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Does physical anhedonia play a role in depression? A 20-year longitudinal study

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Abstract

Background—Anhedonia towards physical or sensory experiences (i.e., physical anhedonia) has most often been examined as a differentia of schizophrenia and not depression, despite the fact that general anhedonia is a core feature of many models of Major Depressive Disorder (MDD).

Methods—Forty-nine participants with non-psychotic MDD were recruited from inpatient settings and followed-up six times over 20 years. The three aims of the study was to assess a) the stability of physical anhedonia over time, b) whether physical anhedonia relates to the course of depressive symptoms over time, and c) whether physical anhedonia relates to three domains of functioning – work, social functioning, or re-hospitalizations.

Results—We found that over time physical anhedonia was relatively stable and related to depressive symptoms (both between and within person). Physical anhedonia was also related to certain aspects of functioning, though less robustly than depressive symptoms.

Limitations—Because depressive symptoms, functioning, and physical anhedonia were measured concurrently at each follow-up, the direction of causality among these variables could not be assessed. Additionally, because our sample was recruited from inpatient settings, our findings may not generalize to individuals with less severe depression.

Conclusions—A trait tendency to experience decreased pleasure to positive physical stimuli is a clinically meaningful variable for those with MDD and may be a behavioral endophenotype for a more severe form of depression.

Introduction

Numerous theoretical conceptualizations of Major Depressive Disorder (MDD) have highlighted anhedonia as a core characteristic of the disorder (Davidson et al., 2000; Klein, 1987; Meehl, 1975; Shankman and Klein, 2003). Moreover, trait-like tendencies to experience anhedonia have been evaluated as a risk factor for MDD. Trait measures of anhedonia have been shown to be elevated both during a depressive episode (Kendall and DiScipio, 1968; Reich et al., 1987) and when remitted (Hirschfeld and Klerman, 1979; Hirschfeld et al., 1983a; Reich et al., 1987), and predict the course of depression (Kerr et al., 1974; Kendler et al., 1997). Anhedonia has also been shown to have etiological significance as it is associated with family history of depression (Hecht et al., 1998; Hirschfeld et al., 1983a, Schrader,

Conflicts of Interest

We have no conflicts of interest to disclose.

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1997) and in longitudinal studies, predicts the onset of depression later in life (van Os et al., 1997).

There are, of course, several types of things that one can be anhedonic about – romantic relationships, professional success, etc. One specific type of anhedonia is Physical Anhedonia (PhA), which is an absence of pleasure from physical or sensory experiences, such as eating, touching, feeling, sex, temperature, movement, smell, and sound (Chapman et al., 1976). Interestingly, PhA has received very little attention in the depression literature and has mostly been examined in relation to schizophrenia. For example, PhA has been proposed to be a stable, trait-like risk factor for the development of schizophrenia (Bernstein and Riedel, 1987; Chapman et al., 1976; Herbener and Harrow, 2002; Loas et al., 2009; Meehl, 1962). It has been associated with poor premorbid functioning (Katsanis et al., 1992; Schuck et al. 1984), poor prospective social and work functioning (Freedman et al., 1998; Herbener et al., 2005), and has been shown to have pathoplastic effects on the course of psychosis (Herbener and Harrow, 2002).

Thus, the purpose of this study is to examine the role of PhA in depression. There are several ways to look at the role of PhA in a sample of depressed individuals. The present study examines three, using data from the Chicago Follow-Up Study (Herbener and Harrow, 2001, 2002; Westermeyer and Harrow, 1986), a 20-year prospective longitudinal study of inpatients with severe psychopathology. First, we will examine the stability of PhA over time in individuals with depression. This is an important first step in order to determine how trait-like PhA is. Second, we will examine whether PhA predicts the course of depressive symptoms over time (i.e., pathoplastic effects). Third, we will examine whether PhA predicts work or social functioning or re-hospitalization. This speaks to the clinical utility of the construct of PhA in individuals with depression. Interestingly, in schizophrenia, studies have shown that PhA is stable, relates to depressive symptoms, and predicts functional outcome (Herbener and Harrow, 2002; Herbener et al., 2005; Loas et al., 2009). The present study sought to address these questions in a sample of depressed individuals.

The few studies that have examined the role of PhA in depression have only looked at between (as opposed to within) person effects. That is, studies have only looked at whether a person's average PhA predicts that person's average score on some other variable. For example, Loas and colleagues found the between subject effect that depressed patients with higher PhA also had greater depression severity (Loas et al., 1992; Loas and Boyer, 1993). Within person effects, however, are equally important as they examine whether an individual's fluctuations in PhA over time predict individual fluctuations in some other variable over time. Even if PhA is relatively stable over time, fluctuations in PhA may have significant correlates. One of the few studies to examine within person effects reported that PhA did not predict within person changes in depressive symptoms at an 18-month follow-up (Katsanis et al., 1992). In the present study, we examined both within and between subjects effects of PhA.

Methods

Participants

Data come from the Chicago Follow-Up Study, a longitudinal study of individuals with a variety of diagnoses (i.e. schizophrenia, schizoaffective disorder, bipolar disorder, MDD) recruited from inpatient hospitals in the Chicago area (Goldberg and Harrow; 2004, 2005; Harrow et al., 1990; Harrow et al., 1997; Harrow and Jobe, 2007; Herbener and Harrow, 2001, 2002; Herbener et al., 2005). Patients were prospectively followed regularly at 2-years, 4.5-years, 7.5-years, 10-years, 15-years, and 20-years after the inpatient index assessment.

The following analyses focused on a subsample of 63 patients who at baseline inpatient hospitalization met criteria for non-psychotic MDD. We excluded those with psychosis in order to have a more homogeneous sample and to distinguish the results from studies on psychosis and PhA. Baseline diagnoses were made utilizing Research Diagnostic Criteria (Spitzer et al., 1978) based on a combination of structured diagnostic research interviews, admission interviews, and detailed inpatient observations for all patients at index hospitalization. At each follow-up, patients were interviewed either in person or via telephone using the Schedule for Affective Disorders and Schizophrenia (SADS, Endicott and Spitzer, 1978), a semi-structured interview used extensively in research. The interviews assessed current symptomatology as well as psychopathology in the past year. Satisfactory inter-rater reliability for diagnoses has been reported previously for the Chicago Follow-Up Study (Pogue-Geile and Harrow, 1984).

Fourteen of the 63 patients with MDD at baseline developed either a manic or psychotic episode at one point during the follow-up. Thus, in order to restrict the sample to those who remained unipolar and non-psychotic, these patients were excluded, yielding an *N* of 49. Of these 49, follow-up evaluations were complete on 95.9% at the 2-year follow-up, 89.8% at the 4.5-year follow-up, 85.7% at the 7.5-year follow-up, 100% at the 10-year follow-up, 93.9% at the 15-year follow-up, and 79.6% at the 20-year follow-up. Because of the length of the assessment protocol at each follow-up, some participants did not complete the self-report scales. Thus, the rates of missing values for the different measures varied across follow-ups (see measures section). However, as noted below, one of the advantages of IGC analyses is that it includes participants with incomplete data.

Demographics and clinical characteristics of the sample are presented in Table 1. Socioeconomic status was rated using the Hollingshead index (Hollingshead and Redlich, 1958). This index is based on the parent's occupation and education when the participant was 18 years old, and ranges from 1 to 5, with higher scores indicating lower socioeconomic status.

Measures

Physical anhedonia—To measure PhA, we used the Revised Physical Anhedonia Scale, the most widely used index of PhA, developed and revised by Chapman and colleagues (Chapman et al., 1976). This 61-item self-report scale was designed to assess the degree to which people receive pleasure from physically sensual experiences, such as feeling velvet or fur, smelling fresh baked bread, or watching a sunset. Higher scores indicated a greater amount of PhA. At each of the assessment, Cronbach's alphas were high, ranging from .78 (10-yr FU) to .90 (15-yr FU). The 49 participants had a median of three PhA assessments across the 6 FUs.

Depressive symptoms—Depressive symptoms were assessed at each follow-up using the composite depressed mood and behavior score (subscale-12) from the Katz Adjustment Scales (KAS; Katz and Lyster, 1963). The KAS is a 55-item self-report instrument assesses various domains of distress, symptoms, and adjustment during the previous few weeks and was the precursor to the SCL-90 (Derogatis et al., 1973). Example of items for subscale-12 include 'feeling blue,' 'having no interest in things' and 'blaming oneself.' Of note is that nearly identical results were obtained when, instead of the KAS, we used the SADS variables 'depressed mood in last year' and 'anhedonia/loss of interest in last year' We chose to focus on the KAS instead of these SADS variables because the rating period was closer to when participants filled out the PhA measure (previous few weeks vs. last year). Participants had median of 6 of 6 KAS assessments and only two participants missed more than two.

Social and work functioning and hospitalization—Standardized measures of functional outcome (Strauss and Carpenter, 1972) were used to assess work and social adjustment and re-hospitalization at each follow-up assessment. The social functioning scale

assessed the frequency and context of social interactions in the month preceding assessment, ranging from 'none' to 'chance encounters with others' to 'regularly planned meetings several times a month.'

The work functioning scale assessed the portion of the prior year during which the subject had been employed, including part-time or partial year employment. In this sample, this variable was skewed as the vast majority of patients were employed full-time at each assessment. Thus, this variable was dichotomized to 1=working full-time and 0=not working full-time. At each assessment, we coded whether each patient was re-hospitalized during the previous year. Participants had median of 6 of 6 social functioning, work functioning, and hospitalization assessments and only two participants missed more than two.

Data Analysis

Analyses were conducted with individual growth curve modeling (IGC; Raudenbush and Bryk, 2002) using the software package HLM6.06. This approach hypothesizes a growth trajectory for each patient. IGC has several advantages over other repeated measures data analytic strategies as it can: a) take into account the hierarchical structure of the data (e.g., observations nested within person), b) examine the within person stability or change over multiple assessments rather than being limited to comparisons between pairs of assessments; and c) model each person's slope (i.e., rate of within-person change) and intercept (i.e., estimated 'beginning' of trajectory when they entered the study) even if they do not have data for all assessment points (Singer and Willett, 2003).

In these analyses, we examined both intercepts and slopes across time where time was defined as the month since baseline that participants completed the assessment. It was important to model the precise month (rather than using the time points outlined in the *Participants* section) as there was some variation in the actual month that participants were assessed. We do not report level-2 predictors of individual trajectories in this study (e.g., gender), as we wanted to focus on the overall role of PhA over time.

For IGC models predicting work functioning and hospitalization, we ran Bernoulli models as these were dichotomous outcomes (e.g., hospitalized or not; see *Measures*).

For the between subjects analyses, we conducted linear regressions for continuous outcomes (e.g., work functioning) and logistic regressions for dichotomous outcomes (e.g., hospitalization).

Results

Patients had dramatically deviant scores on PhA during baseline than during the rest of their follow-up assessments (24.2 vs. 10.2, $p < .001$). This is not surprising as the baseline assessment was conducted while subjects were hospitalized inpatients at an acute stage, and patients in a severe state have been shown to substantially distort their ratings of their true personality (Hirschfeld et al., 1983b; Reich et al., 1987). Thus, in order to not misrepresent the true trajectory of PhA, we excluded data from the baseline assessment in the IGC models below. In order to facilitate interpretation of the intercept, we recoded the time variable so that the first follow-up was '0' for each subject.

Stability of Physical Anhedonia over time

To test the stability of PhA, we first conducted an unconditional means IGC model, which models only an intercept term and assumes no change in scores over time. From this output, we computed a reliability coefficient (intraclass correlation) of .51 which represented the proportion of between-subject variance (τ) to total variance ($\tau + \sigma^2$), i.e., whether scores within

each person are more similar than scores across people (Cohen et al., 2003). Thus, over half of the variance in PhA measured stable individual differences among patients.

Second, we computed the unconditional growth model using the following level-1 equation:

$$\text{PhA} = \pi_{0i} + \pi_{1i}(\text{time}) + e_{it}, \quad \text{equation \#1}$$

with π_{0i} = estimated PhA at first follow-up (intercept), π_{1i} = estimated rate of linear change over time (slope), and e_{it} = random within-subjects error of prediction for individual i at time t . The level-2 equations were:

$$\begin{aligned} \pi_{0i} &= b_{00} + r_{0j} \\ \pi_{1i} &= b_{10} + r_{1j} \end{aligned}$$

with b_{00} = the population average of the level-1 intercepts; b_{10} = the population average of the level-1 slopes; r_{0j} and r_{1j} = residuals that represent the portions of level-2 outcomes that remain unexplained by b_{00} and b_{10} , respectively. In IGC, “ b ’s” are unstandardized coefficients and are analogous to unstandardized b ’s in traditional OLS regression analyses.

We examined whether the unconditional growth model fit the data better than the unconditional means model by subtracting the deviance statistic (-2 log-likelihood function value at convergence) of the two models. The difference of the deviance statistics are distributed as χ^2 , with dfs equal to the difference in the two models parameters (Bryk and Raudenbush, 2002). The unconditional growth model provided a marginally better fit for the data $\chi^2(2) = 4.85, p < .10$, suggesting marginally significant linear change in PhA over time. The slope parameter was not significant ($\pi_{1i} = 0.003, SE = 0.085, p = .98$) and the addition of a quadratic term for time did not improve the model fit, $\chi^2(3) = 1.84, ns$. Additionally, inspection of the level-2 variance components indicated a significant amount of individual differences in linear slope ($p < .01$). Taken together, these results suggest that the average within-person trajectory of PhA was fairly horizontal over time, though these slopes varied to a certain degree (see Figure 1). Patients also varied in their intercept (level-2 variance component for intercept π_{0i} , $p < .001$), indicating individual differences in the ‘elevation’ of these trajectories. The mean overall level of PhA for each patient was 10.2 ($SD = 5.7$).

Conditional models examining relation between PhA and changes in depressive symptoms

We examined the relation between PhA and depression within individuals and between individuals. To test whether fluctuations in PhA significantly covaried with depressive symptoms across time within individuals, we conducted IGC models with depressive symptoms (Katz and Lyster, 1963) as a time-varying (level-1) predictor of PhA. Changes in depressive symptoms significantly predicted changes in PhA (coefficient = 0.21, $SE = .09, p < .05$). The variance component for this coefficient was nonsignificant ($p = .42$) suggesting a lack of individual differences in the relation between depressive symptoms and PhA. The results were the same when we switched the predictor and outcome variable, i.e., PhA as predictor and depressive symptoms as outcome; (coefficient = 0.12, $SE = .05, p < .05$).

We next tested the between subjects relation of these two variables by examining whether patients’ overall mean PhA across follow-ups covaried with their overall mean depressive symptoms across follow-ups. The Pearson product moment correlation between mean depression and mean PhA was significant, $r(N=49) = .34, p < .05$, indicating a moderate association.

PhA, depression, and psychosocial functioning

The next set of analyses examined whether PhA predicted several indicators of functioning - social functioning, work functioning, and re-hospitalization. These analyses were done to evaluate the clinical utility of PhA. Given the above association between PhA and depressive symptoms, we also examined whether depressive symptoms predicted functioning.

We conducted IGC models with PhA as a time-varying (level-1) predictor of either social functioning, employment functioning, or re-hospitalization (see top half of Table 2). Changes in PhA predicted changes in social functioning and rehospitalization at a trend level ($p=.06$), but not employment functioning ($p=.97$). Interestingly, changes in depressive symptoms predicted changes in all three measures of functioning. In order to assess the unique predictive power of PhA on functioning, we conducted IGC models with both PhA and depressive symptoms as time-varying (level-1) predictors (see top half Table 3). Once adjusting for depressive symptoms, PhA no longer predicted re-hospitalization, but continued to predict social functioning.

Lastly, we examined the between subjects association between PhA, depressive symptoms, and functioning. For working and re-hospitalization, we created an overall dichotomous variable for each patient indicating whether at any point during the follow-up the patient was not able to work full-time or were ever hospitalized, respectively. As shown in the bottom half of Table 2, regressions revealed that PhA was not associated with these three measures of functioning. However, mean depressive symptoms were significantly associated with mean social functioning, likelihood of re-hospitalization, and employment functioning at a trend level ($p=.07$). The effects for depressive symptoms on functioning were maintained after adjusting for PhA as well (see bottom half of Table 3).

Discussion

Despite the fact that trait anhedonia is prominent in many theoretical models of MDD (Davidson et al., 2000; Klein, 1987), few studies have examined the role of physical anhedonia (PhA) in MDD. The present study attempted to address this question in a sample of unipolar, nonpsychotic depressives three ways; by examining a) the stability of PhA over time, b) the relation between depressive symptoms and PhA, and c) whether PhA predicts aspects of functioning, thus addressing the clinical utility of PhA. The results of these three aims are separately discussed below.

Stability

Analyzing stability is an essential first step. If PhA is a personality construct, it should be relatively stable over time. In sum, we found that PhA was somewhat stable (i.e., horizontal trajectory) over the 20-year follow-up within individuals, although the slope of each individual's trajectory varied. Our findings are consistent with studies that found that general measures of anhedonia (e.g., Fawcett-Clark Pleasure Capacity Scale; FCPS; Fawcett et al., 1983) are stable over time in samples of depressives, even when depressive symptoms improve (Clark et al., 1984). Few studies, however, have examined stability in the specific domain of physical anhedonia. It is important to look at PhA separately from general anhedonia as a recent factor analytic study in participants with elevated depression showed that PhA does not load with other measures of hedonic capacity (Leventhal et al., 2005). This suggests that PhA taps a separable aspect of anhedonia.

Our results for unipolar depressives support other studies that found that PhA is stable over time in college students (Meyer and Hautzinger, 1999), schizophrenic patients (Herbener and Harrow, 2002; Loas et al., 2009), and inpatients (Berlin et al., 1998). However, unlike those

other studies, we used IGC to assess stability. IGC is superior to conventional test-retest reliability/correlations as it can address the individual trajectories across numerous time-points and can simultaneously examine individual differences in intercept and slope (Bryk and Raudenbush, 2002). It will be important for future studies to compare the stability of PhA in MDD patients to that of non-MDD patients.

Relation to depressive symptoms

We found that a) within person fluctuations in depressive symptoms related to within person fluctuations in PhA and b) individuals with higher overall PhA reported higher overall depressive symptoms across time. To our knowledge, in a depressed sample, no study has examined both these within and between person associations, though our results are consistent with the few reports that have examined these two questions separately (Loas et al., 1992; Katsanis et al., 1992). More importantly, no study has examined this association over a 20-year multiple follow-up, as reported here.

There are several interpretations and implications of these findings. First, PhA may be an indicator of vulnerability to a more severe form of depression. Higher levels of PhA have been shown to predict suicide in depressed individuals (Loas, 2007). Additionally, neuroimaging studies have found that a particularly intractable type of depression is associated with abnormalities in limbic structures associated with pleasure (Brody et al., 1999; Dougherty and Rauch, 2007). Thus, trait anhedonia (and PhA more specifically) may be a useful behavioral marker for identifying at risk cases of MDD. A second, related, interpretation is that PhA may have pathoplastic effects on the course of depression. This interpretation is supported by the finding that related personality dimensions, such as low behavioral activation, have a pathoplastic effect on depression (Kasch et al., 2002; Klein et al., 2008). In order to elucidate these interpretations, it is important that future studies ‘lag’ PhA and depressive symptoms (e.g., a month apart) and/or measure PhA before, during, and after a depressive episode (Klein et al., 2008).

A third interpretation is that these results may reflect a rating bias. Studies have shown that changes in mood state affect depressed participants’ ratings of their core personality tendencies (Ormel et al., 2004; Reich et al., 1987). This does not imply that personality ratings of depressed individuals are invalid, as the personality ratings of depressed individuals deviate from controls, before, during and after recovery from depression (Costa et al., 2005; Fanous et al., 2007; Ormel et al., 2004). Rather, it implies that our within person association may be a result of ‘true’ PhA ratings becoming exaggerated as depressive symptoms increased.

Association with functioning

We found that within person changes in PhA was significantly associated with within person changes in social functioning and re-hospitalization at a trend level. Taken together with the aforementioned finding regarding PhA and suicide (Loas, 2007), these findings argue for the clinical utility and significance of PhA. However, this conclusion is made with caution as depressive symptoms were more robust predictors of these external validators than PhA, both in the within person and between person analyses. In both the between and within person analyses, once adjusting for the presence of the other predictor, depressive symptoms maintained its association with functioning, while PhA did not. This may be due to the fact that depression severity covers more domains of mood and behavior than PhA, and is thus a more important feature of unipolar depression than PhA.

The one exception for this pattern was for the within person associations with social functioning – higher PhA predicted lower social functioning, even after adjusting for depressive symptoms. It is possible this result reflects anhedonic individuals’ interpersonal fear and ambivalence, a

phenomenon Meehl (1962; 1990) attributed to “aversive drift” in his classic discussions of anhedonia. However, this theory does not address why there was no between subjects association between PhA and social functioning.

The study had a number of strengths including multiple follow-ups over a 20-year prospective follow-up period, a well characterized sample of unipolar, non-psychotic depressives recruited from inpatient units, and the analysis of both between and within-person associations using IGC. However, the study also had several limitations. First, because a large percentage of participants were receiving varied treatment (medication, psychotherapy, or both) during the follow-ups, we were unable to examine the impact of treatment on PhA. Similarly, there are other variables that were not assessed (e.g., stressful life events) that would have likely affected the course of the variables that were examined. Second, a lack of variance in the work functioning variable forced us to dichotomize work functioning to be ‘worked fulltime’ vs. ‘didn’t work full-time,’ potentially reducing the likelihood of finding effects for this domain of functioning. Third, while our use of an inpatient recruited sample is a strength, it may also reduce the generalizability of the results to less severe samples for whom PhA may have different associations (Leventhal et al., 2006). Fourth, not all individuals completed each part of the study protocol at every follow-up and our main variable of PhA had the highest rate of missing values. However, IGC takes into the number of observations in estimating slope and intercept coefficients.

In sum, this study found that PhA plays an important role in the longitudinal course of MDD: PhA appeared somewhat stable over time, was associated with changes/differences in depressive symptoms, and was associated with important variables related to functioning (though not as robustly as depressive symptoms). Given that PhA is most often examined in schizophrenia research (Harrow and Herbener, 2002), it will be important for future studies to examine whether PhA plays a different role in depression vs. schizophrenia. Additionally, it will be important for future studies to compare the anhedonia (or hypohedonia) of physically positive stimuli to anhedonia of other positive stimuli such as money, social, and professional rewards. It is possible that these domains play different roles in the course and outcome of MDD.

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Figure 1.
The IGC estimated trajectories of physical anhedonia over 20 years.

Table 1

Baseline characteristics of sample of individuals with MDD. N=49.

% Female	59.2%
Age (SD)	23.31 (3.1)
Mean # of years of school completed (SD)	14.2 (2.3)
Socioeconomic status (SD)	2.9 (1.4)
% Caucasian	77.6%
% never married	63.3%
% with psychiatric hospitalizations prior to one at baseline	30.6%

Physical anhedonia and depressive symptoms (unadjusted for each other) predicting functioning. N=49.

Table 2

Predictor	Functioning variables (outcome)					
	Social functioning		Work functioning		Re-hospitalization	
	Coef.	SE	Coef.	SE	Coef.	SE
Within subjects (time-varying) analyses						
Physical Anhedonia	-.03 *	.01	-.0008	.02	.05 +	.02
Depressive Symptoms	-.04 **	.01	-.13 ***	.03	.17 ***	.03
Between subjects analyses						
Physical Anhedonia	Beta		OR (95% CI)		OR (95% CI)	
Depressive Symptoms	-.20		0.99 (0.90–1.09)		1.02 (0.92–1.14)	
	-.35 *		1.17 (0.99–1.34) +		1.27 (1.06–1.54) *	

Within subjects results are from IGC models where predictors are time-varying predictors. Between subjects results are from linear and logistic regressions using each participants' average ratings during follow-up. For the within subjects IGC analyses, coefficients are unstandardized *b*'s.

+ $p < .10$,
 * $p < .05$,
 ** $p < .01$,
 *** $p < .001$

Physical anhedonia and depressive symptoms (adjusted for each other) predicting functioning. N=49.^a

Table 3

Predictor	Functioning variables (outcome)					
	Social functioning			Work functioning		
	Coef.	SE		Coef.	SE	Re-hospitalization
Within subjects (time-varying) analyses						
Physical Anhedonia	-.02 [*]	.01		.01	.02	.04
Depressive Symptoms	.002	.02		-.11 ^{**}	.03	.19 ^{***}
Between subjects analyses						
Physical Anhedonia	<i>Beta</i>			<i>OR (95% CI)</i>		<i>OR (95% CI)</i>
Depressive Symptoms	-.10			0.95 (0.85–1.06)		0.97 (0.85–1.10)
	-.32 [*]			1.20 (1.00–1.34) ⁺		1.29 (1.06–1.57) [*]

Within subjects results are from IGC models where predictors are time-varying predictors. Between subjects results are from linear and logistic regressions using each participants' average ratings during follow-up. For the within subjects IGC analyses, coefficients are unstandardized *b*'s.

⁺ *p*<.10,
^{*} *p*<.05,
^{**} *p*<.01,
^{***} *p*<.001