

## Do depressive symptoms "blunt" effort? an analysis of cardiac engagement and withdrawal for an increasingly difficult task

By: [Paul J. Silvia](#), Zuzana Mironovová, Ashley N. McHone, Sarah H. Sperry, Kelly L. Harper, [Thomas R. Kwapil](#), and [Kari M. Eddington](#)

Silvia, P.J., Mironovova, Z., McHone, A., Sperry, S., Harper, K.L., Kwapil, T.R., & Eddington, K.M. (2016). Do depressive symptoms "blunt" effort? an analysis of cardiac engagement and withdrawal for an increasingly difficult task. *Biological Psychology*, 118, 52-60. PubMed Central PMCID: PMC4956535.

Made available courtesy of Elsevier: <https://doi.org/10.1016/j.biopsycho.2016.04.068>



This work is licensed under a [Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License](#).

\*\*\*© 2016 Elsevier B.V. Reprinted with permission. This version of the document is not the version of record. \*\*\*

### Abstract:

Research on depression and effort has suggested “depressive blunting”—lower cardiovascular reactivity in response to challenges and stressors. Many studies, however, find null effects or higher reactivity. The present research draws upon motivational intensity theory, a broad model of effort that predicts cases in which depressive symptoms should increase or decrease effort. Because depressive symptoms can influence task-difficulty appraisals—people see tasks as subjectively harder—people high in depressive symptoms should engage higher effort at objectively easier levels of difficulty but also quit sooner. A sample of adults completed a mental effort challenge with four levels of difficulty, from very easy to difficult-but-feasible. Depressive symptoms were assessed with the CESD and DASS; effort-related cardiac activity was assessed via markers of contractility (e.g., the cardiac pre-ejection period [PEP]) obtained with impedance cardiography. The findings supported the theory’s predictions. When the task was relatively easier, people high in depressive symptoms showed higher contractility (shorter PEP), consistent with greater effort. When the task was relatively harder, people high in depressive symptoms showed diminished contractility, consistent with quitting. The results suggest that past research has been observing a small part of a larger trajectory of trying and quitting, and they illustrate the value of a theoretically grounded analysis of depressive symptoms and effort-related cardiac activity.

**Keywords:** Effort | Motivation | Depression | Anhedonia | Self-regulation | Impedance cardiography

### Article:

#### 1. Introduction

Depression is an intriguing problem for theories of motivation because of its many motivational impairments (Brinkmann & Franzen, 2015). Depression affects how people select and pursue goals (Eddington, Silvia, Foxworth, Hoet, & Kwapil, 2015), disengage from unattainable goals (Klinger, 1975), respond to incentives (Treadway & Zald, 2011), and appraise their self-efficacy for challenges (Kanfer & Zeiss, 1983). Recently, research on the psychophysiology of effort—how people expend and withdraw energy to achieve their goals (Gendolla, Wright, & Richter, 2012)—has examined how depressive symptoms influence effort-related cardiac sympathetic activity. Many of these studies have suggested a *depressive blunting*: people higher in depressive symptoms show reduced cardiac reactivity when faced with incentives, challenges, and stressors (e.g., Phillips, 2011, Schwerdtfeger and Rosenkaimer, 2011).

In this article, we revisit an analysis of depression and effort rooted in motivational intensity theory (Brehm & Self, 1989), a general model of effort that has been successfully translated to a wide range of problems (Richter, 2013, Wright, 2008). Motivational intensity theory outlines several conditions in which depressive symptoms should reduce effort (Brinkmann & Franzen, 2015; Gendolla, Brinkmann, & Silvestrini, 2012). But the theory also outlines conditions in which depressive symptoms should increase effort. These more controversial predictions have received some support in early research (Brinkmann & Gendolla, 2008) but have been overshadowed by the broader interest in contexts that reduce effort. The present study expands upon earlier work and tests these predictions about heightened effort.

## **2. Exaggerated or blunted reactivity in depression?**

Depression's influence on cardiovascular reactivity in response to challenges, incentives, and stressors is typically studied using a two-period design: a baseline period followed by a lab task, such as an incentive task, a stressful performance challenge, or an unpleasant experience to be endured. Several studies have found that depressive symptoms—assessed with subclinical self-report scales or with clinical interviews—predicted less cardiovascular reactivity to public performance challenges (e.g., speech and singing tasks: Salomon, Bylsma, White, Panaite, & Rottenberg, 2013; Salomon, Clift, Karlsdóttir, & Rottenberg, 2009; Schwerdtfeger & Gerteis, 2013; Schwerdtfeger & Rosenkaimer, 2011). Several other studies have found no effect (e.g., for mirror-tracing or cold pressor tasks: Salomon et al., 2009, Salomon et al., 2013). And many studies—meta-analyzed by Kibler and Ma (2004)—have found exaggerated reactivity, both for cardiovascular (e.g., Light, Kothandapani, & Allen, 1998; Matthews, Nelesen, & Dimsdale, 2005) and related outcomes (e.g., plasma norepinephrine; Light et al., 1998).

Such variability suggests that moderating factors are at work. One likely factor, suggested by Schwerdtfeger and Rosenkaimer (2011), is whether the tasks primarily call upon alpha- or beta-adrenergic sympathetic processes. Consistent with Obrist's (1981) coping model, tasks that call for active coping (striving to achieve an outcome) vs passive coping (passively enduring a negative event) yield different physiological profiles because they recruit different sympathetic pathways. Studies of passive stressors tend to find null effects or exaggerated effects for alpha-adrenergic markers (e.g., vascular resistance), whereas studies of active coping challenges tend to find effects for beta-adrenergic markers (e.g., higher systolic blood pressure and shortened cardiac pre-ejection period).

In addition, a broader reason why these effects are inconsistent stems from the lack of a guiding theory of why people should engage or withdraw effort when faced with challenges and stressors. For the most part, the literature is exploratory, and the experimental designs are set up to evaluate only linear main effects of depressive symptoms on reactivity. The typical design—a baseline period followed by a challenging task—lacks more than one level of difficulty. Non-linear effects on depressive symptoms thus cannot be observed in any single study, but they can emerge as inconsistent effects in the literature as a whole.

### **3. Applying a model of effort**

Motivational intensity theory (Brehm & Self, 1989) offers a general framework for explaining when people expend or withhold effort when pursuing goals. It has proven fruitful in a wide range of contexts, such as how effort is affected by personality traits (Capa, 2012; Silvia, Jones, Kelly, & Zibaie, 2011b), mood states (Gendolla et al., 2012a), ability beliefs (Wright, 1998), incentives (Richter, 2012), self-relevance (Gendolla & Richter, 2010), task difficulty (Brehm & Self, 1989), uncertainty (Richter & Gendolla, 2006), and prior exertion and fatigue (Wright & Stewart, 2012), among many others. The theory emphasizes two broad and interacting factors: the value of the goal at stake and the appraised difficulty of achieving it. These two pathways—an incentive value path and a task difficulty path—can interact, and the circumstances in which one, the other, or both are involved have been extensively studied (Richter, Gendolla, & Wright, 2016).

#### **3.1. Evidence for the incentive pathway**

According to motivational intensity theory, effort is sometimes solely a function of incentive value: the importance or value of the goal at stake. In some tasks—known as self-paced, piece-rate, or unfixed tasks (Wright, 2008)—the amount of reward is proportional to a person's effort, such as a cognitive task that pays 5 cents for each correct response and allows people to work as quickly as they wish (e.g., Silvia, McHone, Mironovová, Harper, & Sperry, 2016). For such tasks, the intensity of effort is a function of the value of the incentives (Wright, Killebrew, & Pimpalpure, 2002). As a result, individual differences in incentive value will directly affect effort. Depressive symptoms, particularly anhedonic symptoms, should reduce the value of rewards and incentives (Treadway & Zald, 2011), so the theory predicts that depressive symptoms will reduce effort when tasks are unfixed. Several studies of effort during unfixed tasks have supported this prediction (Franzen & Brinkmann, 2015; Silvia, Nusbaum, Eddington, Beaty, & Kwapil, 2014): depressive symptoms predicted lower effort, assessed as baseline-to-task reactivity in the cardiac pre-ejection period (PEP), a measure of beta-adrenergic sympathetic impact on the heart (Kelsey, 2012).

Tasks with uncertain difficulty are another case in which effort is a function of incentive value (Richter and Gendolla, 2006, Richter and Gendolla, 2007). If people don't know how hard a task will be, they adopt a "prepare for the worst" strategy: they gear up effort as a function of how valuable they find the rewards and incentives at stake. This ensures people are prepared for a wide range of difficulty levels when the task starts, and effort can eventually be withheld and calibrated to the task's demands if needed. Depressive symptoms, by reducing the value of incentives, should thus decrease effort when people are uncertain about a task's difficulty, a

prediction supported by many studies (Brinkmann & Franzen, 2013; Brinkmann, Franzen, Rossier, & Gendolla, 2014; Brinkmann, Schüpbach, Joye, & Gendolla, 2009).

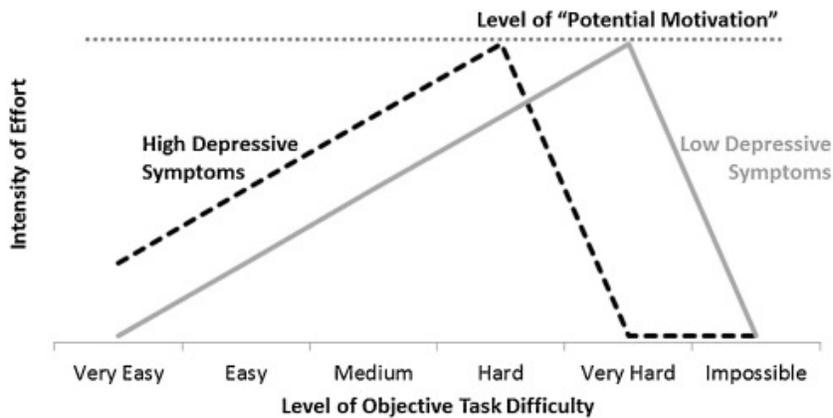
### 3.2. Evidence for the difficulty appraisal pathway

For tasks where difficulty is unfixed or uncertain, then, motivational intensity theory predicts that depressive symptoms will reduce effort via the incentive value pathway: by making the incentives and rewards seem less appealing, depressive symptoms promote withholding effort. For the second pathway—appraisals of a task’s difficulty—the predictions are more subtle, and they include cases in which depressive symptoms should boost effort.

When people work on a task with a known level of difficulty, motivational intensity theory predicts that effort is a function of how hard people find the task (Brehm & Self, 1989). Incentive value sets a ceiling on how much effort people are willing to expend, but difficulty affects how much effort people actually expend (Wright, 1998, Wright, 2008). If a task is easy, effort should be low regardless of how appealing people find the incentives—there’s little point in needlessly mobilizing the body for a task that does not require it (Richter, 2013). As difficulty increases, effort increases to provide the resources needed to accomplish the goal. But when difficulty is very high, people reach a point at which effort declines: either the goal is not worth the effort (i.e., effort hits the ceiling set by incentive value), or the goal seems impossible to achieve. In either case, people withdraw effort, thus conserving energy and resources for other goals.

Any factor that affects the perceived difficulty of a task will thus affect effort. Depressive symptoms, for many reasons, could affect perceived task difficulty. Research shows that negative moods (Gendolla, 2000), fatigue (Wright & Stewart, 2012), and low ability beliefs (Wright, 1998) make people appraise tasks as more difficult. Such experiences and beliefs are typical of subclinical dysphoria and clinical depression (see Brinkmann & Franzen, 2015). As a result, people with elevated depressive symptoms would be expected to exert more effort at objectively easier levels of task difficulty because they experience the task as harder. Likewise, they would be expected to quit at objectively earlier levels of difficulty because they reach the subjective “too hard” limit sooner.

These predictions are depicted in Fig. 1. The dashed horizontal line is the ceiling of “potential motivation” established by the value of the goal (Wright, 2008). The intensity of actual effort is represented by the two non-linear functions. People high in depressive symptoms show higher effort when the task is easier because they see it as relatively more challenging than people low in depressive symptoms do. In turn, they quit at an earlier level of task difficulty: because they see the task as harder, they more quickly reach the point at which the goal is no longer worth the effort. People low in depressive symptoms, in contrast, show less effort when the task is easier but continue engaging effort up to higher levels of difficulty (see Gendolla & Krüsken, 2002, for analogous predictions for the effects of positive and negative mood states on effort).



**Figure 1.** Expected effects of depressive symptoms and objective task difficulty on the intensity of effort.

Unlike its predictions for tasks with uncertain or unfixed difficulty, reviewed earlier, motivational intensity theory's predictions for the effects of depressive symptoms on variable-difficulty tasks have been tested in only a couple of experiments. In two studies, Brinkmann and Gendolla (2008) selected adults who were high or low in depressive symptoms, who then worked on a cognitive task that was either easy or hard. In both experiments, people high in depressive symptoms expended significantly more effort, reflected in systolic blood pressure (SBP) reactivity, when the task was easy. In contrast, people low in depressive symptoms expended more effort when the task was hard. The findings broadly supported the theory's predictions, but the evidence for one crucial prediction—that people high in depressive symptoms will quit at easier levels of task difficulty—was equivocal. In both experiments, people low in depressive symptoms showed a strong increase in SBP reactivity from the easy to the hard condition, as the theory would expect. People high in depressive symptoms, however, showed similar SBP reactivity in the easy and hard conditions, so the evidence for quitting was ambiguous.

#### 4. The present research

The present research tested motivational intensity theory's predictions concerning depressive symptoms and effort, and it expanded upon past research in several key respects. First, the experiment manipulated a much wider range of task difficulty. Motivational intensity theory predicts a non-linear trajectory of trying and quitting, not simple exaggerated or blunted reactivity. It is possible that past experiments did not include a broad enough range of difficulty to illustrate the theory's predictions: people high in depressive symptoms should show both an earlier onset of effort-related cardiac activity as well as an earlier withdrawal of effort (see Fig. 1). The present experiment thus included four levels of task difficulty, from very easy for everyone to challenging-but-feasible for most people.

Second, effort-related cardiac activity was assessed using impedance cardiography. SBP reactivity has a long history in effort research and effectively captures beta-adrenergic sympathetic influence on cardiac inotropy (Wright, 1996). Nevertheless, much recent work in effort has examined measures of contractility derived from impedance cardiography, primarily the pre-ejection period (PEP; Kelsey, 2012), which may offer a more precise measure of

sympathetic activity in effort paradigms (Richter, 2012, Richter and Gendolla, 2009). And third, parasympathetic activity, quantified via high-frequency heart rate variability (HRV), was assessed to explore potential effects associated with the parasympathetic branch. Motivational intensity theory emphasizes sympathetic activity (Wright, 1996), but HRV is worth exploring in light of its relationships with self-regulation (Segerstrom, Hardy, Evans, & Winters, 2012) and depression (Kemp et al., 2010, Rottenberg, 2007).

## 5. Method

### 5.1. Participants

A total of 71 adults—59 women, 12 men—enrolled at the University of North Carolina at Greensboro (UNCG) took part and received credit toward a research option in a psychology class. The mean age was 19.12 years ( $SD = 1.32$ ,  $Min/Max = 18, 25$ ) and racially and ethnically diverse. Based on self-reports, the sample was approximately 49% African American, 42% European American, and 9% Hispanic or Latino; people could pick more than one category or decline to pick any. The average body mass index (BMI), based on self-reported height and weight, was 25.26 ( $SD = 5.98$ ), so the sample as a whole was on the boundary of normal weight and overweight. Participants were asked ahead of time to avoid caffeine, nicotine, or exercise prior to the experiment. The 71 participants were the final sample from a larger group that agreed to participate. Participants were excluded because the session was interrupted by fire alarms (1 person) or because they misunderstood the effort task (1 person), smoked immediately before the session (1 person), or indicated having taken medications that day that affect sympathetic or parasympathetic influences on the heart (10 people, primarily for medications related to seasonal allergies, asthma, anxiety, or ADHD).

Using randomized blocks, each person was randomly assigned to one of four between-group levels of task difficulty: *easy*, *medium*, *hard*, and *very hard*.

### 5.2. Procedure

This research was approved and monitored by the UNCG IRB, and all participants provided informed consent. Each person took part individually in sessions run by a gender-matched experimenter.

The experimenter explained that the task was about the psychology of mental effort and how the body responded to mental challenges. The participants expected to complete some cognitive tasks along with measures of personality and individual differences while connected to physiological recording equipment. The experimenter placed the electrodes for impedance cardiography, and after the signals stabilized, participants began a baseline period in which they completed innocuous demographic and self-report questionnaires throughout the baseline period. The last 5 min were used to quantify baseline physiological values. This is a kind of “vanilla baseline” (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992) that seeks to hold constant irrelevant factors (e.g., sitting upright, reading from a screen, and responding with a mouse and keyboard) across the baseline and task periods, as opposed to an active relaxation or basal level

baseline period (Obrist, 1981). Participants were seated upright in a non-reclining comfortable chair throughout the experiment.

### 5.2.1. The d2 task

After the baseline period, participants worked on the d2 task (Brickenkamp & Zillmer, 1998), a popular task in mental effort research (e.g., Brinkmann and Gendolla, 2007, Gendolla and Krüsken, 2001, Silvia, 2012; Silvia, Jones, Kelly, & Zibaie, 2011a, 2011b). In this task, people see a lowercase *d* or *p*. The letter has 0, 1, or 2 apostrophes above and below it. People had to press a yellow key if the item was a d2 (a *d* with exactly 2 apostrophes: 2 above, 2 below, or 1 above and below) or a blue key if the item was a *p* or any other *d*.

Task difficulty was manipulated by varying the amount of time people had to respond to an item. In the *easy* condition, all the items appeared for 2000 ms; in the *medium* condition, 67% of the items appears for 2000 ms and 33% appeared for 750 ms; in the *hard* condition, 33% of the items appeared for 2000 ms and 67% appeared for 750 ms; and in the *very hard* condition, all the items appeared for 750 ms. These response windows were based on our extensive past research with this task with similar samples (e.g., Silvia, Kelly, Zibaie, Nardello, & Moore, 2013; Silvia, McCord, & Gendolla, 2010; Silvia, Moore, & Nardello, 2014). In all cases, the item remained on screen for the full response window. This prevents people from working at their own pace and thus fixes the level of task difficulty (Wright et al., 2002). The items were presented randomly, and the task was controlled by Direct RT (Empirisoft, NY).

The goal of the task was a 90% accuracy standard. Participants were instructed: “The goal is to be *accurate*, not to be as fast as possible. The standard for this task is to get 90% of the items right; stated the other way, try not to make mistakes on more than 10% of the items.” We took care to avoid making the task an “uncertain difficulty” task (Richter, 2010, Richter and Knappe, 2014), in which people are wholly unaware of the level of challenge they will face (e.g., how hard the items will be, how long the task will take, and what the performance standard it). To ensure that people understood the task and its level of difficulty, people first completed 20 practice trials that were consistent with the participant’s condition. In addition, before starting, the software reminded participants that all the items would be the same speed (*easy* and *very hard* conditions) or that most of the items would be slow (*medium* condition) or fast (*hard* condition), that the task would be basically like the practice session, and that the performance standard was to get 90% of the items correct. The participants then worked on the task for 4 min.

The d2 task yields two behavioral outcomes. Percentage correct is the primary outcome because the task’s performance goal is to get 90% right. Response times for correct responses are a secondary outcome.

After completing the task, people completed several self-report items and manipulation checks that assessed subjective task performance (“In your opinion, how well did you do on the d2 task?” [1 = *very poorly*, 7 = *very well*]), effort (“How much effort did you spend on the d2 task?” [1 = *very little effort*, 7 = *intense effort*]), importance (“How important was it to you to get 90%

right?” [1 = *not at all important*, 7 = *very important*]), and task difficulty (“In your opinion, as a whole, how hard was the d2 task?” [1 = *very easy*, 7 = *very hard*]).

### 5.2.2. Depressive symptoms

Depressive symptoms were measured with the 20-item Center for Epidemiological Studies—Depression Scale (CESD; Radloff, 1977) and the 7-item Anhedonic Depression subscale of the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995). Both scales are popular in the broader depression literature and in past research on effort (Brinkmann & Gendolla, 2008; Silvia, Nusbaum et al., 2014c). Both scales asked people to rate how much a series of statements described or applied to their experiences during the past week. Both scales used 4-point response formats (CESD: 0 = *Rarely or none of the time (less than 1 day)*, 3 = *Most or all of the time (5–7 days)*; DASS: 0 = *Did not apply to me at all*, 3 = *Applied to me very much*). The CESD covers a wide range of depressive symptoms (e.g., “My sleep was restless”), whereas the DASS emphasizes anhedonic symptoms (e.g., “I felt that I had nothing to look forward to”).

It seemed unlikely that completing the CESD and DASS before the d2 task would affect effort, but the possibility was worth preventing. Completing the Beck Depression Inventory can influence mood (e.g., Mark, Sinclair, & Wellens, 1991), an important input to task appraisals (Gendolla, 2000), and asking participants questions about a task’s difficulty or importance can influence task appraisals (Richter, 2010; Richter & Knappe, 2014). Both scales were thus completed after the d2 task.

### 5.2.3. Physiological assessment

Cardiac autonomic activity was assessed using impedance cardiography. An electrocardiogram (ECG) signal was acquired using 3 electrodes placed in a modified Lead II configuration (the right collarbone and lowest left and right ribs). An impedance cardiogram (ICG) was acquired using 4 spot electrodes in a standard tetrapolar configuration. The two receiving electrodes were placed on the chest (an upper electrode on the left collarbone at the level of the suprasternal notch, and a lower electrode at the xiphoid process). The upper and lower sending electrodes were placed on the back and displaced 4 cm above and below the receiving electrodes, respectively. All signals were acquired using a Mindware Bionex chassis using a sampling rate of 1000 Hz. The signals were filtered offline (ECG: 0.5–45 Hz;  $dZ/dt$ : 0.5–50 Hz;  $Z_0$ : 10 Hz cutoff; 60 Hz notch filter). Respiration rate (cycles per minute) was derived from the  $Z_0$  thoracic impedance signal (Ernst, Litvack, Lozano, Cacioppo, & Berntson, 1999).

The 5 baseline minutes and 4 d2 task minutes were divided into 60-s periods. Our primary outcomes were markers of beta-adrenergic sympathetic influence on the heart. The cardiac pre-ejection period (PEP)—the time (in ms) between the ECG Q point (the onset of ventricular depolarization; Berntson et al., 2004) and the  $dZ/dt$  B point (the opening of the aortic valve and start of left ventricular ejection; Lozano et al., 2007)—is widely used in effort research (Richter, 2012). As a secondary sympathetic outcome, we also computed the RZ interval: the time (in ms) between the ECG R point (the point of peak electrical activity) and the  $dZ/dt$  Z point (the  $dZ/dt$  peak and point of maximal diameter of the aortic arch; van Eijnatten, van Rijssel, Peters, Verdaasdonk, & Meijer, 2014). Also known as the initial systolic time interval (ISTI; Meijer,

Boesveldt, Elbertse, & Berendse, 2008), RZ is increasingly popular as a measure of contractility. Several effort studies have found that it works at least as well as PEP (Silvia, Beaty, Nusbaum, Eddington, & Kwapil, 2014; Silvia, Nusbaum et al., 2014c; Silvia et al., 2016), possibly because it uses the salient R and Z points instead of the subtle Q and B points (van Lien, Schutte, Meijer, & de Geus, 2013). PEP and RZ were assessed by ensemble averaging (Kelsey et al., 1998) the ECG and  $dZ/dt$  waveforms within each 60 s period. The Q point was identified as the lowest point in the 35 ms window prior to R (Berntson et al., 2004), and B was estimated via the Lozano et al. (2007) slope/intercept method (i.e.,  $RB = RZ \times 0.55 + 4$ ). These points were identified automatically by the software (IMP 3.1.1) and corrected in only a small number of cases.

Heart rate variability (HRV) was quantified as respiratory sinus arrhythmia (RSA). Spectral methods computed high-frequency heart rate variability in the 0.15–0.40 Hz frequency range (Berntson et al., 1997). The interbeat interval (IBI, in ms) and respiration rate (in cycles per minute) were included as additional autonomic outcomes.

## 6. Results

### 6.1. Data reduction

The CESD ( $\alpha = 0.86$ ) and DASS Depression scales ( $\alpha = 0.86$ ) both yielded reliable scores. We observed a wide range of scores for each scale. CESD sum scores ranged from 0 to 50 ( $M = 12.77$ ,  $SD = 8.64$ ); DASS Depression mean scores ranged from 0 to 2.43 ( $M = 0.41$ ,  $SD = 0.52$ ). The scores thus range from the floor of the scale to values typical of adults diagnosed with major depressive disorder (e.g., Antony, Beiling, Cox, Enns, & Swinson, 1998; Santor, Zuroff, Ramsay, Cervantes, & Palacios, 1995). Neither CESD nor DASS Depression scores varied significantly between the 4 conditions, according to one-way analyses of variance (both  $F_s < 1$ ). The CESD and DASS Depression total scores correlated very highly with each other ( $r = 0.84$ , 95% CI: 0.77, 0.93), as in our past research ( $r = 0.81$ ; Silvia, Nusbaum et al., 2014c, p. 783), so the CESD and DASS scores were standardized and then averaged to create an overall depressive symptoms score. (Consistent with their very high correlation, the scales had essentially identical relationships with effort when analyzed alone.)

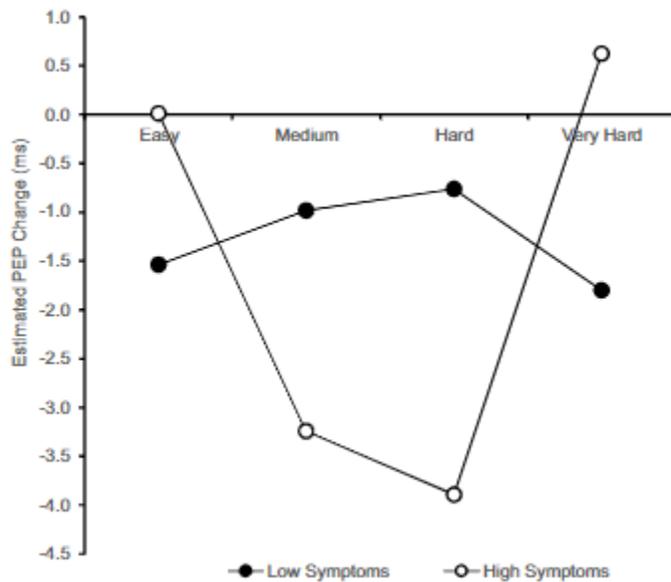
Table 1 shows the descriptive statistics for all outcomes. The data were analyzed using polynomial regression models. Delta scores—change in a physiological outcome from baseline to task—were specified as the outcome. The predictors were (1) contrast terms for the linear (–3, –1, 1, 3) and quadratic (1, –1, –1, 1) main effects of task difficulty; (2) a main effect of depressive symptoms, standardized and thus centered at zero; (3) two interaction terms reflecting the interaction between depressive symptoms and the linear and quadratic components, respectively; (4) the baseline physiological value to control for initial values; and (5) an intercept. All regression coefficients are unstandardized. Note that depressive symptoms were analyzed as a continuous variable rather than discrete groups. The figures depict estimated values for high and low depressive symptoms groups for the sake of clarity.

Performance on the d2 task was quantified as the percentage of correct responses (to adjust for the different number of trials in each difficulty condition) and as response times (in ms) for correct responses.

**Table 1.** Baseline and d2 task physiological values.

Outcome	Easy		Medium		Hard		Very Hard	
	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>
PEP Baseline	126	2	131	1	135	2	130	2
PEP Task	125	1	130	2	132	3	129	2
RZ Baseline	166	3	176	2	178	4	174	3
RZ Task	163	3	173	3	173	5	171	4
RSA Baseline	6.61	0.20	6.04	0.25	6.37	0.32	5.75	0.30
RSA Task	6.68	0.21	6.30	0.23	6.16	0.32	5.75	0.29
IBI Baseline	798	21	773	22	794	39	749	30
IBI Task	802	20	783	19	784	38	744	26
Respiration Baseline	18.01	0.50	18.30	0.79	18.05	0.53	18.32	0.62
Respiration Task	18.72	0.54	19.01	0.74	20.30	0.90	19.52	0.72

*Note:* PEP (pre-ejection period), RZ (R to Z interval), and IBI (interbeat interval) values have been rounded to the nearest millisecond. Respiration is in cycles per minute; RSA (respiratory sinus arrhythmia) values are in  $ms^2$ . *SE* = standard error. Condition *ns* = 20, 19, 15, and 17, respectively.



**Figure 2.** Estimated change in PEP (in ms) as a function of task difficulty condition and depressive symptoms.

## 6.2. PEP and RZ

For PEP, the regression model found a significant interaction between depressive symptoms and quadratic change ( $b = 0.95$ ,  $SE = 0.42$ ,  $p = 0.025$ ). No other main effects or interactions in the model were significant, all  $t_s < 1$ . Fig. 2 depicts the pattern of estimated values based on the upper and lower 20% points of depressive symptoms. Note that PEP values become more negative—the PEP interval is shorter in the task than in the baseline—as sympathetic activity increases. The pattern supports the predictions and findings from Brinkmann and Gendolla (2008). For people higher in depressive symptoms, PEP reactivity was near-zero in the easy condition, higher when the task was medium and hard, and near-zero again when the task was very hard. This pattern is consistent with engaging effort at easier levels of task difficulty and

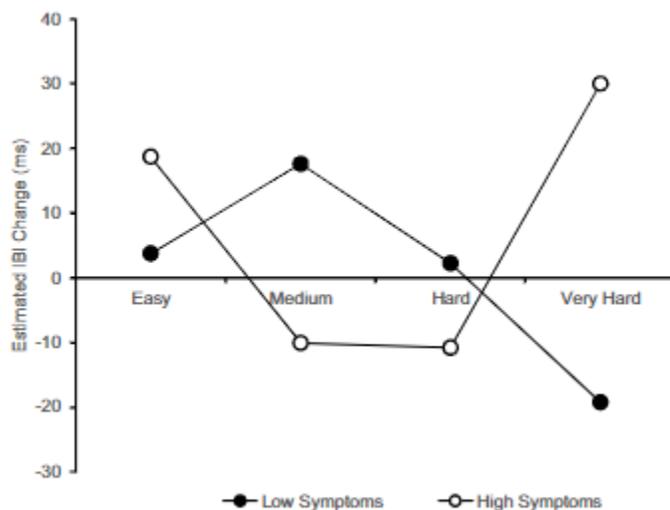
then withdrawing effort when the task is perceived as too difficult. For people lower in depressive symptoms, however, engagement was moderate and consistent across the difficulty conditions, and the largest PEP change was at the highest level of task difficulty. At the highest level of task difficulty, people high in depressive symptoms had withdrawn effort, but people low in depressive symptoms showed PEP reactivity consistent with continued engagement.

An essentially similar pattern appeared for RZ. There was a significant interaction between depressive symptoms and quadratic change ( $b = 1.89, SE = 0.72, p = 0.011$ ). No other main effects or interactions in the model were significant, all  $t_s < 1$ . The pattern, shown in Table 1, essentially paralleled the pattern for PEP. As an aside, the model  $R^2$  was slightly higher for RZ (11.9%) than for PEP (10.6%), which replicates the larger effect sizes found for RZ found in recent experiments using incentives (Silvia et al., 2016) and other effort paradigms (Silvia, Beaty et al., 2014a; Silvia, Nusbaum et al., 2014c).

### 6.3. HRV, respiration, and IBI

For RSA reactivity, a regression model (with change in respiration rate included as a covariate) found no significant main effects or interactions for task difficulty or depressive symptoms. Similarly, a model for change in respiration rate found no significant main effects or interactions involving task difficulty or depressive symptoms.

For IBI, the analysis found a significant linear contrast ( $b = -3.54, SE = 2.62, p = 0.032$ ) and a significant interaction between depressive symptoms and quadratic change ( $b = 7.73, SE = 3.62, p = 0.037$ ). Fig. 3 depicts the pattern of results, which roughly parallels the pattern of effects for PEP and RZ.



**Figure 3.** Estimated change in IBI (in ms) as a function of task difficulty condition and depressive symptoms.

### 6.4. Task performance and subjective self-Reports

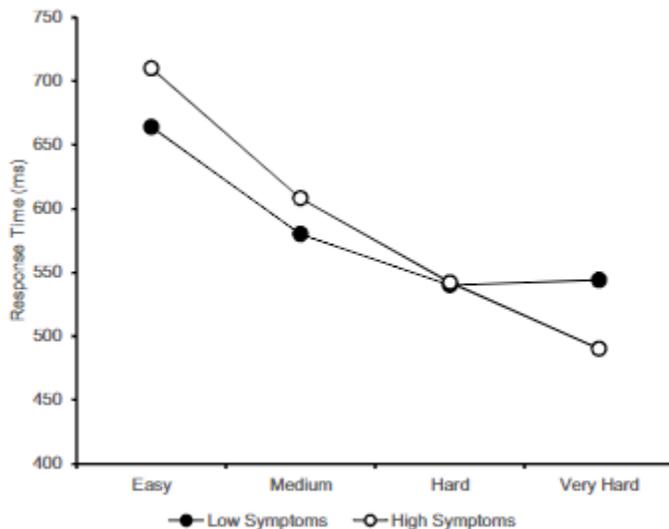
Additional regression models explored whether task difficulty and depressive symptoms influenced task performance (see Table 2). For our primary measure, percent correct (arcsine transformed), there was a significant linear contrast ( $b = -0.05$ ,  $SE = 0.01$ ,  $p < 0.001$ ), reflecting a decrease in the percent correct in the harder conditions. Notably, there was also a significant main effect of depressive symptoms ( $b = -0.05$ ,  $SE = 0.02$ ,  $p = 0.030$ ): as depressive symptoms increased, people got a smaller percent correct.

**Table 2.** Subjective reports and task performance.

Outcome	Easy		Medium		Hard		Very Hard	
	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>
Percent Correct	0.97	0.01	0.94	0.01	0.90	0.02	0.88	0.02
Response Time	683	22	578	10	557	9	522	11
Difficulty	1.45	0.17	2.53	0.31	3.07	0.25	3.71	0.32
Effort	3.20	0.48	5.26	0.37	4.67	0.42	4.94	0.30
Importance	5.55	0.32	6.05	0.24	6.00	0.31	5.94	0.23
Performance	6.10	0.27	5.16	0.26	4.73	0.28	4.47	0.33

Note: *SE* = standard error. Response times are rounded to the nearest millisecond. The self-reported items were assessed on 1–7 scales, with higher numbers reflecting higher self-reported task difficulty, effort, importance of doing well, and quality of task performance. Condition *ns* = 20, 19, 15, and 17, respectively.

For response times (RT) for correct responses, the regression model revealed significant linear ( $b = -25.73$ ,  $SE = 3.39$ ,  $p < 0.001$ ) and quadratic ( $b = 19.60$ ,  $SE = 7.60$ ,  $p = 0.012$ ) trends, reflecting faster RTs as the response window shortened. In addition, a marginal interaction between depressive symptoms and the linear contrast appeared ( $b = -6.05$ ,  $SE = 3.36$ ,  $p = 0.077$ ). The pattern, shown in Fig. 4, suggests that people higher in depressive symptoms tended to be relatively slower when the d2 task was easy and relatively faster when it was very hard.



**Figure 4.** Effects of task difficulty condition and depressive symptoms on response times (in ms) to the d2 task.

Responses to the self-report questions (see Table 2) showed that the manipulation of task difficulty worked as intended. A regression model for self-reported task difficulty (“In your opinion, as a whole, how hard was the d2 task?”) revealed only a significant linear contrast

( $b = 0.37$ ,  $SE = 0.06$ ,  $p < 0.001$ ): self-reported task difficulty increased linearly across the 4 conditions. Similarly, for self-reported task performance (“In your opinion, how well did you do on the d2 task?”), only the linear contrast was significant ( $b = -0.28$ ,  $SE = 0.06$ ,  $p < 0.001$ ): people reported performing worse as task difficulty increased.

For self-reported effort (“How much effort did you spend on the d2 task?”), only the linear ( $b = 0.23$ ,  $SE = 0.10$ ,  $p = 0.018$ ) and quadratic ( $b = -0.48$ ,  $SE = 0.21$ ,  $p = 0.030$ ) contrasts were significant, reflecting an increase in self-reported effort from the easy condition to the other three (see Table 2). Finally, no significant effects appeared for self-reported goal importance (“How important was it to you to get 90% right?”).

## 7. Discussion

To date, research on depression and effort has emphasized contexts in which depression should impair effort. As a result, several researchers have suggested a “blunted effort” interpretation (Phillips, 2011, Salomon et al., 2009, Salomon et al., 2013, Schwerdtfeger and Rosenkaimer, 2011). Using a general theory of effort, however, allows us to understand when depressive symptoms will reduce or increase effort. The present experiment examined one of the contexts in which depressive symptoms should increase effort, according to a motivational intensity theory analysis (Brehm & Self, 1989). As Brinkmann and Gendolla (2008) proposed, people higher in depressive symptoms should experience tasks as more demanding for many reasons, such as low self-efficacy and negative mood states. As a result of appraising tasks as more difficult, they should engage higher effort at lower levels of objective difficulty but also quit earlier. These predictions have been tested in only a couple of experiments (Brinkmann & Gendolla, 2008), which broadly supported the analysis but found equivocal evidence for earlier quitting.

The results strongly supported the model’s predictions (Brinkmann & Gendolla, 2008). People higher in depressive symptoms showed high effort engagement, reflected in PEP and RZ reactivity, at easier levels of task difficulty. At the most difficult level, however, they disengaged effort. People lower in depressive symptoms, however, showed more moderate engagement at lower difficulty levels—consistent with easy tasks requiring little effort—but showed their highest engagement at the most difficult level. At the highest level of difficulty, people high in depressive symptoms had disengaged, showing essentially no reactivity, whereas people low in depressive symptoms had continued to engage. The full trajectory of trying and quitting was thus observed.

The one condition that was least consistent with the predicted pattern was when people low in depressive symptoms worked on the easiest form of the task. In this case, sympathetic reactivity was greater than would be predicted. Nevertheless, the findings as whole—and particularly the trajectory of trying and quitting found when depressive symptoms were high—support motivational intensity theory’s analysis, and future work should examine the effects of depressive symptoms for easy tasks to see if this particular finding replicates.

The physiological effects were limited to PEP and RZ, two related sympathetic metrics of contractility. This pattern is consistent with the broader motivational intensity literature, which has emphasized sympathetic markers during appetitive, approach-oriented tasks (Gendolla et al.,

2012b). A large literature has developed around depression and HRV, including baseline levels, reactivity differences, and treatment relevance (Kemp et al., 2010, Rottenberg, 2007). Other research suggests that HRV itself reflects self-regulation and self-control (Segerstrom et al., 2012). No significant effects appeared for HRV in the present research, which used an appetitive, approach-oriented task. Although research testing the theory has primarily studied sympathetic effects, future research on motivational intensity theory should continue to measure both sympathetic and parasympathetic influences, if only to identify cases in which both branches are influential (e.g., Silvia et al., 2013a).

An interesting side finding concerned depressive symptoms and task performance. People higher in depressive symptoms got a smaller percent correct. The pattern of response times, shown in Fig. 4, suggests that they adopted a speed-over-accuracy strategy as the task became harder. This approach is not optimal for the d2 task, which has an accuracy standard (get at least 90% correct) that is more likely to be met by slowing down and using the full response window. This effect is consistent with the broader literature that shows how dysphoria and depression affect people's task strategies and reactions to making mistakes (e.g., Douglas, Porter, Frampton, Gallagher, & Young, 2009; Eddington & Foxworth, 2012). Thus, in the face of a more demanding task, participants higher in depression seemed to engage in less effective responding.

### 7.1. Revisiting “blunted effort”

By now, it should become clear that examining the effects of depressive symptoms requires manipulating factors like incentive value and task difficulty. Virtually all studies in this literature, however, use only a single level of challenge. Because task difficulty is held constant, an interaction between depressive symptoms and task difficulty can't be observed. If task difficulty is held constant at an objectively high level, an experiment will find “blunted effort” if the difficulty level is in the region where dysphoric participants have quit but non-dysphoric participants have not. As the present experiment and past studies show (Brinkmann & Gendolla, 2008), this design will obscure dysphoric participants' higher reactivity at easier levels as well as quitting by all participants when the task becomes impossible. In short, studies that find “blunted effort” are observing only one part of a much larger trajectory of trying and quitting.

Finally, it is worth reconsidering the use of terms like “blunted effort” in this literature, even in cases where reduced effort is predicted, instead of simply “less effort.” Motivational intensity theory is rooted in a rational resource conservation approach to effort (Richter, 2013). People expend or withhold effort based on their subjective analysis of the value of the incentives in light of the nature of the task and how difficult it seems. When incentives seem less appealing and goals seem harder to achieve, people should show the patterns of engagement and withdrawal that people high in depressive symptoms show. These subjective appraisals may stem from dysfunctional beliefs and processes that impair people's daily functioning, but people higher in depressive symptoms are nevertheless allocating effort in ways that are subjectively rational and systematic. The motivational architecture underlying effort thus remains flexible and functional. The inputs might be biased by dysfunctional processes associated with appraising goals, incentives, and tasks, but the effort system is nevertheless responding as expected to its subjective inputs.

## Acknowledgements

This research was supported by the National Institute of Mental Health of the National Institutes of Health under award number R15MH079374. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

## References

- Antony, M. M., Beiling, P. J., Cox, B. J., Enns, M. W., & Swinson, R. P. (1998). Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety Stress Scales (DASS) in clinical groups and a community sample. *Psychological Assessment, 10*, 176–181.
- Berntson, G. G., Bigger, T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., et al. (1997). Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology, 34*, 623–648.
- Berntson, G. G., Lozano, D. L., Chen, Y., & Cacioppo, J. T. (2004). Where to Q in PEP. *Psychophysiology, 41*, 333–337.
- Brehm, J. W., & Self, E. A. (1989). The intensity of motivation. *Annual Review of Psychology, 40*, 109–131. <http://dx.doi.org/10.1146/annurev.ps.40.020189.000545>
- Brickenkamp, R., & Zillmer, E. (1998). *d2 test of attention*. Seattle, WA: Hogrefe & Huber.
- Brinkmann, K., & Franzen, J. (2013). Not everyone's heart contracts to reward: insensitivity to varying levels of reward in dysphoria. *Biological Psychology, 94*, 263–271.
- Brinkmann, K., & Franzen, J. (2015). Depression and self-regulation: a motivational analysis and insights from effort-related cardiovascular activity. In G. H. E. Gendolla, M. Tops, & S. L. Koole (Eds.), *Handbook of biobehavioral approaches to self-regulation* (pp. 333–347). New York: Springer.
- Brinkmann, K., & Gendolla, G. H. E. (2007). Dysphoria and mobilization of mental effort: effects on cardiovascular reactivity. *Motivation and Emotion, 31*, 71–82.
- Brinkmann, K., & Gendolla, G. H. E. (2008). Does depression interfere with effort mobilization? Effects of dysphoria and task difficulty on cardiovascular response. *Journal of Personality and Social Psychology, 94*, 146–157.
- Brinkmann, K., Schüpbach, L., Joye, I. A., & Gendolla, G. H. E. (2009). Anhedonia and effort mobilization in dysphoria: reduced cardiovascular response to reward and punishment. *International Journal of Psychophysiology, 74*, 250–258.
- Brinkmann, Franzen, J., Rossier, C., & Gendolla, G. H. (2014). I do not care about others' approval: dysphoric individuals show reduced effort mobilization for obtaining a social reward. *Motivation and Emotion, 38*, 790–801.

Capa, R. L. (2012). Clarifying achievement motives and effort: studies of cardiovascular response. In R. A. Wright, & G. H. E. Gendolla (Eds.), *How motivation affects cardiovascular response: mechanisms and applications* (pp. 383–398). Washington, DC: American Psychological Association.

Douglas, K. M., Porter, R. J., Frampton, C. M., Gallagher, P., & Young, A. H. (2009). Abnormal response to failure in unmedicated major depression. *Journal of Affective Disorders*, 119, 92–99.

Eddington, K. M., & Foxworth, T. E. (2012). Dysphoria and self-focused attention: effects of feedback on task strategy and goal adjustment. *Journal of Social and Clinical Psychology*, 31, 933–951.

Eddington, K. M., Silvia, P. J., Foxworth, T. E., Hoet, A., & Kwapil, T. R. (2015). Motivational deficits differentially predict improvement in a randomized trial of Self-System Therapy for depression. *Journal of Consulting and Clinical Psychology*, 83, 602–616.

Ernst, J. M., Litvack, D. A., Lozano, D. L., Cacioppo, J. T., & Berntson, G. G. (1999). Impedance pneumography: noise as signal in impedance cardiography. *Psychophysiology*, 36, 333–338.

Franzen, J., & Brinkmann, K. (2015). Blunted cardiovascular reactivity in dysphoria during reward and punishment anticipation. *International Journal of Psychophysiology*, 95, 270–277.

Gendolla, G. H. E., & Krüsken, J. (2001). The joint impact of mood and task difficulty on cardiovascular and electrodermal reactivity in active coping. *Psychophysiology*, 38, 548–556.

Gendolla, G. H. E., & Krüsken, J. (2002). Mood, task demand, and effort-related cardiovascular response. *Cognition and Emotion*, 16, 577–603.

Gendolla, G. H. E., & Richter, M. (2010). Effort mobilization when the self is involved: some lessons from the cardiovascular system. *Review of General Psychology*, 14, 212–226.

Gendolla, G. E., Brinkmann, K., & Silvestrini, N. (2012). Gloomy and lazy? On the impact of mood and depressive symptoms on effort-related cardiovascular response. In R. A. Wright, & G. H. E. Gendolla (Eds.), *How motivation affects cardiovascular response: mechanisms and applications* (pp. 139–155). Washington, DC: American Psychological Association.

Gendolla, G. H. E., Wright, R. A., & Richter, M. (2012). Effort intensity: some insights from the cardiovascular system. In R. M. Ryan (Ed.), *The Oxford handbook of human motivation* (pp. 420–438). New York: Oxford University Press.

Gendolla, G. H. E. (2000). On the impact of mood on behavior: an integrative theory and a review. *Review of General Psychology*, 4, 378–408.

- Jennings, J. R., Kamarck, T., Stewart, C., Eddy, M., & Johnson, P. (1992). Alternate cardiovascular baseline assessment techniques: vanilla or resting baseline. *Psychophysiology*, 29, 742–750.
- Kanfer, R., & Zeiss, A. M. (1983). Depression, interpersonal standard setting: and judgments of self-efficacy. *Journal of Abnormal Psychology*, 92, 319–329.
- Kelsey, R. M., Reiff, S., Wiens, S., Schneider, T. R., Mezzacappa, E. S., & Guethlein, W. (1998). The ensemble-averaged impedance cardiogram: an evaluation of scoring methods and interrater reliability. *Psychophysiology*, 35, 337–340.
- Kelsey, R. M. (2012). Beta-adrenergic cardiovascular reactivity and adaptation to stress: the cardiac pre-ejection period as an index of effort. In R. A. Wright, & G. H. E. Gendolla (Eds.), *How motivation affects cardiovascular response: mechanisms and applications* (pp. 43–60). Washington, DC: American Psychological Association.
- Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. *Biological Psychiatry*, 67, 1067–1074.
- Kibler, J. L., & Ma, M. (2004). Depressive symptoms and cardiovascular reactivity to laboratory behavioral stress. *International Journal of Behavioral Medicine*, 11, 81–87.
- Klinger, E. (1975). Consequences of commitment to and disengagement from incentives. *Psychological Review*, 82, 1–25.
- Light, K. C., Kothandapani, R. V., & Allen, M. T. (1998). Enhanced cardiovascular and catecholamine responses in women with depressive symptoms. *International Journal of Psychophysiology*, 28, 157–166.
- Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: comparison of the depression anxiety stress scales (DASS) with the beck depression and anxiety inventories. *Behaviour Research and Therapy*, 33, 335–343.
- Lozano, D. L., Norman, G., Knox, D., Wood, B. L., Miller, B. D., Emery, C. F., et al. (2007). Where to B in dZ/dt. *Psychophysiology*, 44, 113–119.
- Mark, M. M., Sinclair, R. C., & Wellens, T. R. (1991). The effect of completing the Beck Depression Inventory on self-reported mood state: contrast and assimilation. *Personality and Social Psychology Bulletin*, 17, 457–465.
- Matthews, S. C., Nelesen, R. A., & Dimsdale, J. E. (2005). Depressive symptoms are associated with increased systemic vascular resistance to stress. *Psychosomatic Medicine*, 67, 509–513.

- Meijer, J. H., Boesveldt, S., Elbertse, E., & Berendse, H. W. (2008). Method to measure autonomic control of cardiac function using time interval parameters from impedance cardiography. *Physiological Measurement*, 29, 383–391.
- Obrist, P. A. (1981). *Cardiovascular psychophysiology: a perspective*. New York: Plenum.
- Phillips, A. C. (2011). Blunted cardiovascular reactivity relates to depression, obesity, and self-reported health. *Biological Psychology*, 86, 106–113.
- Radloff, L. S. (1977). The CES-D Scale: a self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1, 385–401.
- Richter, M., & Gendolla, G. H. E. (2006). Incentive effects on cardiovascular reactivity in active coping with unclear task difficulty. *International Journal of Psychophysiology*, 61, 216–225.
- Richter, M., & Gendolla, G. H. E. (2007). Incentive value, unclear task difficulty, and cardiovascular reactivity in active coping. *International Journal of Psychophysiology*, 63, 294–301.
- Richter, M., & Gendolla, G. H. E. (2009). The heart contracts to reward: monetary incentives and pre-ejection period. *Psychophysiology*, 46, 451–457.
- Richter, M., & Knappe, K. (2014). Mood impact on effort-related cardiovascular reactivity depends on task context: evidence from a task with an unfixed performance standard. *International Journal of Psychophysiology*, 93, 227–234.
- Richter, M., Gendolla, G. H. E., & Wright, R. A. (2016). Three decades of research on motivational intensity theory: what we have learned about effort and what we still do not know. *Advances in Motivation Science*, 3, 149–186.
- Richter, M. (2010). Pay attention to your manipulation checks! Reward impact on cardiac reactivity is moderated by task context. *Biological Psychology*, 84, 279–289.
- Richter, M. (2012). Cardiovascular response to reward. In R. A. Wright, & G. H. E. Gendolla (Eds.), *How motivation affects cardiovascular response: mechanisms and applications* (pp. 79–91). Washington, DC: American Psychological Association.
- Richter, M. (2013). A closer look into the multi-layer structure of motivational intensity theory. *Social and Personality Psychology Compass*, 7, 1–12.
- Rottenberg, J. (2007). Cardiac vagal control in depression: a critical analysis. *Biological Psychology*, 74, 200–211.
- Salomon, K., Clift, A., Karlsdóttir, M., & Rottenberg, J. (2009). Major depressive disorder is associated with attenuated cardiovascular reactivity and impaired recovery among those free of cardiovascular disease. *Health Psychology*, 28, 157–165.

Salomon, K., Bylsma, L. M., White, K. E., Panaite, V., & Rottenberg, J. (2013). Is blunted cardiovascular reactivity in depression mood-state dependent? A comparison of major depressive disorder, remitted depression, and healthy controls. *International Journal of Psychophysiology*, 90, 50–57.

Santor, D. A., Zuroff, D. C., Ramsay, J. O., Cervantes, P., & Palacios, J. (1995). Examining scale discriminability in the BDI and CES-D as a function of depressive severity. *Psychological Assessment*, 7, 131–139.

Schwerdtfeger, A. R., & Gerteis, A. K. S. (2013). Is the blunted blood pressure reactivity in dysphoric individuals related to attenuated behavioral approach. *International Journal of Psychophysiology*, 90, 58–65.

Schwerdtfeger, A., & Rosenkaimer, A. K. (2011). Depressive symptoms and attenuated physiological reactivity to laboratory stressors. *Biological Psychology*, 87, 430–438.

Segerstrom, S. C., Hardy, J. K., Evans, D. R., & Winters, N. F. (2012). Pause and plan: self-regulation and the heart. In R. A. Wright, & G. H. E. Gendolla (Eds.), *How motivation affects cardiovascular response: mechanisms and applications* (pp. 181–198). Washington, DC: American Psychological Association.

Silvia, P. J., McCord, D. M., & Gendolla, G. H. E. (2010). Self-focused attention, performance expectancies, and the intensity of effort: do people try harder for harder goals? *Motivation and Emotion*, 34, 363–370.

Silvia, P. J., Jones, H. C., Kelly, C. S., & Zibaie, A. (2011a). Masked first name priming increases effort-related cardiovascular reactivity. *International Journal of Psychophysiology*, 80, 210–216.

Silvia, P. J., Jones, H. C., Kelly, C. S., & Zibaie, A. (2011b). Trait self-focused attention, task difficulty: and effort-related cardiovascular reactivity. *International Journal of Psychophysiology*, 79, 335–340.

Silvia, P. J., Eddington, K. M., Beaty, R. E., Nusbaum, E. C., & Kwapil, T. R. (2013). Gritty people try harder: grit and effort-related cardiac autonomic activity during an active coping challenge. *International Journal of Psychophysiology*, 88, 200–205.

Silvia, P. J., Kelly, C. S., Zibaie, A., Nardello, J. L., & Moore, L. C. (2013). Trait self-focused attention increases sensitivity to nonconscious primes: evidence from effort-related cardiovascular reactivity. *International Journal of Psychophysiology*, 88, 143–148.

Silvia, P. J., Beaty, R. E., Nusbaum, E. C., Eddington, K. M., & Kwapil, T. R. (2014). Creative motivation: creative achievement predicts cardiac autonomic markers of effort during divergent thinking. *Biological Psychology*, 102, 30–37.

Silvia, P. J., Moore, L. C., & Nardello, J. L. (2014). Trying and quitting: how self-focused attention influences effort during difficult and impossible tasks. *Self and Identity*, 13, 231–242.

Silvia, P. J., Nusbaum, E. C., Eddington, K. M., Beaty, R. E., & Kwapil, T. R. (2014). Effort deficits and depression: the influence of anhedonic depressive symptoms on cardiac autonomic activity during a mental challenge. *Motivation and Emotion*, 38, 779–789.