (stimulus expectancy) and the device's capacity to affect perception of colors on an inhibition task (response expectancy), participants who were told that the EEG device would improve their visual perception (positive response expectancies) showed decreased interference on the Stroop test compared to baseline, whereas those who were told that the EEG device would impair their visual perception (negative response expectancies) showed increased interference (Magalhães De Saldanha da Gama et al., 2013). In contrast, Lookatch and colleagues (2017) found that performance on attention tests was unaffected by expectancies. Future studies should investigate expectancy effects on neuropsychological tests beyond sustained attention and working memory to add to these findings.

In addition, self-reported ratings of beliefs about neurofeedback effectiveness significantly changed across time, but only for participants who received false feedback inducing negative expectancies. These results suggest that negative feedback about one's treatment progress may have greater impact on beliefs about treatment effectiveness. Results of exploratory analyses, however, indicated that beliefs about neurofeedback effectiveness did not moderate or mediate expectancy effects on ADHD symptom report or neuropsychological test performance. According to prior research on expectancy theory, positive expectancies can strengthen beliefs about treatment effects and increase the likelihood of treatment use compared to negative expectancies which may reduce beliefs about treatment effects (Benedetti, 2009; Kirsch, 1985). This finding, coupled with the existing literature, suggests that negative expectancies may impact beliefs about treatment effectiveness more than positive expectancies. However, these

results require replication in follow-up studies with larger sample sizes.

Determining whether there are expectancy effects on subjective and objective outcomes is a useful step toward understanding treatment expectancies. Evidence of sizable expectancy effects on symptom report and working memory test performance, but lack of expectancy effects on sustained attention performance suggest that the effect of expectancies may be limited to certain neuropsychological domains. However, it is also possible that different mechanisms are involved in positive and negative expectancy effects on self-report measures relative to test performance. For example, anxiety and reward mechanisms have been proposed as potential mechanisms through which expectancies affect health status (Benedetti, 2014). It would be particularly valuable to investigate the role of anxiety in expectancy effects because anxiety has been shown to significantly impact cognitive task performance (Clarke & MacLeod, 2013). Moving forward, it is recommended that researchers continue to evaluate underlying mechanisms of expectancy effects on both subjective and objective outcomes.

As with all studies, the present results should be interpreted considering several study limitations. The sample constituted predominantly Caucasian college students who were seeking treatment for ADHD and only eight of the participants endorsed a history of ADHD diagnoses. Although individuals diagnosed with ADHD represent a minor percentage of the study sample and less than half of the sample (31%) endorsed a pattern of symptoms highly consistent with adult ADHD on a screening measure, it is notable that participants were concerned enough about having ADHD to seek a treatment for ADHD. However, it cannot be assumed that the results of this study will generalize to individuals diagnosed with ADHD or more diverse samples. Furthermore, caution in interpretation of results in relation to active neurofeedback is warranted until the findings are replicated with active form of the treatment, rather than placebo. Prior research indicates that expectancy effects are not limited to placebos. For example, studies report that patients who expect to receive treatment (i.e., open administration) show significantly greater reduction in symptoms than those who do not expect to receive treatment (i.e., hidden administration) even when the same active treatment is provided (e.g., Benedetti et al., 2003; Colloca, Lopiano, Lanotte, & Benedetti, 2004). The lower effectiveness of hidden, or unexpected, treatments suggests that the psychosocial factors that are involved in the process of treatment administration (e.g., clinician-patient communication) may lead to significant changes in health outcomes (Colloca & Miller, 2011). Therefore, it would be beneficial for studies of active neurofeedback to include measures of expectancies and investigate expectancy effects in relation to treatment outcomes.

Despite these limitations, the results have valuable implications. Given the significant expectancy effects found on self-report of ADHD symptoms and working memory test performance, clinicians should consider maximizing positive expectancy effects and minimizing negative expectancy effects when administering interventions for neurocognitive dysfunctions, such as neurofeedback. For example, neurofeedback training sessions typically involve patients receiving reinforcements to help them activate areas of the brain thought to be underaroused during the neurofeedback task (Holtmann et al., 2014). The feedback that is provided to patients via the neurofeedback training program and interactions with the

therapist can affect whether they perceive themselves as succeeding at the neurofeedback task and can impact their beliefs about the effectiveness of neurofeedback for their symptoms. These expectancy effects may, in turn, reinforce their engagement in treatment. Although additional evidence from studies of active neurofeedback are necessary to corroborate these findings, merely minimizing negative expectancy effects during neurofeedback training may be enough to help patients improve in their health status.

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