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Replication Study

Is narcissism associated with baseline cortisol in men and women?

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ABSTRACT

Narcissism is a personality trait characterized by feelings of grandiosity, a craving for admiration, and a lack of empathy. Although there are reasons to expect that narcissism might have adverse physiological and health implications, very little research has directly assessed such claims. Moreover, prior research specifically assessing links between narcissism and basal levels of the stress hormone cortisol yields mixed evidence. In an attempt to reconcile previously inconsistent findings, we examined narcissism and basal cortisol in a sample of 366 young adults. We found little evidence for an association between narcissism and cortisol. We discuss how narcissism may be related to physiological outcomes over time and across situations.

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1. Introduction and rationale

Narcissism is a personality trait characterized by feelings of grandiosity, a craving for admiration, and a lack of empathy (Emmons, 1987; Miller & Campbell, 2008; Raskin & Hall, 1979). Narcissistic individuals may exaggerate their positive personality traits, attractiveness, and intelligence (Paulhus & John, 1998; Rhodewalt & Eddings, 2002). Despite their grandiose self-views, however, narcissists' self-esteem is thought to be fragile, leading them to experience a sense of inferiority and worthlessness, especially in the face of self-esteem threats (Horvath & Morf, 2009). To manage feelings of inferiority, narcissists tend to use defensive strategies, such as choosing partners who are especially likely to admire them and to boost their ego (Campbell, 1999). Additionally, narcissists tend to react with displaced aggression when confronted with feedback that does not align with their overly positive self-perceptions (Martinez, Zeichner, Reidy, & Miller, 2008). Although these types of defensive strategies may be protective in the short-term, they can also be costly in the long-term.

For instance, chronic reliance on defensive strategies can lead to dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, the body's main stress response system (Rutledge, 2006). During acute stressors or challenges, particularly those that are socially evaluative in nature, the HPA axis releases the stress hormone cortisol, which facilitates action in response to stress or threat (Dickerson & Kemeny, 2004). Although short-term HPA responses

are adaptive, chronic HPA dysregulation has been associated with adverse health outcomes, including poor cardiovascular health and suppressed immune functioning (Johnson, Kamilaris, Chrousos, & Gold, 1992; Kelsey, Ornduff, Reiff, & Arthur, 2002; Rutledge, 2006). Thus, overreliance on defensive strategies have been hypothesized to negatively affect narcissistic individuals' HPA regulation and ultimately lead to health problems (Edelstein, Yim, & Quas, 2010; Reinhard, Konrath, Lopez, & Cameron, 2012). Yet, this hypothesis has rarely been directly examined and, more generally, very little is known about the physiological and health implications of narcissism.

Basal cortisol is moderately stable over time (Liening, Stanton, Saini, & Schultheiss, 2010), and has been linked with physical health (Johnson et al., 1992), providing a rationale for testing its association with personality traits such as narcissism. However, the two studies that have examined links between basal cortisol and narcissism specifically have yielded mixed results. In one study, 90 undergraduates either delivered an unexpected speech in front of an audience (to elicit social-evaluative threat) or completed a set of questionnaires (as a control task) (Edelstein, Yim et al., 2010). Cortisol was measured at baseline (20 min after participants arrived at the laboratory to allow for an adaptation period) and at 7 additional times during and after the speech or control task. Baseline measures of cortisol were not significantly correlated with narcissism as measured by the Narcissistic Personality Inventory (NPI) in the total sample ($r = -0.05$, $p = 0.66$, 95% CI [-0.25, 0.16]) or when broken down by gender ($r_{men} = -0.02$, $p = 0.92$, 95% CI [-0.32, 0.28], $n = 44$; $r_{women} = -0.06$, $p = 0.69$, 95% CI [-0.35, 0.24], $n = 46$). However, narcissistic men showed greater cortisol reactivity and negative affect following the laboratory

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stressor. These findings suggest that narcissistic individuals may not necessarily have higher *baseline* levels of cortisol, but that narcissistic men were more reactive to or distressed by the public speaking task, both psychologically and physiologically.

In a subsequent study, Reinhard et al. (2012) examined the association between narcissism (measured with the NPI) and cortisol in a sample of 106 undergraduate students (79 women). Cortisol was measured at two time points: once at baseline (immediately after participants' arrival to the lab) and another following a 25-min period of filler tasks. These two assessments were averaged together for subsequent analyses. The overall association between narcissism and cortisol was marginally significant and positive ($r = 0.18$, $p = 0.07$, 95% CI $[-0.02, 0.38]$, $n = 98$; after controlling for sex, this effect became significant: $\beta = 0.23$; $p = 0.04$). Additionally, there was a significant narcissism \times sex interaction predicting cortisol ($\beta = 0.23$ – 0.27 ; $p = 0.08/0.04$; estimates and significance varied depending on the covariates included). Narcissism was positively associated with cortisol among men ($r = 0.42$, $p = 0.04$, 95% CI $[0.24, 0.66]$, $n = 25$) but not women ($r = 0.07$, $p = 0.56$, 95% CI $[-0.16, 0.30]$, $n = 73$). Moreover, analyses of specific facets of narcissism revealed that unhealthy narcissism (i.e., entitlement/exploitativeness) was positively associated with cortisol levels overall ($\beta = 0.39$; $p < 0.05$) and among both men ($\beta = 0.72$; $p = 0.01$) and women ($\beta = 0.27$; $p = 0.06$); the sex \times unhealthy narcissism interaction was marginally significant ($\beta = 0.49$; $p < 0.10$). Healthy narcissism was not significantly related to cortisol levels overall ($\beta = -0.12$; $p > 0.05$) or when analyses were conducted separately for men ($\beta = -0.30$; $p = 0.26$) and women ($\beta = -0.13$; $p = 0.36$); the sex \times healthy narcissism interaction was not significant ($\beta = -0.05$; $p > 0.05$). Reinhard et al. interpreted these findings as suggesting that narcissistic men, especially those with high levels of entitlement and exploitativeness, may have particularly high levels of baseline cortisol, perhaps reflecting chronic HPA activation, even in the absence of an explicit social stressor.

Thus, taken together, there is conflicting evidence regarding the association between narcissism and basal cortisol. One study found a positive association between narcissism and baseline cortisol levels among men. Another study found no significant association between narcissism and baseline cortisol levels among either men or women. However, there are several methodological differences across studies that may have contributed to these divergent results. Edelstein, Yim et al. (2010)'s sample included 90 undergraduate participants with approximately equal numbers of men and women. Reinhard et al. (2012) sampled 106 undergraduate participants but included very few men (~25%). Findings based on small sample sizes and p -values just under $p = 0.05$ may be less replicable and trustworthy effects (Simonsohn, Nelson, & Simmons, 2014). As we describe below, 63 men would be required (at minimum) to replicate the significant association between narcissism and men's *baseline* cortisol obtained by Reinhard et al. (2012). It is also likely that studies with small samples greatly overestimate the effect size and a much larger sample would be required to find an effect should it exist (Open Science Collaboration, 2015; Slavin & Smith, 2009). Thus, a reexamination of these findings in a larger sample that includes more men is warranted. In our replication study, we include similar proportions of men and women and a larger sample size than these two studies combined.

Second, the two studies differed in their operationalization of baseline cortisol concentrations. It is important to note that, because the cortisol response is relatively slow, reactivity to a particular event can only be detected in saliva approximately 20 min following that event (Dickerson & Kemeny, 2004). Edelstein, Yim et al. (2010) obtained their first saliva samples 20 min after an adaptation (relaxation) period, giving participants time to adapt to the laboratory context, which could have contributed to the null

association between narcissism and baseline cortisol. Reinhard et al. (2012), in contrast, collected baseline saliva at two time points: directly after consent and after 25 min of filler tasks. One advantage of this approach is that the aggregation of the two "baseline" samples arguably improved measurement reliability for participants in the Reinhard et al. (2012) study (Liening et al., 2010). However, this aggregated measure also likely reflected events that may have occurred both prior to and after entering the lab. Anxiety about the novel setting or participating in research could, therefore, have contributed to the baseline measurement, perhaps contributing to the positive association between narcissism and (men's) cortisol. Thus, although Reinhard et al. (2012) may have benefitted from the greater reliability of aggregating two samples, it is nevertheless unclear what psychological state was being assessed, given the timing of their measurement within a session. Because all participants in the current replication study provided saliva samples after a 20-min adaptation period, our baseline cortisol measurements are less dependent on events occurring outside the laboratory. Although we do not have multiple baseline assessments per participant, we have increased measurement precision by assessing cortisol in a much larger sample of participants.

The primary goal of the present study was to replicate the association between narcissism and *basal* cortisol as reported in Reinhard et al. (2012). We simultaneously address previous methodological inconsistencies in the number and gender of participants and measurement of baseline cortisol. From a practical perspective, understanding individual differences in baseline cortisol levels could help identify who is at an increased risk for certain types of health problems (e.g., cardiovascular and immune health). From a theoretical perspective, our findings can contribute to knowledge about individual differences in physiological and health outcomes.

2. Method

2.1. Participants and procedure

Our data were collected as part of multiple projects that examined associations between personality traits and hormones (Edelstein, Chopik, & Kean, 2011; Edelstein, Kean, & Chopik, 2012; Edelstein, Stanton, Henderson, & Sanders, 2010). Participants were 366 undergraduate students (48.2% female) for whom data on both narcissism and cortisol levels were available. They received course credit or monetary compensation for their participation. Participants ranged in age from 18 to 37 ($M = 19.24$, $SD = 2.06$) and the ethnic composition of the sample was 63.8% Caucasian, 17.3% Asian-American, 7.7% African-American, and 11.2% of mixed or other ethnicities. Female participants were tested during all phases of their menstrual cycles and women reported being, on average, 18.88 days ($SD = 17.09$) past the onset of their last menstruation. Twenty-two percent of women reported being on oral contraceptives.² We have not previously published data on cortisol or narcissism from these samples.

All procedures were approved by the University of Michigan Institutional Review Board. Participants were asked to refrain from eating, drinking (except for water), smoking, or brushing their teeth for one hour prior to the beginning of their experimental session. After informed consent was obtained, participants provided a saliva sample that was later used to assess baseline cortisol levels. Participants then completed a series of questionnaires, including

² Women on hormonal contraceptives did not differ in cortisol levels compared to women not on hormonal contraceptives ($t = 0.40$, $p = 0.69$) and contraceptive use did not moderate the association between narcissism and women's cortisol ($b = -0.03$, $\beta = 0.01$, $t = -0.06$, $p = 0.95$). Thus, contraceptive use is not considered further.

measures of personality and demographics, which included information about oral contraceptive use and any other medical conditions that might affect hormone levels.

2.2. Narcissism measure

2.2.1. Narcissistic personality inventory

Grandiose narcissism was assessed using the 40-item Narcissistic Personality Inventory (NPI-40; Raskin & Terry, 1988). Participants chose one of two options they most identified with (e.g., “The thought of ruling the world frightens the hell out of me” vs. “If I ruled the world it would be a better place”). Responses were averaged, such that higher scores indicate greater levels of grandiose narcissism. Internal consistency of the NPI in the present study was 0.83.

Reinhard et al. (2012) found that cortisol was associated with “unhealthy narcissism” (e.g., items related to entitlement and exploitativeness) and unrelated to “healthy narcissism” (e.g., items related to leadership, authority, self-sufficiency, superiority, and vanity) (Watson & Biderman, 1993). Thus, in an exploratory analysis, we examined whether these subscales of narcissism were related to cortisol. *Unhealthy* (maladaptive) narcissism scores were created by summing the Entitlement and Exploitativeness subscales of the NPI; *healthy* (adaptive) narcissism scores were created by summing the Leadership/Authority, Self-Sufficiency, Superiority, and Vanity subscales (Watson & Biderman, 1993). Internal consistency of the Healthy and Unhealthy subscales in the present study was 0.62 and 0.75, respectively.

Given the psychometric history of the NPI, we also examined whether the subscales identified by previous researchers were associated with cortisol by considering two additional solutions in the exploratory analyses: a three-factor solution (i.e., leadership/authority ($\alpha = 0.76$), entitlement/exploitativeness ($\alpha = 0.52$), and grandiose exhibitionism ($\alpha = 0.71$)) (Ackerman et al., 2011), and a four-factor solution (i.e., leadership/authority ($\alpha = 0.76$), self-absorption/self-admiration ($\alpha = 0.66$), superiority/arrogance ($\alpha = 0.41$), and exploitativeness/entitlement ($\alpha = 0.58$)) (Emmons, 1984).

2.2.2. Self-esteem

The Rosenberg Self-Esteem scale includes 10 items that assess global self-esteem (e.g., “On the whole, I am satisfied with myself.”). Items are rated on a 4-point scale ranging from 1 (*strongly disagree*) to 4 (*strongly agree*). Items were averaged to yield an overall score for self-esteem ($\alpha = 0.76$). Because narcissism and self-esteem are positively intercorrelated, we ran an additional exploratory analysis controlling for self-esteem (Campbell, Rudich, & Sedikides, 2002; Sedikides, Rudich, Gregg, Kumashiro, & Rusbult, 2004).

2.3. Salivary cortisol: Collection and assessment

Participants produced 7.5 mL of saliva in a sterile polypropylene vial. The vials were placed in frozen storage immediately after the experimental session was complete. The majority of saliva was collected via passive drool, as in Reinhard et al., but a subset ($n = 100$; Edelstein, Stanton et al., 2010) was collected using sugarless gum as a saliva stimulant. (Edelstein, Yim et al., 2010 used a salivette sampling device). The results reported below are consistent when comparing samples that did and did not use gum to stimulate saliva. The majority of participants (88.5%) completed the study after 12 PM to control for diurnal changes in cortisol, and all samples were collected between 9:50 AM and 7:15 PM ($M_{time} = 2:58$ PM, $SD = 2$ h). We also included time of day as a covariate in our analyses. We did not collect data on waking times and thus participants' number of waking hours was not available for

analysis in the current study. Samples were placed in frozen storage in our laboratory immediately after collection until further processing in the University of Michigan Core Assay Facility. Samples were freed from mucopolysaccharides and other residuals by three freeze–thaw cycles followed by centrifugation. All samples were assayed by radioimmunoassay (RIA), using commercially available kits from Salimetrics, Inc. and samples were assayed in duplicate. The inter-assay CV for cortisol at high levels ranged from 13.25% to 21.50% and at low levels ranged from 0.39% to 5.04%. The intra-assay CV ranged from 2.75% to 6.01%.

2.4. Sample size calculations

To determine the minimum sample size required to detect a significant association between narcissism and cortisol, we conducted a power analysis informed by the standardized regression estimates from Reinhard et al. (2012), the one study that reported significant associations between narcissism and baseline cortisol levels. Typically, a partial R^2 can be used as a measure of effect size to calculate the number of participants needed to find a particular effect. However, Reinhard et al. (2012) reported only standardized regression coefficients and not the corresponding t -values or R^2 (or partial correlation) statistics. As a remedial measure, a standardized beta coefficient, which is mathematically similar to a partial correlation, was used in the power analysis. Standardized beta coefficients are actually more *conservative* estimates of effect size as they are either equal to or less than the partial correlation. The association between (men's) total narcissism scores and cortisol in the Reinhard et al. (2012) study was significant and positive, $\beta = 0.23$, $p < 0.05$. Using the standardized beta as a stand-in for a partial correlation, we calculated an effect size of $f^2 = 0.056$. The minimum required sample size to replicate an effect (at $f^2 = 0.056$) is 235 participants (at 95% power) or 143 participants (at 80% power). To replicate the significant association between unhealthy narcissism and cortisol ($\beta = 0.42$, $p < 0.05$; $f^2 = 0.176$), the minimum sample size required is 63 (for 95% power) and 39 (at 80% power).

The sample size in the current study is 366 participants (47% female), which enables us to conduct a strong replication attempt of the Reinhard et al. (2012) effects as well as detect an effect as small as $f^2 = 0.036$ (at 95% power) and $f^2 = 0.022$ (at 80% power).

2.5. Transformations and flexibility in data analysis

To maximize the use of all available data, cortisol values that were larger than three standard deviations above the mean were replaced with values corresponding to three standard deviations above the mean (i.e., Winsorized; Reifman & Keyton, 2010). This Winsorization procedure was applied to ten participants (2.7% of the total sample). As in both previous studies (Edelstein, Yim et al., 2010; Reinhard et al., 2012), (Winsorized) cortisol values were log-transformed to reduce skewness. Results reported below did not differ when these 10 participants were excluded rather than Winsorized. We also conducted a series of supplementary analyses examining narcissism–cortisol associations using raw (i.e., untransformed) cortisol values and cortisol values that were z -scored within each of the three samples. This z -score approach was adopted to alleviate the concern that some between-subject differences in cortisol levels may stem from the fact that, although our saliva samples were assayed at the same core assay facility and using the same kits, these saliva samples were obtained from different studies and were thus assayed in different batches at different times. The results from regressions predicting raw cortisol (Supplementary Table 1) and z -score cortisol (Supplementary Table 2) did not substantively differ from those presented in

Table 2. Adding study source as a covariate in our analyses did not change any of the effects reported below.

2.6. Analytic plan

Our goal was to reexamine the association between narcissism and baseline cortisol levels in men and women to resolve the contradictory findings reported in previous work. We conducted linear regression analyses predicting variability in (log-transformed) cortisol from narcissism, gender, and their interaction, controlling for time of day of cortisol assessment. We entered gender and narcissism into the regression model on Step 1, time of day on Step 2, and the interaction between gender and narcissism on Step 3. As is recommended, all continuous variables were centered prior to creating the interaction terms (Cohen, Cohen, West, & Aiken, 2003). Gender was contrast-coded.

In the first exploratory analysis, we tested whether baseline cortisol was related to different facets of narcissism (healthy vs. unhealthy). Reinhard et al. (2012) found that cortisol was associated with unhealthy aspects of narcissism (e.g., entitlement) but was unrelated to healthier aspects (e.g., leadership); we also examined whether any of the narcissism subscales identified by previous researchers (see Ackerman et al., 2011; Emmons, 1984; Watson & Biderman, 1993) were associated with cortisol. In the second exploratory data analysis, we re-ran our main analyses controlling for self-esteem.

3. Results

3.1. Confirmatory analyses

3.1.1. Preliminary analyses

Table 1 reports the bivariate correlations between narcissism and baseline cortisol values: raw (pre-Winsorizing), raw (post-Winsorizing), log-transformed, and z-scored. The raw, log-transformed, and z-scored values of cortisol were highly intercorrelated, suggesting that relations between narcissism and cortisol should be similar across raw and standardized values.

Table 1
Bivariate correlations between narcissism and cortisol (untransformed, log-transformed, z-scored).

	M	SD	1	2	3	4	5
1. Gender	–	–					
2. Narcissism	1.41	0.17	0.15* [0.05,0.25]				
3. Cortisol (untransformed; un-Winsorized; nmol/L)	2.78	2.24	0.02 [–0.10,0.12]	–0.05 [–0.15,0.05]			
4. Cortisol (untransformed; Winsorized; nmol/L)	2.72	1.97	0.01 [–0.09,0.11]	–0.03 [–0.13,0.07]	0.97* [0.96,0.98]		
5. Cortisol (log-transformed)	0.79	0.62	0.04 [–0.06,0.14]	–0.02 [–0.12,0.10]	0.89* [0.87,0.91]	0.93* [0.92,0.94]	
6. Cortisol (z-scored)	0.00	1.00	0.02 [–0.10,0.12]	–0.06 [–0.16,0.04]	0.87* [0.84,0.89]	0.91* [0.89,0.93]	0.96* [0.95,0.97]

Note. *N*s range from 362 to 365. Gender: –1 = Women, 1 = Men. Confidence intervals are presented in brackets.
* $p < 0.01$.

Table 2
Regression analyses predicting cortisol (log-transformed).

	Step 1					Step 2					Step 3				
	<i>b</i>	<i>SE</i>	β	<i>t</i>	<i>p</i>	<i>b</i>	<i>SE</i>	β	<i>t</i>	<i>p</i>	<i>b</i>	<i>SE</i>	β	<i>t</i>	<i>p</i>
Intercept	0.79	0.03		24.13	<0.001	2.52	0.21		11.78	<0.001	2.51	0.21		11.76	<0.001
Narcissism	–0.11	0.19	–0.03	–0.59	0.55	–0.13	0.18	–0.04	–0.71	0.48	–0.14	0.18	–0.04	–0.77	0.44
Gender	0.04	0.03	0.06	1.06	0.29	0.04	0.03	0.06	1.30	0.20	0.04	0.03	0.06	1.31	0.19
Time of day						<0.001	<0.001	–0.40	–8.15	<0.001	<0.001	<0.001	–0.40	–8.15	<0.001
Narcissism × Gender											0.09	0.18	0.03	0.52	0.61
<i>R</i> ²				0.004					0.16					0.16	
<i>F</i>				0.66	0.52				22.69	<0.001				17.05	<0.001
ΔR^2									0.16					0.001	
ΔF									66.49	<0.001				0.27	0.61

Note. Gender: –1 = Women, 1 = Men.

Indeed, narcissism was not significantly correlated with baseline cortisol, regardless of whether cortisol values were raw, log-transformed, or z-scored. Consistent with previous research, men had higher narcissism scores than women (e.g., Grijalva, Harms, Newman, Gaddis, & Fraley, 2015).

3.1.2. Regression analyses

We conducted linear regressions predicting basal cortisol from narcissism and gender (Step 1), narcissism, gender, and time of day (Step 2), and narcissism, gender, time of day, and the interaction between narcissism and gender (Step 3). The narcissism × gender interaction was included to test whether the strength of the association between narcissism and cortisol was different between men and women, as found in previous research (Reinhard et al., 2012). The inclusion of covariates and interactions in separate steps was done to examine how the association between narcissism and cortisol varied according to the presence of other variables that might explain additional variance (Simmons, Nelson, & Simonsohn, 2011).

The results of these analyses are presented in Table 2. Consistent with the bivariate associations, narcissism was unrelated to baseline cortisol. Further, the narcissism × gender interaction was not a significant predictor of cortisol, suggesting that the association between narcissism and baseline cortisol was similar in size for both men and women. The regression analyses predicting raw cortisol (Supplementary Table 1) and z-scored cortisol (Supplementary Table 2) did not substantively differ from the results presented in Table 2.

3.2. Exploratory analyses

3.2.1. Narcissism subscales

Given the psychometric history of the NPI, we also examined whether the subscales identified by previous researchers were associated with cortisol. We considered three different solutions: a two-factor solution (i.e., healthy and unhealthy narcissism; Watson & Biderman, 1993), a three-factor solution (i.e., leadership/authority, entitlement/exploitativeness, and grandiose

exhibitionism; Ackerman et al., 2011; Reinhard et al., 2012), and a four-factor solution (i.e., leadership/authority, self-absorption/self-admiration, superiority/arrogance, and exploitativeness/entitlement; Emmons, 1984). Indeed, Reinhard et al. (2012) found that cortisol was associated with “unhealthy narcissism” (e.g., items related to entitlement and exploitativeness) but not with “healthy narcissism” (e.g., items related to leadership, authority, self-sufficiency, superiority, and vanity) (Watson & Biderman, 1993).

To test whether these factors were associated with cortisol, we ran linear regressions in which each of these factors were entered as predictors of our three measures of cortisol. Results from these analyses are presented in Supplementary Tables 3 (for the two-factor solution), 4 (for the three-factor solution), and 5 (for the four-factor solution). Across all of these different solutions and cortisol measures, no narcissism factors were associated with cortisol.

3.2.2. Self-esteem analyses

Self-esteem and narcissism were positively intercorrelated ($r = 0.23$, $p < 0.001$), consistent with previous research (Campbell et al., 2002; Sedikides et al., 2004). Self-esteem was not significantly correlated with basal cortisol levels, however ($r = 0.03$, $p = 0.60$). In an exploratory regression analysis, we examined the association between narcissism and cortisol, controlling for self-esteem. As shown in Supplementary Table 6, neither self-esteem nor narcissism was significantly related to cortisol in a regression controlling for gender and time of day.

4. Discussion

The goal of the present study was to reexamine the association between narcissism and baseline cortisol in a larger sample of men and women in an attempt to reconcile previously inconsistent findings. One prior study reported a null association between narcissism and baseline cortisol in both men and women (Edelstein, Yim et al., 2010); a second study found a positive association between men’s baseline cortisol and narcissism and particularly higher levels of unhealthy narcissism (i.e., entitlement and exploitativeness) (Reinhard et al., 2012). Baseline cortisol levels may reflect activity of the HPA axis and may thus provide a window into narcissists’ physiological and health functioning according to previous research. In the current study, we addressed methodological differences between prior studies by including similar proportions of men and women, a larger sample size than these two studies combined, and baseline cortisol measurements that were less dependent on events occurring outside the laboratory.

Our results were consistent with those reported by Edelstein, Yim et al. (2010), such that narcissism was unrelated to baseline cortisol for both men and women, regardless of whether cortisol values were in raw form, log-transformed, or z-scored. We additionally examined whether baseline cortisol was related to different facets of narcissism (see Ackerman et al., 2011; Emmons, 1984; Watson & Biderman, 1993); however, none of the various narcissism factors were associated with men or women’s baseline cortisol. Overall, we did not find evidence to support the association between narcissism and basal cortisol reported by Reinhard et al. (2012). Thus, at least in an undergraduate sample such as our own, narcissism may not be related to chronic HPA activation (in the absence of an explicit social stressor).

Did we replicate Reinhard et al.’s finding that narcissists have higher baseline cortisol? Unfortunately, there is no single, definitive criterion for evaluating whether a result successfully replicates an effect found in the original study. We evaluated our replication attempt according to three criteria, the first two of which were adopted by the Reproducibility Project (Open Science

Collaboration, 2015). The first criterion is whether the replication effect is significantly different from zero and in the same direction as the original effect. Our effect was not different from zero and, if anything, was in the opposite direction than the original effect, suggesting a failure to replicate. The second criterion is whether the effect size of the original study fell within the confidence interval observed in the replication study, suggesting that the original effect was a plausible value detectable by the replication study. The original effect size ($r = 0.18$) fell outside of the confidence interval in the replication study ($-0.12, 0.10$), again suggesting a failure to replicate. The third criterion is whether the observed effect from the replication attempt is too small to have been detected in the original study (i.e., the “small telescopes” approach; Simonsohn, 2015). In this approach, researchers calculate the largest effect size detectable if the original study had 33% power to detect an effect. If the replication effect size is lower than this effect size, there is little evidence for replication. Reinhard et al.’s original sample size of 106 could detect an r of 0.15 with 33% power. The current effect size in the replication attempt was $r = -0.02$, which is lower than this lower bound and again suggests that we failed to replicate their original finding. Thus, given these three criteria, we consider the current study a failure to replicate the original effect of Reinhard et al. However, it is worth acknowledging that there are additional criteria on which to judge replications (Maxwell, Lau, & Howard, 2015; Tryon, 2016). Future research can contribute additional replications of narcissism–cortisol associations to the literature, and meta-analyses of these combined studies can more accurately estimate the effect size of this association.

Of course, our findings should be considered in light of the limitations of our study. First, our use of chewing gum as a stimulant for saliva in one of the samples was potentially problematic (Schultheiss, 2013). Specifically, there is some evidence that the use of chewing gum might attenuate the levels of cortisol (or other hormones, van Anders, 2010) measured in saliva, which may have reduced our ability to detect an effect. Worth noting, however, is that the two remaining samples *did not* use chewing gum. In a supplementary analysis, we found that narcissism was unrelated to cortisol in the sample that did use chewing gum ($\beta = -0.03$, $p = 0.70$) and in the samples that did not use chewing gum ($\beta = -0.05$, $p = 0.43$). Further, the Edelstein, Yim et al. study did not use chewing gum and found a null association between narcissism and cortisol. Thus, associations between narcissism and cortisol likely do not depend on whether salivary cortisol was collected using chewing gum. It is also unlikely that the use of chewing gum completely invalidates effects observed with cortisol (O. Schultheiss, personal communication, June 7, 2016), particularly given the large volume of replicable results published prior to the revelation that chewing gum poses a methodological problem. Nevertheless, future research examining personality–hormone associations can use passive drool techniques exclusively and/or examine whether these associations hold under the use of other saliva stimulants (Schultheiss, 2013).

In addition, although our sample size is large relative to past research and particularly in the context of studies of hormone–personality links, the effect sizes that we estimated are likely to be quite small. Despite finding a null association that was in the opposite direction of Reinhard et al., it could be that our sample size was still too small to detect an effect should it exist. Thus our study is far from definitive regarding how narcissism is related to physiological functioning. Future studies should extend our findings with larger samples and more diverse populations. Future research can also further elucidate the mechanisms that might link narcissism to higher or lower levels of cortisol (e.g., Cheng, Tracy, & Miller, 2013). Reinhard et al. (2012) suggest that narcissists’ defensive strategies may be associated with greater cardiovascular

reactivity, higher blood pressure, and cardiovascular disease. Although there is some evidence for defensive coping styles being maladaptive over long periods of time (Rutledge, 2006), it is unclear whether narcissism is causally linked to defensive strategy used, which in turn leads to higher stress and ultimately higher circulating levels of cortisol.

These limitations notwithstanding, our findings suggest that associations between narcissism and baseline cortisol levels might be somewhat complex. Given the health implications of cortisol reactivity, understanding individual differences in cortisol output could help identify who is at an increased risk for certain types of health problems, including issues with cardiovascular and immune health. Future research should examine associations between narcissism and cortisol in larger, more diverse samples and over time to better understand how narcissism may be associated with long-term health outcomes (e.g., Edelstein, Newton, & Stewart, 2012).

Author note

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Appendix A. Supplementary materials

Supplementary data associated with this article can be found, in the online version, at <http://dx.doi.org/10.1016/j.jrp.2016.07.006>.

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