

Appetitive motivation in depressive anhedonia: Effects of piece-rate cash rewards on cardiac and behavioral outcomes

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Abstract:

Deficits in self-regulation and motivation are central to depression. Using motivational intensity theory (Brehm & Self, 1989), the present research examined how depressive anhedonia influences effort during a piece-rate appetitive task. In piece-rate tasks, people can work at their own pace and are rewarded for each correct response, so they can gain rewards more quickly by expending more effort. A sample of community adults ($n = 78$) was evaluated for depressive anhedonia using a structured clinical interview, yielding depressive anhedonia and control groups. Participants completed a self-paced cognitive task, and each correct response yielded a cash reward (3 cents or 15 cents, manipulated within-person). Using impedance cardiography, effort-related physiological activity was assessed via the cardiac pre-ejection period (PEP). The results indicated lower reward responsiveness in the anhedonia group. Compared to the control group, the depressive anhedonia group showed significantly less baseline-to-task change in PEP, and they performed marginally worse on the task. The experiment supports the predictions made by applying motivational intensity theory to depression and offers a useful paradigm for evaluating anhedonic effects on effort while people are striving for appealing rewards.

Keywords: depression | anhedonia | effort | motivation | reward

Article:

Anhedonia—impairments in anticipating, seeking, and experiencing rewards—is central to the etiology, maintenance, and treatment of depression (Eddington, Strauman, Vieth, & Kolden, 2017). Because anhedonia is a transdiagnostic feature in several forms of psychopathology (Shankman et al., 2014), we use “depressive anhedonia” for anhedonia within the context of depression. The present research applies the principles of motivational intensity theory—a general framework for understanding the dynamics of effort engagement (Brehm & Self, 1989)—to explain how depressive anhedonia affects effort to attain appealing rewards in a sample of clinically screened community adults.

Motivational intensity theory (Brehm & Self, 1989) organizes the many variables that influence the intensity of effort into two broad factors: the importance of the goals and incentives at stake, and the difficulty of attaining them (see Richter, Gendolla, & Wright, 2016, for a review). These factors influence effort depending on the nature of the goal and structure of the task. In research that has applied the theory to understanding how depressive symptoms affect effort, we would classify most of the studies as using “all-or-none” tasks: people get the incentive only if they meet a performance standard. For example, people may receive \$3 if they get at least 20 items correct on a task within 5 min but \$0 if they get less than 20—people thus get all or none of the reward at stake. Another kind of all-or-none task uses uncertain levels of task difficulty: People do not know how hard the task will be or how well they need to do, but they do know that surpassing the unknown standard will gain the incentive (Richter & Gendolla, 2006). When difficulty is uncertain, people’s effort level reflects the importance of the incentive—people’s effort is proportional to how much they value the goal. Many studies have used uncertain-difficulty paradigms to study how depressive symptoms affect effort (e.g., Ahles, Mezulis, & Crowell, 2017; Brinkmann & Franzen, 2013, 2017; Franzen & Brinkmann, 2016a, 2016b), including one study with a clinical sample (Franzen, Brinkmann, Gendolla, & Sentissi, 2018). When the performance standard is unclear, dysphoric and depressed participants expend less effort, suggesting that the incentives are less valuable.

One of motivational intensity theory’s major task paradigms, however, remains largely untapped. Piece-rate tasks—also known as unfixed-difficulty, self-paced, and pay-for-performance tasks (Wright, Killebrew, & Pimpalpure, 2002)—allow people to work at their own pace and give a reward for each correct response. Piece-rate tasks are useful for examining how people value incentives: The intensity of effort reflects the value of the incentive at stake (Wright, 2008). Only a few studies have used this paradigm to evaluate incentives and depression (Brinkmann & Gendolla, 2007; Brinkmann, Grept, & Gendolla, 2012; Franzen & Brinkmann, 2015; Silvia, Nusbaum, Eddington, Beaty, & Kwapil, 2014). They all measured subclinical, self-reported depressive symptoms in college samples, and only one study had a tangible reward on the line (Franzen & Brinkmann, 2015)—in the rest, participants were simply instructed to “do their best.”

Studies without tangible incentives are problematic because individual differences affect how people frame the task and incentives. People with a promotion orientation, for example, place greater value on goal attainment (Brodsholl, Kober, & Higgins, 2007). Depression and dysphoria are associated with lower promotion orientation (e.g., Eddington, Silvia, Foxworth, Hoet, & Kwapil, 2015), lower intrinsic motivation toward approach goals (Winch, Moberly, & Dickson, 2015), and attenuated neural activation to promotion goals (Eddington et al., 2009). By contrast, people who approach tasks with a prevention orientation place greater value on avoiding losses or maintaining the status quo (Brodsholl et al., 2007). As a result, if incentives are not explicitly provided, a do-your-best task is not appetitive for all participants.

Motivational intensity research has emphasized cardiac autonomic outcomes, particularly markers of beta-adrenergic sympathetic nervous system (SNS) activity (Richter et al., 2016). As it acts more strongly on the heart, the SNS increases the conduction velocity of the sinoatrial and atrioventricular nodes and causes the ventricles to contract more forcefully (Drew & Sinoway, 2012). As a result, higher SNS activity causes shorter pre-ejection periods (PEP), the time from

the onset of ventricular depolarization to the aortic valve's opening. Widely used in the literature (Richter et al., 2016), PEP is the primary outcome in the present study.

The present research examined depressive anhedonia in appetitive motivation and expands on past work in several ways. First, we selected participants via interview-based assessment of clinically significant anhedonia. With one exception (Franzen et al., 2018), past research has assessed self-reported symptoms that range in severity. Second, we used a piece-rate task that isolated the effects of depressive anhedonia on appetitive effort. Unlike most past studies, we used a concrete incentive—cash for each correct response—instead of a vague do-your-best goal. The reward structure was straightforward: The task was novel but simple, so errors were rare; no money was lost for errors, so people needn't avoid losses; and all correct responses were rewarded. This paradigm provides a clear look at effort during the process of attaining rewards. Finally, we used cardiac autonomic activity to measure effort-related SNS activity. We expected, based on past work, that depressive anhedonia would predict diminished effort—indicated by weaker PEP reactivity—in response to incentives.

Method

Participants

The research was approved by the UNCG Institutional Review Board, and all participants provided informed consent. See the online supplemental materials for more demographic, recruiting, and methodological details.

Clinical screening interview. Interested people contacted the research team and took part in a brief telephone screening interview, which ruled out obviously ineligible participants. Of 156 phone screenings, 88 people took part in a face-to-face session and received \$15. This session involved a structured clinical interview, questions about medications and cardiovascular health, and assessment of height, weight, heart rate, and blood pressure. To be eligible, people had to be between 18 and 45 years old and in good cardiovascular health.

Trained clinical psychology graduate students conducted a structured clinical interview using select modules of the Structured Clinical Interview for DSM Disorders (SCID-5-RV; First, Williams, Karg, & Spitzer, 2015; SCID-II: First, Gibbon, Spitzer, Williams, & Benjamin, 1997). Participants were ineligible if they had taken antidepressants within the past eight weeks, reported any past manic or hypomanic symptoms, reported any clinically significant psychotic symptoms, reported current substance abuse or dependence, had active suicidal ideation, or met diagnostic criteria for antisocial or borderline personality disorder. Current anhedonic symptoms were assessed with Criterion 2 (markedly diminished interest or pleasure, most of the day nearly every day for at least two weeks), which was coded according to SCID conventions (*absent/false, subthreshold, or present/true*).

Final sample. Seventy-eight people who were eligible ultimately took part in the laboratory part of the study. Of the 78, anhedonic symptoms were ruled “absent” in 56 (71.8%), “subclinical” in 4 (5.1%), and “present at clinical levels” in 18 (23.1%). For analysis, the subclinical and clinical levels were combined, yielding a depressive anhedonia condition ($n = 22$; 28.2%) and a control

condition ($n = 56$; 71.8%). Of the 22 people in the anhedonia condition, 17 (77.3%) had concurrent major depressive disorder. The final sample was predominantly young ($M = 23.26$ years, $SE = .61$), female ($n = 59$, 75.6%), and diverse (15.4% Hispanic/Latinx, 35.9% European American, 47.4% African American).

Procedure

For the day of the laboratory session, participants were instructed to abstain from exercise, physical exertion, caffeine, and nicotine for the time of waking until after the session. They received \$20 plus whatever they earned on the effort tasks. The sessions were conducted by a same-gender experimenter who was unaware of the participants' diagnostic status, and all information collected during the screening interview. The participants expected to complete computer-based cognitive tasks while cardiovascular activity was measured. After the electrodes were placed, participants sat quietly for three minutes for the signals to stabilize. Baseline physiological readings were then taken while participants completed a range of self-report and demographic items, which holds constant irrelevant factors (e.g., sitting upright, reading from a monitor, and using a keyboard) that would otherwise vary between baseline and task periods (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). The surveys took around 10 min to complete; readings from Minutes 2 through 8 were used to compute the baseline physiological values.

Behavioral task and manipulation of incentive. After the baseline period, participants completed a digit parity task (Framorando & Gendolla, 2018; Harper, Eddington, & Silvia, 2016; Silvia, Sizemore, Tipping, Perry, & King, 2018). People see a word in the center of the screen flanked by two numbers (e.g., 7 BOAT 9, 8 BENCH 5). They must ignore the word and decide if the numbers have the same parity (i.e., they are both odd or both even) or different parity (i.e., one is odd, one is even). Participants responded by pressing one of two buttons on a high-speed keyboard. The parity task is self-paced—the trials remain on screen until people respond, which starts another trial—and thus unfixed in difficulty (Wright, 2008).

We manipulated incentives by rewarding each correct response with cash. We offered two incentive levels (cf. Harper, Silvia, Eddington, Sperry, & Kwapil, 2018). Participants completed two blocks of the parity task. In one block, they received 3 cents for each correct trial; in the other, they received 15 cents per correct trial. Each block was 3 min long, so people could do as many trials as they wished within that time. Incentive value (3 cents vs. 15 cents) was manipulated within-person, so all participants completed both blocks. To avoid uncertainty, both the experimenter and the software informed the participants which incentive they would get prior to each block of the task. There was no penalty for errors, and no trial-by-trial feedback was given. The experimenter emphasized that the goal was to get as many correct as possible and that each correct response was rewarded, so the main outcome from the parity task is the number of correct responses. Participants were paid what they earned in cash after the session, for an average of roughly \$15. There was a 90-s break between the task blocks.

The parity task had two counterbalancing factors structured via randomized blocks. First, participants began with either the 3-cents block or the 15-cents block. Counterbalancing incentive value prevents confounding incentive level with other factors (e.g., practice or fatigue).

Second, two sets of parity items (i.e., different nouns and digits) were created to avoid item-familiarity effects across blocks. The item sets were manipulated orthogonally to incentive value.

Physiological assessment. An electrocardiogram (ECG) signal was acquired with 3 spot electrodes in a modified Lead II configuration (the lowest left and right ribs and the right collarbone). An impedance cardiogram (ICG) signal was acquired with four spot electrodes in a tetrapolar configuration: two receiving electrodes on the chest (one on the left collarbone, lateral to the suprasternal notch, and another on the sternum at the xiphoid process), and two sending electrodes on the back (4 cm above and below the receiving electrodes). Using a Mindware Bionex chassis, the signals were sampled at 1000 Hz and filtered offline (60 Hz notch; ECG: .5 to 45 Hz; Z0: 10 Hz cutoff; dZ/dt: .5 to 50 Hz).

The physiological periods of interest were the baseline (7 min), the 3-cent task period (3 min), and the 15-cent task period (3 min). These were divided into 60-s epochs, and the ECG and ICG points were identified on ensemble-averaged waveforms. PEP is the time between the ECG Q-point (reflecting the start of ventricular depolarization; Berntson, Lozano, Chen, & Cacioppo, 2004) and the dZ/dt B-point (reflecting the opening of the aortic valve; Lozano et al., 2007). Q and B were identified with automated methods via IMP 3.1.1 software. Q was identified as the lowest point in the 35-ms window prior to R (for issues in Q identification, see Berntson et al., 2004; Seery, Kondrak, Streamer, Saltsman, & Lamarche, 2016); B was estimated using Lozano et al.'s (2007) slope/intercept method. All points were visually inspected and corrected manually if necessary. Finally, heart rate was assessed in beats per minute. We had no predictions for HR, which is affected by the sympathetic and parasympathetic branches (Drew & Sinoway, 2012) and thus inconsistently associated with mental effort (Richter, 2012).¹

Results

Table 1 shows descriptive statistics for PEP and HR. The seven baseline minutes, three 3-cent task minutes, and three 15-cent task minutes were averaged to create overall baseline, 3-cent, and 15-cent values. Of the 78 people who took part, 7 participants were omitted from the analysis: One person appeared confused and had an unusually long session time, 3 people's physiological data were too noisy or erratic (primarily in the dz/dt wave), and 3 people consumed significant nicotine immediately before the session. This left a final sample of 71 people (55 healthy control, 16 anhedonic).

Table 1. Raw Descriptive Statistics for Cardiac Outcomes

Outcome	Control			Depressive anhedonia		
	Baseline	3 cents	15 cents	Baseline	3 cents	15 cents
PEP						
<i>M</i>	110.49	108.35	107.96	114.14	112.67	113.30
<i>SE</i>	1.83	2.01	1.98	2.89	2.67	2.93
HR						
<i>M</i>	80.85	81.91	82.71	78.06	78.67	78.89
<i>SE</i>	1.25	1.38	1.42	2.42	2.66	2.57

Note. Total $n = 71$ (55 healthy control, 16 anhedonic). PEP = pre-ejection period (in ms); HR = heart rate (in bpm).

¹ See the online supplemental materials for information on the RZ interval, a similar cardiac outcome (Silvia, McHone, et al., 2018).

We analyzed the data in Mplus 8.1 using multilevel models, which accommodate the nested observations due to several task periods (see the online supplemental materials for details). The within-level's predictor was *period*, with three levels: baseline, 3 cents, 15 cents. Linear (-1, 0, 1) and quadratic (1, -2, 1) effects were estimated to evaluate the nature of within-person change across the baseline/3 cents/15 cents periods. The between-level's predictor was *depressive anhedonia* (controls vs. anhedonia), which was grand-mean centered. The model thus estimates the within-person main effect of period (e.g., how PEP varies across the baseline, 3-cent, and 15-cent periods), the between-person main effect of depressive anhedonia, and—critical for our hypotheses—the interaction of depressive anhedonia and period (e.g., whether the change in PEP across the periods differed for the control and anhedonia groups). Based on nonsignificant findings from preliminary models, the counterbalancing factors were omitted. The models were estimated with maximum likelihood with robust standard errors. All regression effects are unstandardized; effect sizes are expressed as R^2 (%).

Cardiac Outcomes

PEP. For PEP, there were significant linear ($b = -1.08$, $SE = .23$, $p < .001$) and quadratic ($b = .31$, $SE = .11$, $p = .007$) within-person main effects of period ($R^2 = 13.4\%$). The trend reflects an overall decline in PEP from the baseline to the 3 cents to the 15 cents condition, with a flattening between the 3 cents and 15 cents condition (see Table 1). Because lower PEP values reflect greater sympathetic influence on the heart, the within-person main effects of period indicate that, for the sample overall, the incentives in the parity task motivated increased effort (Richter, 2012).

Furthermore, depressive anhedonia moderated the effect of period on PEP. There was no between-person main effect of anhedonia on PEP ($b = 4.44$, $SE = 3.32$, $p = .181$, $R^2 = .9\%$), but anhedonia significantly predicted the linear ($b = .85$, $SE = .42$, $p = .045$, $R^2 = 3.3\%$) but not the quadratic ($b = .06$, $SE = .25$, $p = .812$, $R^2 = .1\%$) change in PEP.

The interaction was consistent with diminished reward responsiveness. The effect of period on PEP was “flatter” for people with depressive anhedonia than for healthy controls, reflecting less change in PEP from the baseline to the 3 and 15 cent conditions. Figure 1 depicts the difference in estimated linear slopes. The Y-axis is the estimated linear effect of period on PEP; the boxes depict the slopes in the control and anhedonia groups. The slopes for the control participants are more negative, reflecting greater PEP change as the incentives increased, whereas the slopes for the anhedonic participants are closer to zero, reflecting weaker change in PEP in response to incentives.

HR. For HR, there was a significant linear ($b = .81$, $SE = .28$, $p = .004$) but not quadratic ($b = -.05$, $SE = .10$, $p = .623$) within-person main effect of time period ($R^2 = 3.6\%$). Overall, heart rate increased across the time periods as incentive value increased. Depressive anhedonia did not have a main effect on HR ($b = -3.28$, $SE = 2.85$, $p = .249$, $R^2 = .9\%$) or interactions with the linear ($b = -.52$, $SE = .52$, $p = .323$, $R^2 = .5\%$) or quadratic ($b = -.02$, $SE = .22$, $p = .935$, $R^2 = .1\%$) effects of time period (see Table 1). The effects of depressive anhedonia were thus specific to PEP, the outcome that more specifically reflects beta-adrenergic sympathetic activity.

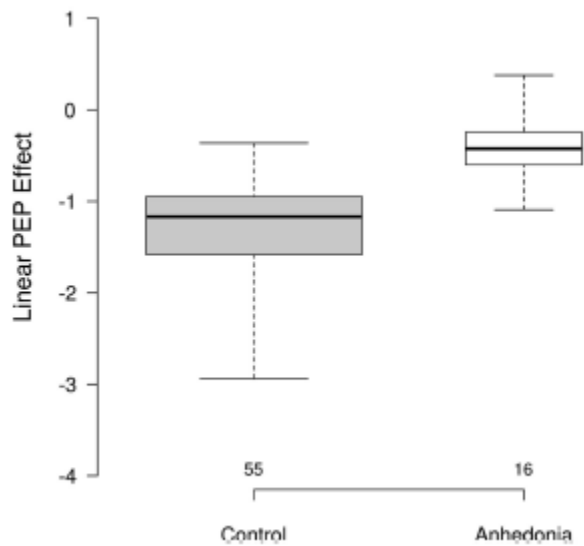


Figure 1. The linear effect of time period (baseline → 3 cents → 15 cents) on PEP for control (n = 55) and anhedonic (n = 16) participants. The Y-axis is the estimated linear effect of time period on PEP—the boxes depict the slopes in the control and anhedonia groups. The boxplot whiskers indicate the minimum and maximum values in each group.

Behavioral Performance

For behavioral performance, we analyzed the effects of depressive anhedonia on the number of correct responses during the 3-cent and 15-cent task periods (see Table 2). A multilevel model found no effect of period (coding 3 cents as -1 and 15 cents as 1), $b = .23$, $SE = .57$, $p = .684$, $R^2 = .5\%$, so people got roughly the same amount correct in each block regardless of the incentive. Depressive anhedonia had a marginal main effect on correct responses ($b = -5.35$, $SE = 3.18$, $p = .092$, $R^2 = 1.6\%$), and there was no interaction ($b = 2.12$, $SE = 1.60$, $p = .185$, $R^2 = 3.1\%$). Consistent with the cardiac effort deficits, depressive anhedonia was associated with a trend toward fewer correct responses and hence less money.²

Table 2. Number of Correct Responses on the Parity Task

Statistic	Control		Depressive anhedonia	
	3 cents	15 cents	3 cents	15 cents
<i>M</i>	83.65	83.16	76.19	79.94
<i>SE</i>	1.88	1.87	2.60	3.58

Note. Total $n = 71$ (55 healthy control, 16 anhedonic).

Discussion

In the present research, we applied the general principles of motivational intensity theory to explain how people self-regulate effort in response to a piece-rate task with a sample of adults

² A wrinkle of self-paced tasks is that completing more trials involves more motor activity, so movement could confound physiological outcomes. The number of correct responses, however, was uncorrelated with the respective baseline-to-period PEP change for either the 3-cent ($r = -.05$, $p = .623$) or 15-cent periods ($r = .00$, $p = .991$), so movement confounding is highly unlikely.

who were clinically assessed for depressive anhedonia. Thus far, research grounded in motivational intensity theory has almost exclusively used self-report measures of anhedonic and depressive symptoms within generally high-functioning samples (e.g., university students). In addition, most past work on effort and incentive value has used tasks either with no obvious incentive (i.e., do-your-best instructions; Brinkmann & Gendolla, 2007; Silvia et al., 2014) or with uncertain difficulty levels (e.g., Franzen & Brinkmann, 2016a, 2016b). These studies suggest that anhedonia reduces the perceived value of incentives, but it is difficult to disentangle incentive value from other depressive effects when no rewards are at stake or when people do not know what the task will be like. When incentives and task features are ambiguous, lower incentive value can be conflated with other correlates of depression that influence motivation, such as pessimistic beliefs, low self-efficacy, prevention focus, and risk-aversion (Douglas, Porter, Frampton, Gallagher, & Young, 2009; Eddington & Foxworth, 2012; Strauman & Eddington, 2017).

The present research thus evaluated anhedonia and effort in a paradigm designed to target reward responsiveness. The task involved making a novel but simple judgment, and people could work as quickly or slowly as they wished within the task period. All correct responses were rewarded, errors were not punished, and the reward was tangible and notably large (people averaged around \$15). As expected, anhedonic participants' effort was less affected by the incentives. PEP declined from the baseline to the incentive periods for the sample as a whole, reflecting effortful response to attain the incentives. But the decline was steeper for the control participants and flatter for the anhedonic participants, consistent with an effect of depressive anhedonia on diminished incentive value.

Behavioral performance roughly paralleled the cardiac outcomes. Anhedonic participants got fewer responses correct and hence earned less money, but the effect was marginal. In motivational intensity research, physiological and behavioral measures sometimes converge but commonly diverge. In addition to effort, task performance is affected by ability and task strategies to different degrees, so greater effort will not always translate into better performance. This common finding illustrates why biological measures are valuable complements to behavioral approaches.

Regarding limitations, blood pressure was not assessed. PEP is influenced both by SNS-mediated contractility and by ventricular preload and afterload (Obrist, Light, James, & Strogatz, 1987). Preload differences can be ruled out because of nonsignificant HR differences, but conclusively ruling out afterload would require assessing diastolic blood pressure. It is rare to find afterload-biased PEP changes with participants who are still, seated, and working on mental tasks, however, because such effects are much more common for tasks involving alpha-adrenergic influence on the peripheral vasculature (e.g., enduring cold temperatures; Obrist et al., 1987). In addition, the sample sizes of the depressive anhedonia and control groups were unequal, largely because of the complexity of recruiting young, depressed adults who were otherwise healthy and not taking antidepressants, which could potentially affect estimates of variance and effect sizes. Finally, for future work, it would be worth manipulating the complexity of the self-paced task. The parity task is novel but people make almost no mistakes. Other studies, in contrast, have used memory tasks that yield much higher error rates (Franzen & Brinkmann, 2015). The conceptual replication across these studies is encouraging, and it would be interesting to evaluate

if depressive anhedonia causes shifts in strategies as the task becomes more complex (e.g., shifting to a cautious, “preventing mistakes” strategy; Eddington & Foxworth, 2012).

Effects of depression on effort and motivation are often described as “abnormal” or “dysfunctional.” An underappreciated implication of using motivational intensity theory as a framework, however, is that depression’s effects on effort are largely rational—the level of effort might be lower, but the process of effort regulation is not dysfunctional or dysregulated. Depression appears to shift effort by shifting beliefs about the value of incentives and the difficulty of achieving them (Brinkmann & Franzen, 2015; Silvia et al., 2016). Effort follows predictably from these input values, not from a breakdown of an ordinarily rational motivation system. Depressive anhedonia might cause lower effort in some cases, but the dynamics of effort per se are not apparently dysfunctional.

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