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The longitudinal association between individual differences in recall of positive specific autobiographical memories and daily cortisol

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Abstract

The present study examines the longitudinal association between cortisol (dys)regulation – mean cortisol awakening response (CAR) and area under the curve with respect to ground (AUCg) for total daily cortisol – and autobiographical memory. 135 participants (*mean age at baseline* = 16.1; *Females* = 78.5%) provided cortisol samples (T1). Seven months later participants retrieved autobiographical memories cued by positive and negative words (T2). Four years subsequently, participants provided cortisol samples again (T3). The retrieval of more specific memories cued by positive words, but not negative words, was associated with higher AUCg four years later, independent of sex, recent life stressors and self-reported negative self-related cognitions. There were no associations between CAR and autobiographical memory. Neither AUC nor CAR at T1 predicted subsequent autobiographical memory abilities. People who retrieve more positive specific memories may be more likely to imagine and seek out positive experiences and this may be associated with higher cortisol levels.

Introduction

Problems recalling memories of specific autobiographical events (e.g., *walking my dog in the park last week*) are common amongst people with a range of psychiatric disorders (Barry, Del Rey, et al., 2018; Farina et al., 2019; Ono et al., 2015) and have also been associated with exposure to stressful or negative life events (Barry, Lenaert, et al., 2018; Ono et al., 2015). These memory problems are thought to be related to perturbed activation in areas of the brain such as the prefrontal cortex and hippocampus (Barry, Chiu, et al., 2018; Young et al., 2012, 2013). As these brain regions are rich in glucocorticoid receptors, and as psychiatric diagnoses (Adam et al., 2017) and exposure to negative life events (Doane et al., 2013; Stroud et al., 2019) have both been associated with dysregulated cortisol, researchers have also examined whether difficulty recalling specific autobiographical memories is associated with dysregulated cortisol (re)activity. The present study investigates the longitudinal association between difficulty recalling specific autobiographical memories and dysregulated cortisol in order to better understand the mechanisms by which reduced specificity might emerge or might influence a person's subsequent ability to manage life stress.

Results regarding the association between autobiographical memories and cortisol are mixed. Increased cortisol levels, through the administration of hydrocortisone or in response to a psychosocial stressor, have been found to induce problems recalling specific autobiographical memories (Buss et al., 2004; Schlosser et al., 2010; Tollenaar et al., 2009). However, other studies have failed to replicate these effects (Fleischer et al., 2017; Wingenfeld et al., 2012) or found *improved* recall of specific autobiographical memories after administration of hydrocortisone relative to placebo (Wingenfeld et al., 2012). Similarly, when sampling cortisol levels on a single afternoon, participants with higher cortisol were found to recall more specific autobiographical memories than participants with lower cortisol (Barnhofer et al., 2005). In a longitudinal study, the recall of more specific

autobiographical memories in response to positive cue words (e.g., *happy*) was associated with lower morning cortisol levels a year later (Askelund et al., 2019). People who were better able to access positive autobiographical memories were posited to be able to use these memories to regulate negative emotions and thoughts about themselves that in turn regulated cortisol output over time (Askelund et al., 2019). Together these findings suggest that cortisol and autobiographical memory are associated with another, however, there are limitations to existing studies that may explain their mixed findings.

First, hypercortisolism has been associated with structural (Bruehl et al., 2009; Cox et al., 2017; Geerlings et al., 2015) and functional (Abercrombie et al., 2011; Henckens et al., 2012; van Stegeren et al., 2010) abnormalities in the hippocampus, and the hippocampus plays an important role in the retrieval of specific autobiographical memories (Young et al., 2013). As such, although previous research indicates that retrieval of specific autobiographical memories can predict cortisol levels (Askelund et al., 2019), it is also likely that these levels in turn predict specific autobiographical memory retrieval abilities through their effects on the hippocampus. To our knowledge, no study has examined whether cortisol levels longitudinally predict subsequent memory specificity or whether differences in memory specificity mediate the association between cortisol levels measured at two times.

Second, Askelund et al. (2019) quantified morning cortisol in terms of cortisol levels at 8am. Morning cortisol samples that are taken at a fixed time (e.g., 8am) have been found to have poor test-retest reliability, especially compared to samples that are waketime-aligned (Pruessner et al., 1997). Best practice guidelines recommend that morning cortisol is best quantified as the change in cortisol from waking up to within 45 minutes later – the cortisol awakening response (CAR) – as non-waketime-aligned cortisol measures are likely to be contaminated by individual differences in waking time (Stalder et al., 2016). The CAR is thought to represent preparation for the physical and cognitive demands of the day, however,

dysregulated CAR (too much/too little) has been associated with a range of negative psychiatric outcomes (Adam et al., 2010; Clow et al., 2004). To our knowledge, no study has examined the association between CAR and memory specificity.

Third, CAR is not the only index of cortisol dysregulation. Total cortisol secreted daily captures morning cortisol levels as well as levels at other stages of the day. CAR and total cortisol are likely to represent distinct aspects of hypothalamic-pituitary-adrenal axis (dys)function (Golden et al., 2013). Golden et al. (2013) suggest that the CAR may reflect HPA responsiveness to acute stress and awakening whereas diurnal cortisol amy reflect the cumulative burden of stress and broader dysregulation in arousal, metabolic and immune processes, that explain its association with a range of negative physical and mental health outcomes (Adam et al., 2017). This is of particular relevance here given the suggestion that chronic stress can lead to impairments in autobiographical memory specificity (Barry, Lenaert, et al., 2018; Ono et al., 2015) as well as evidence that cortisol levels in the afternoon may also be associated with recall of specific autobiographical memories (Barnhofer et al., 2005). As such, there is a need for longitudinal studies that measure CAR as well as other important indices of cortisol levels throughout the day in order to clarify which facets of cortisol activity are associated with autobiographical memory.

The present investigation used existing data from the Youth Emotion Project (YEP; Zinbarg et al., 2010) regarding diurnal cortisol, autobiographical memory specificity, and other important covariates (e.g., life stress, dysfunctional attitudes towards the self). YEP assessed cortisol levels at an initial assessment and at the same time included a range of other self-report and interview measures (Zinbarg et al., 2010). So as not to over-burden participants, a measure of autobiographical memory specificity, the Autobiographical Memory Test (AMT)(Williams & Broadbent, 1986) was included at a second assessment some months later; cortisol was in turn assessed again at a third timepoint some years

thereafter. At each cortisol assessment, salivary cortisol levels were sampled on each of several successive days immediately after waking, 40 minutes later and a further four times throughout each day.

Measuring cortisol in this way enables us to follow up on previous investigations that have examined morning cortisol but which have not been able to account for differences in waking time within their analysis (Askelund et al., 2019). We can also examine whether any observed effects are specific to morning cortisol or whether there are also effects evident for mean total daily cortisol levels – operationalised as Area Under the Curve with respect to ground (AUCg) – or any other specific sample time, such as in the afternoon as indicated by the findings of Barnhofer et al. (2005). Also, our analysis includes a measure of chronic life stress over the year prior to the cortisol assessments (Hammen, 1991; Hammen et al., 1987). The lengthy follow-up therefore also enables us to explore the long-term association between autobiographical memory and cortisol and the contribution of chronic stressors to these effects.

Participants with high levels of morning and daily cortisol at baseline (Time 1; T2) were expected to have difficulty recalling specific autobiographical memories at a subsequent timepoint (Time 2; T2). This difficulty recalling specific memories was in turn expected to predict heightened cortisol levels measured subsequently (Time 3; T3). The available data enable us to examine these longitudinal associations between timepoints in isolation as well as testing whether cortisol at T1 is associated with cortisol at T3, through autobiographical memory measured at T2. As in previous research, we examine these effects whilst additionally accounting for differences in life stress and negative self-related cognitions (Askelund et al., 2019). As Askelund et al. (2019) reported differential effects between memories retrieved for positive and negative cues and their association with cortisol, the number of specific memories retrieved for each of these cues were analysed separately.

Method

Participants

Participants were drawn from the University of California, Los Angeles (UCLA) and Northwestern University (NU) Youth Emotion Project (YEP), a longitudinal study of risk factors for psychopathology in late adolescence to early adulthood (total $N = 627$). High school juniors were recruited from two demographically diverse public schools in greater Los Angeles and Chicago and followed annually for up to nine years. A full description of the recruitment process, along with sample characteristics and other measures given to participants, is provided elsewhere (Zinbarg et al., 2010). 135 participants who completed cortisol sampling, which was collected during the first four years of the study, and the autobiographical memory test were eligible for inclusion in the analyses.

The participants included in the present analysis (*mean age at baseline* = 16.1, *SD* = 0.4, *range* = 15-17) were mostly female (78.5%) and White (52.6%); 14.1% identified as Hispanic or Latino, 12.4% African American or Black, 4.4% Asian, 5.2% identified as an unlisted racial/ethnic group and 11.1% identified as multiracial/multi-ethnic. Participants' mean socioeconomic status ($M = 49.2$; $SD = 12.2$), as measured using the Hollingshead index (Hollingshead, 1975) indicated that their parents were mostly minor professionals (e.g., sales representatives) or workers at mid-sized businesses.

As the sample was drawn from available data from participants in a broader study, no a priori analysis of required sample size was conducted. However, the present sample size was within the range of 10 participants per the maximum number of model parameters (13) as recommended elsewhere (Askelund et al., 2019). Post-hoc sensitivity analysis with alpha level set at .05, 80% power and 13 predictors, suggested that 135 participants was sufficient to detect medium sized effects ($f^2 = .14$).

Measures

Autobiographical memory specificity

Autobiographical memory specificity was assessed using the Autobiographical Memory Test (AMT)(Williams & Broadbent, 1986). Participants were presented, in alternating order, six positive (safe, ambitious, peace, hope, brave, interested) and six negative cues (disappoint, inferior, hurt, frustrated, tense, regret) and for each cue word they were asked to retrieve a personally experienced past event. Participants' verbal responses were recorded and later coded as specific (an event lasting less for 24 hours or less), extended (an event lasting more than a day), categoric (an event that occurred several times), semantic associate (a concept semantically related to the cue word) or omission (no response). As reported elsewhere (Barry, Vinograd, et al., 2019) within-site and cross-site analyses of the reliability of the these codes was good (both $\kappa = .78$). Askelund et al. (2019) used a ratio index of memory specificity (the ratio of specific autobiographical memories retrieved [those referring to personally experienced events lasting less than 24 hours] relative to the number of categorical memories retrieved [those referring to personally experienced events that occurred frequently; e.g., *when I walk my dog*]). Our analyses test the same ratio index for both positive and negative memories separately. Also, as this index is not typical within the literature our analysis uses a typical index of specificity for which there is psychometric support, the raw number of specific memories that participants recalled (Griffith et al., 2009; Griffith, Kleim, et al., 2012; Griffith, Sumner, et al., 2012; Heron et al., 2012; Takano et al., 2017).

Dysfunctional attitudes

Participants' negative cognitions about themselves and others were assessed using the 40-item self-report Dysfunctional Attitudes Scale Form A (DAS-A)(Weissman & Beck, 1978). Participants rated the extent to which they agreed with each of 40 statements (e.g., *If a person asks for help, it is a sign of weakness*) on 7-point Likert scales (1= fully disagree; 7 = fully

agree). A higher score represents greater dysfunction. At baseline assessment (Time 1; T1), 11 participants were missing one item and one participant was missing two items. One participant was missing all items. At follow-up assessment (Time 3; T3), eight participants were missing one item and nine participants were missing all items. For participants with one or two missing data points, person-mean data were imputed. Participants with all missing data were retained for analyses as DAS was included as a covariate only within the analyses. In the present study internal consistency for the DAS at both timepoints was acceptable (T1 ωh = .79; T3 ωh = .65).

Life stress

The presence of chronic interpersonal life stress was assessed using the semi-structured UCLA Life Stress Interview (LSI)(Hammen, 1991; Hammen et al., 1987). This measure quantifies the degree of interpersonal stress related to one's closest friend, social circle, romantic life (whether one is in a committed relationship or is *dating*) and family over the year prior to the interview. The LSI examines the degree of stress or conflict within each relationship domain and the degree of support or mutuality that is perceived within the relationship. Each domain then receives a score by trained interviewers from one (*superior conditions*) to five (*exceptionally poor conditions*) such that a higher score is indicative of greater stress. Interviewers were trained to consider scores of one and five to be rare and relatively extreme cases and used general probes to elicit relevant, objective information from participants. Focus was placed on interpersonal stress as previous research has shown that memory specificity is associated positively with the quality of interpersonal relationships (Barry, Vinograd, et al., 2019). As in this previous study, the stress score in each of the domains was totaled to give an overall score at each timepoint. LSI scores were available at T1 and T3. There was no missing LSI data at T1. At T3, one participant was missing a score for one of the domains of relational stress and one participant was missing data for all domains. These participants were retained

for analysis as LSI was included as a covariate only in the analysis. No data were imputed. In the present study, the LSI showed good intra-class coefficients (ICC) within-site at T1 (ICC = .87) and T3 (ICC = .81) and cross-site at T1 (ICC = .84) and T3 (ICC = .80). LSI interviewers were blind to participants' scores on the other measures.

Cortisol

The protocol for cortisol sampling has been described elsewhere (Adam et al., 2010, 2014; Doane & Adam, 2010). Participants were asked to passively drool six times per day for three consecutive days: immediately after waking and then 40 minutes, three hours, eight hours and twelve hours post-waking, and then again at bedtime. Participants were instructed not to eat, drink or brush their teeth within the 30 minutes prior to the samples taken at waking, 40 minutes post-waking and bedtime. Participants stored their samples in their refrigerators and returned them to their school or they mailed them. Samples were refrigerated in the lab at -20ºC. Once all samples were received they were shipped to Trier, Germany and were assayed in duplicate using time-resolved fluorescent-detection immunoassay (Intra-assay variation = 4.0 - 6.7%; Inter-assay variation = 7.1% - 9.0%).

A Cortisol Awakening Response (CAR) index was computed by subtracting the cortisol levels at waking from levels 40 minutes later. An index of total cortisol across the day was also computed in terms of the area under the curve with respect to ground (AUCg) for the cortisol levels for each of the six sample points. Indices were calculated for each day and then averaged across the three days. In line with the procedure of Askelund et al. (2019) participants with absolute Z scores greater than 3 for CAR (T1: $n = 0$; T3: $n = 2$) and AUCg (T1: $n = 2$; T3: $n = 1$) were excluded from analyses of those variables. These participants are included in the broader description of participants as they were not excluded from all analyses.

Procedure

The larger study, from which the data in this study were drawn, was independently approved by Institutional Review Boards (IRB) at Northwestern and UCLA. Each study site maintained compliance with continuing reviews for study approval. Participants attended their respective test to complete the LSI and DAS at T1 and within approximately a month participants' T1 cortisol samples were taken. Approximately 223 days (*SD* = 74.6) later, at T2, participants completed the AMT. Approximately 1273 days (3.5 years; *SD* = 724.3) after this, participants completed their T3 cortisol samples and within the same month participants again completed the LSI and DAS.

Statistical procedure

The sem() function of the lavaan package in R was used to examine the linear associations between variables. The first mediation models examined whether cortisol levels at T1 were indirectly associated with cortisol levels at T3 through individual differences in positive memory specificity. These models included indirect pathways from T1 cortisol to T3 cortisol through T2 positive memory specificity. In order to replicate the analyses of Askelund et al. (2019) memory specificity was first operationalised in terms of the ratio score for memories retrieved for positive cues. Two models were then computed: the first operationalised cortisol levels in terms of the Cortisol Awakening Response and the second used the Area Under the Curve. We then repeated these two models but instead used the more typical index of the raw number of specific memories that were retrieved for positive cues. Bootstrapped standard errors were used in these mediation models with 5000 bootstrap samples.

Subsequent linear regression models, again using the sem() function but with the indirect pathways removed, were then used to explore the direct pathways in greater detail. These models accounted for violations of normality using a robust maximum likelihood estimator and they accounted for missing data using full information maximum likelihood (FIML). Spearman's rank correlations with bootstrap CIs are also given. Significant

associations between specificity and AUCg were followed up with spearman's rank correlations (given the skew that is common in cortisol data) to explore whether the association between these variables were driven by particular timepoints.

As in Askelund et al. (2019), sex was included as a covariate in each model. Models predicting specificity at T2 included life stress and negative cognitions measured at T1 as covariates. Models predicting T3 cortisol values included T1 cortisol as a covariate as well as life stress and negative cognitions measured at both T1 and T3.

The analysis scripts, data and final mediation models (and their associated parameter estimates) are available online (https://osf.io/jef2g/).

Results

Specific to categorical ratio score

Cortisol Awakening Response

CAR at T1 was not significantly associated with the positive memory specificity ratio score at T2, $b = 0.414$, $SE = 0.926$, $Z = 0.447$, $P = .655$, and the specificity score at T2 was not significantly associated with CAR at T3, $b = 0.003$, $SE = 0.008$, $Z = 0.429$, $P = .668$. The direct pathway from T1 CAR to T3 CAR was significant, $b = 0.210$, $SE = 0.064$, $Z = 3.269$, $P = .001$, but the indirect pathway from T1 cortisol to T3 cortisol through T2 specificity was not significant, $b = 0.001$, $SE = 0.009$, $Z = 0.156$, $P = .876$. The only other significant association within the model was between greater life stress at T3 and lower T3 CAR, $b = -0.028$, $SE = 0.011$, $Z = -2.425$, $P = .015$, whereas all other associations were not significant (smallest $p = .266$ for sex on T2 memory specificity).

Area Under the Curve with respect to ground

AUCg at T1 was not significantly associated with the positive memory specificity ratio score at T2, $b = -0.197$, $SE = 0.182$, $Z = -1.078$, $P = .281$. The ratio score at T2 was not significantly associated with AUCg at T3, $b = 0.067$, $SE = 0.042$, $Z = 1.595$, $P = .111$. In this

model, the direct pathway from T1 AUCg to T3 AUCg was not significant, *b* = -0.005, *SE* = 0.073, *Z* = -0.064, *P* = .949, and the indirect pathway from T1 AUCg to T3 AUCg through T2 positive specificity was not significant, *b* = -0.013, *SE* = 0.016, *Z* = -0.799, *P* = .424. Greater life stress at T3 was associated with lower T3 AUCg, *b* = -0.187, *SE* = 0.070, *Z* = -2.687, *P* = .007. Sex was also a significant predictor of T3 AUCg, *b* = 0.859, *SE* = 0.211, *Z* = 4.076, *P* < .001. There were no other significant effects (smallest *p* = .056 for DAS score at T3 on T3 AUCg).

Raw specific memory score

Cortisol Awakening Response

CAR at T1 was not significantly associated with number of specific memories recalled for positive cues at T2, *b* = 0.162, *SE* = 0.542, *Z* = 0.298, *P* = .766, and the specificity score at T2 was not significantly associated with CAR at T3, *b* = 0.001, *SE* = 0.011, *Z* = 0.093, *P* = .926. The direct pathway from T1 CAR to T3 CAR was significant, *b* = 0.211, *SE* = 0.064, *Z* = 3.266, *P* = .001, but the indirect pathway from T1 cortisol to T3 cortisol through T2 specificity was not significant, *b* = 0.000, *SE* = 0.006, *Z* = 0.026 *P* = .979. The only other significant association within this model was between T3 life stress and T3 CAR, *b* = -0.028, *SE* = 0.011, *Z* = -2.502, *P* = .012, whereas all other associations were not significant (smallest *p* = .270 for sex on T3 CAR).

Area Under the Curve with respect to ground

Higher AUCg at T1 was not significantly associated with the raw number of specific memories recalled for positive cues at T2, *b* = -0.134, *SE* = 0.124, *Z* = -1.081, *P* = .280. However, the number of specific memories retrieved for positive cues at T2 was significantly associated with higher AUCg at T3, *b* = 0.132, *SE* = 0.062, *Z* = 2.117, *P* = .034. In this model, the direct pathway from T1 AUCg to T3 AUCg was not significant, *b* = 0.004, *SE* = 0.074, *Z* = 0.050, *P* = .960, and the indirect pathway from T1 AUCg to T3 AUCg through T2

positive specificity was not significant, b = -0.018, SE = 0.019, Z = -0.910, P = .363. Life stress at T3 was associated with T3 AUC, b = -0.182, SE = 0.069, Z = -2.635, P = .008. Sex was also a significant predictor of AUCg at T3, b = 0.822, SE = 0.208, Z = 3.950, P < .001. There were no other significant effects (smallest p = .058 for LSI score at T1 on T3 AUCg).

Positive memory specificity and subsequent differences in daily cortisol

Subsequent analyses focused on the association between the memory specificity at T2 and AUCg measured at T3 to explore this association in more detail. In each of the subsequent regression models, the same covariates as in the previous models were considered (sex, self-related negative cognitions and life stress measured at T1 and T3) and T1 AUCg was also included as a covariate.

Positive memory specificity

To hone in on the association between T2 memory specificity and T3 AUCg, the pathway from T1 AUCg to T2 specificity and the indirect pathway from T1 AUCg to T3 AUCg were removed. Higher positive memory specificity at T2 was associated with higher AUCg at T3, b = 0.132, SE = 0.058, Z = 2.267, P = .023. As in the previous models, life stress at T3, b = -0.182, SE = 0.069, Z = -2.760, P = .006, and sex, b = 0.822, SE = 0.196, Z = 4.186, P < .001, were significant predictors of T3 AUCg. Greater life stress at T1 also predicted lower T3 AUCg, b = -0.115, SE = 0.057, Z = -2.008, P = .045. Neither DAS at T1, b = 0.000, SE = 0.003, Z = 0.069, P = .945, nor at T3, b = -0.005, SE = 0.196, Z = -1.721, P = .085, were significant predictors. This model explained 21.3% of the variance in T3 AUCg. See Figure 1 for a scatterplot of the association between positive memory retrieval and T3 AUCg.

For additional clarification of the previous null effects from the path models, this model was repeated first replacing the T3 AUCg outcome variable with T3 CAR scores. Two additional models also tested whether the positive ratio score predicted T3 AUCg and T3 CAR scores. None of these three models showed a significant association between the

specificity predictor and the cortisol outcome variable (smallest p = .156 for the association between the positive ratio score and T3 AUCg).

Spearman's rank correlations between specificity and cortisol values were significant for cortisol sampled in the afternoon (8 hours post-waking), ρ = .240, P = .005, 95% CI[0.092, 0.404], and evening (12 hours post-waking), ρ = .144, P = .097, 95% CI[-0.023, 0.304]. Correlations with samples taken immediately after waking, ρ = -0.047, P = .586, 95% CI[-0.232, 0.132], 40 minutes post-waking, ρ = 0.079, P = .366, 95% CI[-0.078, 0.240], three hours post-waking, ρ = 0.116, P = .182, 95% CI[-0.054, 0.278], and bedtime, ρ = 0.011, P = .904, 95% CI[-0.163, 0.169], did not approach significance.

Positive and negative memory specificity

Additional models explored whether the association between specificity at T2 and T3 AUCg was unique to positive specific memories or also apparent for negative specific memories. When this model was repeated but the number of specific memories for positive cues and the number of specific memories for negative cues were both included as predictors, only the number of specific memories recalled for positive cues was a significant predictor, b = 0.172, SE = 0.072, Z = 2.398, P = 0.016. The number of specific memories recalled for negative cues was not a significant predictor of AUCg scores at T3, b = -0.070, SE = 0.081, Z = -0.858, P = .391. This model predicted 21.7% of the variance in T3 AUCg.

Categorical and specific memories

To confirm that the effect was unique to specific memories and was not present for the number of categorical memories, the above models were repeated but the number of categorical memories and specific memories retrieved for positive cues at T2 were both included as predictors. As in the earlier models, the number of specific memories retrieved for positive cues at T2 was a significant predictor of T3 AUCg, b = 0.172, SE = 0.081, Z = 2.118, P = 0.034, whereas the number of categorical memories retrieved for positive cues at

T2 was not a significant predictor of T3 AUCg, $b = 0.074$, $SE = 0.086$, $Z = 0.867$, $P = .386$. This model explained 21.6% of the variance in T3 AUCg.

Discussion

The present investigation examined whether cortisol levels, operationalized as the awakening response and total daily cortisol, predicted subsequent recall of memories of specific autobiographical events and the extent to which memory differences were in turn associated with subsequent cortisol levels. Unlike previous research (Askelund et al., 2019), we found no evidence that recall of specific autobiographical memories cued by positive words was longitudinally associated with subsequent cortisol awakening response, nor did the cortisol awakening response predict subsequent autobiographical memory specificity. Similarly, AUCg for total daily cortisol did not predict subsequent autobiographical memory specificity. However, the recall of more specific autobiographical memories cued by positive words was positively associated with total daily cortisol four years later. This association was present despite the lengthy interval between assessment points and whilst also accounting for life stressors that occurred near to cortisol sampling, individual differences in negative self-related cognitions and sex.

The findings partially replicate those of Askelund et al. (2019) in so far as it was only the number of specific autobiographical memories cued by positive words that was related to cortisol, and not the number of specific autobiographical memories cued by negative words. However, whereas Askelund et al. (2019) found that the ratio of specific positively cued memories was negatively correlated with morning cortisol levels, we observed a positive association between the number of specific positively cued memories and daily cortisol levels. Correlations with specific sample points suggested that this association was present for samples taken in the afternoon and evening (eight and twelve hours post-awakening). Similarly, in their cross-sectional study, Barnhofer et al. (2005) found a positive correlation

between increases in afternoon cortisol and the number of specific autobiographical memories that participants retrieved, although cued by neutral cues.

Barnhofer et al. (2005) attributed their findings to the inverted U-shaped curve that typically characterizes the association between cortisol and general memory performance. In particular, cortisol is associated with improved recall when there is optimal cortisol occupation of mineralo- and gluco- corticoid receptors but recall is impaired when there is too much or too little cortisol (De Kloet et al., 1999). Their conceptualization explains the association between cortisol levels and subsequent recall abilities, but the present investigation suggests that the recall of positive specific autobiographical memories is positively associated with subsequent cortisol levels. The driving mechanisms of this unexpected positive association cannot be ascertained from the current design. However, it is of note that although cortisol is typically characterized as a stress hormone, cortisol is also associated with a number of adaptive functions and that higher cortisol levels are neither good nor bad (Hoyt et al., 2016). In particular, cortisol levels can rise in anticipation of positive, active experiences (Carré et al., 2006) and increases in cortisol levels have been associated with an "energy-boosting" effect of increases in activity and alertness (Hoyt et al., 2016). As such, the retrieval of more specific positive autobiographical memories may have a motivational effect that is observed in higher cortisol levels. People who retrieve more of these memories may be better able to use them to simulate and plan for future positive experiences, and therefore be more likely to seek out such experiences which is associated with higher levels of cortisol. Indeed, autobiographical memory and event prospection are strongly associated with one another in healthy populations (Barry, Hallford, et al., 2019). Such an effect would support the suggestion that the retrieval of positive specific autobiographical memories can play an important role in behavioural activation within the context of cognitive-behavioural interventions (Hitchcock et al., 2019). Future investigations

must now quantify the extent to which retrieval of positive memories is associated with engagement with positive experiences and the extent to which this is associated with higher cortisol levels.

There are several regions of the brain that are similarly implicated in the retrieval and re-experiencing of autobiographical memories and Hypothalamic-Pituitary-Adrenal (HPA) axis functioning, and which might explain the effects observed here. In particular, the retrieval of positive autobiographical information has been associated with enhanced amygdala activity, compared to negative information (Markowitsch et al., 2003), and activity in the amygdala has elsewhere been associated with enhanced HPA activity (Jankord & Herman, 2008). Also, currently depressed people have shown particular difficulty recalling positive specific autobiographical memories compared to diagnoses-free control participants and this difficulty has in turn been associated with hypoactivity in the amygdala and reduced connectivity with other key regions of the brain involved in salience and self-referential processing (Young et al., 2016). Participants in a randomized controlled trial involving real-time neurofeedback who were able to increase the activity of their amygdala during retrieval of positive autobiographical memories, were better able to retrieve such memories and also showed reduced depression symptoms post-treatment, relative to a sham control group (Young et al., 2017). Future investigations could explore whether structural and functional differences in the amygdala explain the positive association between retrieval of positive specific autobiographical memories and cortisol.

The observed association between autobiographical memory and cortisol was only found for the number of positively cued specific memories that were recalled and was not evident for categorical memories, and hence the ratio score for autobiographical memory specificity was not significantly associated with cortisol. To our knowledge, there is no precedent for such a ratio score within the literature other than Askelund et al. (2019).

Psychometric analyses suggest that individual differences in autobiographical memory specificity are best operationalised as either the number of specific memories retrieved or the proportion of specific memories retrieved relative to the number of cues given (Griffith, Kleim, et al., 2012; Griffith, Sumner, et al., 2012; Heron et al., 2012; Takano et al., 2017). Future studies in this area should continue to operationalise specificity in line with this established precedent so that studies regarding the association between cortisol dysregulation and specificity can align with broader evidence base regarding autobiographical memory specificity.

The present investigation is not without limitations. First, we were not able to include IQ as a covariate even though prior research has indicated its association with cortisol (Askelund et al., 2019). Second, our measure of cortisol awakening response (changes in cortisol from immediately after awakening to 40 minutes later) did not capture peak responses that occur up to 45 minutes after awakening (Stalder et al., 2016). Future investigations should preferably use five morning sample points (e.g., awakening and 15, 30, 45 and 60 mins post-awakening) so that the CAR can be more accurately classified for each individual.

Third, the cortisol sampling points included in this analysis were asymmetrical with regards to the time between them and our measure of autobiographical memory specificity and there was substantial variability between participants in the timing of each assessment point. There was approximately seven months between T1 and T2 and, on average, four years between T2 and T3. This asymmetry, variability, and the fact that cortisol and specificity were not sampled at any of the same timepoints, reflects the nature of the broader study design from which the present data were sampled. This may also explain the null findings in the mediational models. It remains possible that although autobiographical memory specificity does not mediate the longitudinal association between cortisol levels, that cortisol

levels nonetheless do mediate the longitudinal association between autobiographical memory specificity measured at separate timepoints. Nevertheless, it is remarkable that cortisol at T1 was not associated with specificity measured seven months later but there was an association between specificity measured at T2 and subsequent cortisol levels approximately four years later. Future investigations must include measures of autobiographical memory and cortisol at each of several timepoints that are symmetrically timed and where there is minimal variability between data collection times. Subsequent investigations could also manipulate cortisol using an acute social stressor or dexamethasone, in order to more directly test whether differences in cortisol can influence autobiographical memory retrieval or whether retrieval of positive specific autobiographical memories can influence cortisol output even following suppression.

Fourth, the valence of memories themselves was not analyzed. Although the memories that participants retrieve in the AMT typically share the same valence as the cue that preceded them (Barry, Del Rey, et al., 2018), coding of the valence of memories would allow examination of valence-specific associations between cortisol and memory specificity. Future studies should employ a more symmetrical design with cortisol and specificity sampled at the same several timepoints and where memories are additionally coded for valence.

Finally, the present data come from a broader investigation that oversampled participants thought to be at risk of psychopathology given their relatively high scores in a measure of neuroticism (Zinbarg et al., 2010). Askelund et al. (2019) had a similar criterion for recruiting participants recruitment. As such, the effects observed in both investigations may be a function of risk for psychopathology, especially since poor autobiographical memory specificity has been associated with early exposure to stressful life events (Barry, Lenaert, et al., 2018; Ono et al., 2015) and exposure to stress is a known contributor to

cortisol dysregulation (Stroud et al., 2019; Strüber et al., 2014). However, it is of note that the analysis presented here accounted for individual differences in chronic life stressors. Also, simulations indicate that such oversampling is only likely to have a nominal effect on observed effect sizes (Hauner et al., 2014). Relatedly, our study quantified life stress in terms of interpersonal stress. This is in line with other studies in this sample that showed an association between this form of stress and autobiographical memory (Barry, Vinograd, et al., 2019). However, it remains possible that the inclusion of a measure of other kinds of life stress (e.g., work-related stress) or negative life events may have elicited different associations with autobiographical memory and cortisol.

In overview, the present investigation explored the longitudinal associations between cortisol, operationalised in terms of cortisol awakening response and AUCg for daily cortisol, and autobiographical memory specificity. The retrieval of more specific autobiographical memories cued by positive words was prospectively associated with higher daily cortisol levels four years later. This effect was independent of recent life stressors, negative self-related cognitions and sex. These effects were not present for the number of specific autobiographical memories retrieved for negative cues. There was no evidence for an association between autobiographical memory specificity and cortisol awakening response. Future investigations that quantify cortisol and autobiographical memory specificity at each of several symmetrical timepoints and which include multiple morning cortisol samples, rather than the two used here, are needed in order to further examine whether these effects replicate in a more robust longitudinal design. Future studies must also examine the extent to which the effects observed here are explained by motivation for and engagement with positive experiences.

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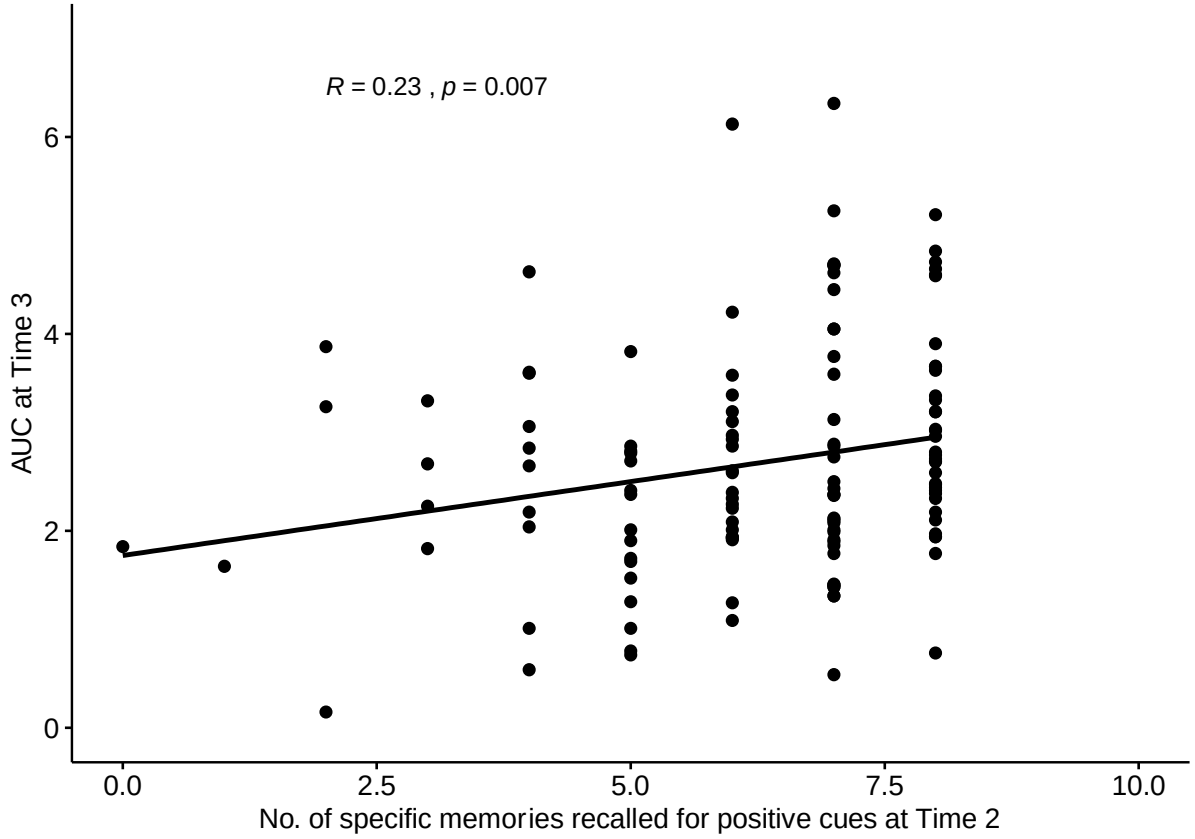
Table 1. Regression model predicting T3 Area Under the Curve with respect to ground

	b	*SE*	*Z*	*P*
Spec. Pos. T2	0.132	0.058	2.267	**0.023**
AUCg T1	0.004	0.069	0.054	0.957
LSI T1	-0.115	0.057	-2.008	**0.045**
LSI T3	-0.182	0.066	-2.760	**0.006**
DAS T1	0.000	0.003	0.069	0.945
DAS T3	-0.005	0.003	-1.721	0.085
Sex	0.822	0.196	4.186	< **.001**

Note. Regression model predicting average cortisol Area Under the Curve with respect to ground measured at Time 3. Spec. Pos. T2: The number of specific memories recalled for positive cues at Time 2. AUCg T1: Cortisol Area Under the Curve with respect to ground at Time 1. LSI T1/T3s: Life Stress Interview score at Time 1 and Time 3. DAS T1/T3: Dysfunctional Attitudes Scale total scores at Time 1 and Time 3. Sex: 0 = Male; 1 = Female.

Figure 1. Scatter plot



Note. Scatter plot of the number of specific memories recalled for positive cues at Time 2 against average cortisol Area Under the Curve with respect to ground (AUC) at Time 3. Spearman's rho correlation coefficient is also given.

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