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Inducing Death Thoughts Reduces the Cortisol Response to Psychosocial Stress Similar to the Effects of Early-life Adversity: A Life-history Perspective

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Abstract

Purpose Early-life adversity (ELA) affects health by altering the hypothalamic–pituitary–adrenal (HPA) axis. Most studies show that ELA blunts HPA responsivity while others show the opposite. To explain this inconsistency, researchers investigate factors that alter associations between ELA and HPA responsivity. One factor could be conditions that participants encounter before exposure to stressors. Life-history theory suggests ELA alters HPA function by signalling high mortality. Similarly, death thoughts signal acute mortality. Research suggests that thinking about death induces behaviors typical of ELA subjects. We therefore tested whether death thoughts before acute stress mimics the effects of ELA on HPA responsivity.

Methods One hundred twenty eight healthy young men were classified as high or low ELA based on retrospective self-report, and then primed with death thoughts (experimental group) or completed neutral questionnaires (control group). They then underwent a psychosocial stress task. Salivary cortisol was sampled repeatedly to assess HPA responsivity to stress.

Results In the control group, higher ELA correlated with lower cortisol responsivity. In the experimental group, subjects with high ELA did not show altered cortisol responsivity, but low ELA participants displayed significantly blunted responsivity in response to death thoughts. Thus, low ELA participants primed with death thoughts resembled high ELA participants not exposed to death thoughts.

Conclusion Our findings suggest that subtle death cues present in the testing environment may confound associations between ELA and HPA function and should be controlled for in future studies. We discuss how life-history theory could explain how both long-term (ELA) and acute (mortality salience) experiences alter HPA function.

Keywords Early-life adversity · Hypothalamic–pituitary–adrenal axis · Cortisol · Stress · Life-history theory · Mortality salience

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Introduction

Early-life adversity (ELA) is exposure to stressful conditions during childhood (e.g. abuse, neglect, or parental loss). ELA can have profound effects on health and development (De Bellis & Zisk, 2014; Pechtel & Pizzagalli, 2011; Smith & Pollak, 2020), and is associated with early sexual debut (Chisholm et al., 2005; Kogan et al., 2015; Nettle, 2010; Thomas et al., 2023), aggression (Campbell et al., 2010; Madole et al., 2020), impulsivity (Lovallo, 2013; Sanchez & Bangasser, 2022), risky sex (Alley & Diamond, 2021; James et al., 2012; Kogan et al., 2015; Thomas et al., 2023) and other risky behaviors (Kogan et al., 2015; Simpson et al., 2012; Wolitzky-Taylor et al., 2017; Wood et al., 2021). ELA affects health and behavior in part by altering the hypothalamic–pituitary–adrenal (HPA) axis, a neuroendocrine system that helps vertebrates adapt to changing environments. The HPA axis responds to acute stressors by increasing levels of glucocorticoids (cortisol in humans) (Charmandari et al., 2005). Human and animal research shows ELA affects HPA responsivity to stressors (Juruena et al., 2021; Lupien et al., 2009; Tarullo & Gunnar, 2006), however, the direction of this effect varies between studies. According to one meta-analysis (Bunea et al., 2017), in most human studies, high ELA individuals exhibit blunted HPA responsivity relative to those reared in supportive conditions. Fewer studies associate ELA with greater (not blunted) responsivity (Hackman et al., 2013; Heim, 2000; Korczak et al., 2016; Pesonen et al., 2010; Pruessner et al., 2004). Some find no effect of ELA on responsivity (Maria et al., 2010; Murali & Chen, 2005; Steudte-Schmiedgen et al., 2015).

The effect of ELA on HPA function may vary depending on genes (Buchmann et al., 2014; Sumner et al., 2014), temperament (Davies et al., 2011), as well as timing (Bosch et al., 2012; Raymond et al., 2021), type (Carpenter et al., 2011; Kuhlman et al., 2015), and severity of ELA (Gunnar et al., 2009; Schwartz et al., 2023). Harsh rearing environments and unstable/unpredictable environments also exert distinct effects on the stress response systems (Ellis et al., 2022). Sex may also influence how ELA affects the HPA axis. As reviewed by Bath (2020), there are documented sex differences in the prevalence of stress-related disorders, as well as the impact of ELA on brain development. Males and females may experience qualitatively distinct kinds of ELA, for instance, childhood sexual abuse is more prevalent among girls (Walker et al., 2004). Some studies have observed a more pronounced affect of ELA on HPA axis function in females compared to males (DeSantis et al., 2011).

These factors may explain some differences between studies. Nonetheless, even when these factors are constant, findings still vary inexplicably (e.g., Gunnar et al., 2009; McLaughlin et al., 2015), suggesting that other factors may also moderate effects of ELA on HPA function. For instance, recent events may confound the effect of ELA on HPA responsivity, especially since the cortisol stress response is usually assessed once in the laboratory. One study (Engert et al., 2010) exposed adults with low or high ELA to a psychosocial stressor three times. Initially, high ELA individuals showed blunted HPA responsivity compared to

low ELA participants. Over time, responsivity stayed the same in high ELA participants, but became blunted in low ELA participants such that they no longer differed from the high ELA group. This suggests that recent events may temporarily mask differences in HPA responsivity between low ELA and high ELA participants.

In addition to constitution and experience, there are evolutionary theories for why stressful events might alter HPA function – these suggest that associations between ELA and HPA responsivity may also be obfuscated by transient thoughts and considerations, such as contemplating one's own death. Life-history theory, a mature branch of evolutionary biology, is such a theory and provides a possible rational for our a priori hypothesis about ELA, death thoughts and HPA responsivity. Authors using this theoretical perspective (e.g., Del Giudice et al., 2011) emphasize that the HPA axis, like other physiological systems, evolved ultimately to maximize reproductive fitness, not to prolong survival. While adversity may have harmful effects, from an evolutionary perspective, adversity can also serve as a cue to optimize reproductive strategies in harsh, or unpredictable, environments (Belsky et al., 1991; Chisholm, 1996; Chisholm et al., 1993; Del Giudice et al., 2011; Ellis & Del Giudice, 2019; Ellis et al., 2017). Life-history theory claims that when organisms encounter cues signaling high premature mortality, such as maternal neglect or other forms of adversity, or when they encounter unpredictable environments, they evolved to adapt by changing life-history strategy (Chisholm, 1996; Chisholm et al., 1993; Del Giudice et al., 2011; Ellis & Del Giudice, 2019; Ellis et al., 2017). Life-history strategy is one of the main concepts in life-history theory and is the way an organism divides finite time and energy between reproduction, growth and survival (Hill & Kaplan, 1999; Stearns, 1992; Yang et al., 2022). Life-history strategy varies on a continuum from slow to fast. As Table 1 summaries, slow and fast life-history strategies emerge and are adapted for different environments and are associated with distinct patterns of behavior and HPA function.

Organisms with a fast life-history strategy prioritize reproduction over long-term growth/survival. Fast life-history strategy manifests as early reproduction, sexual promiscuity, impulsivity, and risky behavior, while slow life-history strategy does the opposite, prioritizing long-term growth/survival over reproduction (Figueredo et al., 2007; Kaplan & Gangestad, 2005; Yang et al., 2022). Different life-history strategies are adaptive in different environments (Ellis et al., 2009; Yang et al., 2022). Fast is optimal in harsh conditions where premature mortality is high because organisms are more likely to reproduce even if they die prematurely. In contrast, slow life-history strategy is optimal in supportive environments where mortality is low. Since mortality fluctuated rapidly throughout human evolution, life-history theory further claims that humans evolved to detect environmental cues that predict local mortality rates, and to adjust their life-history strategy accordingly to maximize reproductive fitness (Del Giudice et al., 2011; Ellis & Del Giudice, 2019; Kuzawa & Bragg, 2012; Yang et al., 2022). When premature mortality is unpredictable, individuals respond accordingly by shifting to a fast life-history strategy, or their life-history strategy becomes less responsive to environmental cues (Ellis et al., 2009, 2022). Since ELA tends to occur in environments with high mortality, or in unstable environments where

Table 1 Comparison of fast and slow life-history strategy

		Fast life-history strategy	Slow life-history strategy
Behavior	Prioritizes…	Reproduction over long-term growth/survival	Prioritizes long-term growth/survival over reproduction
	Reproductive timing	Precocious reproduction	Delayed reproduction
	Mating	Sexually promiscuous	Monogamous pair-bonds
	Offspring	Many	Few
	Risky behavior	More risky behavior	Avoids risk
Physiology	HPA responsivity	Low responsivity (blunted)	High responsivity
Ecological determinants	Specialized for…	Hash environments (*high* mortality)	Safe, supportive environments (*low* mortality)
	Triggered by…	Organism detects ecological cues signaling *high* mortality	Organism detects ecological cues signaling *low* mortality
	Chronic early-life triggers	High early-life adversity, other cues predicting *high* mortality	Low early-life adversity, other cues predicting *low* mortality
	Acute situational triggers (hypothetical)	Death thoughts, other cues predicting high mortality	Cues predicting *low* mortality

premature mortality is unpredictable, several authors (Chisholm et al., 2005; Csathó & Birkás, 2018; Del Giudice et al., 2011; Ellis & Del Giudice, 2019; Nettle, 2010) argue that this could explain why high ELA participants show signs of fast life-history strategy (e.g., sexual promiscuity and risky behavior) while individuals reared in supportive conditions exhibit slow life-history strategy (e.g., delayed reproduction, risk avoidance, monogamy).

It is important to note however that some studies report that individuals with high mate value, which typically emerges in individuals with low ELA (Antfolk & Sjölund, 2018), can exhibit certain sexual characteristics typically associated with a fast life-history, such as having more partners (Csajbók et al., 2023). Elevated attraction to masculine physical features among heterosexual women is another sexual preference associated with a faster life-history strategy (Niu & Zheng, 2020) that was found in women with higher mate value (Garza & Byrd-Craven, 2021; Reeve et al., 2017).

The HPA axis helps implement life-history strategy by controlling growth, reproduction, and energy balance (Carere et al., 2010; Crespi et al., 2013; Del Giudice et al., 2011; Réale et al., 2010; Wingfield & Sapolsky, 2003; Zakreski et al., 2018). While the association between life-history strategy and HPA axis activity is complex and may vary according to sex or species (Crespi et al., 2013; Duckworth et al., 2023), in numerous species, fast life-history strategy is generally associated with blunted HPA responsivity while slow life-history strategy is facilitated by greater responsivity (Carere et al., 2010; Réale et al., 2010). Theoretically, events signaling high mortality blunt HPA responsivity, facilitating faster life-history strategy, while events signaling low mortality increase responsivity, facilitating slow life-history strategy. This is consistent with fact that in most human studies, individuals exposed to ELA show blunted HPA responsivity to stress (Bunea et al., 2017).

Mortality cues are events that reliably correlate with premature mortality (Nettle et al., 2013). Death thoughts, like ELA, correlate with actual mortality (Abdel-Khalek & Lester, 2003; Alaimo et al., 2002; Cox et al., 2012; Robertson et al., 2006; Shakil & Yousaf, 2015), which may be why death thoughts induce effects similar to ELA. An interesting question to examine is whether inducing death thoughts is sufficient to change life-history strategy. With regard to the HPA axis, Byrd-Craven et al. (2015) showed that inducing death thoughts shortly before a psychosocial stressor led to blunted HPA responsivity—the more commonly observed effect of ELA in humans (Bunea et al., 2017). However, these authors did not interpret their findings with regard to life-history theory, nor did they assess ELA. Complementary behavioral experiments suggest that inducing acute death thoughts in the laboratory, like ELA, evokes fast life-history characteristics such as increased risk taking and short-term mating orientation (Fritsche et al., 2007; Gillath et al., 2011; Hart et al., 2010; Hirschberger et al., 2002a, 2002b; Hirschberger et al., 2002a, 2002b; Kelley & Schmeichel, 2015; Lam et al., 2009; Mathews & Sear, 2008; Routledge et al., 2004; Silveira et al., 2014; Taubman Ben-Ari, 2004; Taubman Ben-Ari et al., 1999; Wisman & Goldenberg, 2005). Thus, here we hypothesize that ELA and death thoughts have similar effects on HPA function and life-history strategy, however we speculate that the effects of death thoughts are momentary, whereas the effects of ELA are chronic.

Research also shows that death reminders temporarily interact with ELA to affect various life-history strategy related behaviors (Griskevicius et al., 2011a, 2011b; Griskevicius et al., 2011a, 2011b). While these studies create precedence for combining acute death thoughts with varying ELA, no study has investigated how death thoughts affect HPA responsivity in interaction with ELA. If death thoughts moderate effects of ELA on HPA responsivity, this may partly explain why studies examining effects of ELA on HPA function yield mixed results. Given our theoretical rationale, we hypothesized a priori that participants with high ELA would show blunted HPA responsivity to an acute stressor. Since death thoughts signal premature mortality, we further hypothesized that acute death thoughts would also blunt HPA responsivity in low ELA individuals. We also hypothesized that death thoughts would not affect HPA responsivity in high ELA participants since they should already present with blunted HPA responsivity.

To test these hypotheses, we recruited healthy young men who were low or high on self-reported ELA and assigned them to death thoughts or a no death thoughts control condition before exposing them to a psychosocial stressor. The main dependent variable was salivary cortisol output as a marker of HPA responsivity to the stressor. We also assessed subjective stress.

Method

Participants (N = 128) from two different experimental protocols as part of a larger research protocol were pooled together to increase statistical power. Study 1 (N = 65) and Study 2 (N = 63) tested different samples drawn from the same population: healthy young men with naturally occurring variations in ELA. In both studies, men were classified as low or high ELA using retrospective self-report questionnaires. To manipulate death thought accessibility, men were exposed to either a mortality cue (see *Death Thought Manipulation* below) or a control condition before a psychosocial stressor. Study 1 participants performed the Trier Social Stress Test (TSST; Kirschbaum et al., 1993). Study 2 used the Montreal Imaging Stress Test (MIST; Dedovic et al., 2005). Study 1 and 2 used different stress tests because, as part of a separate investigation, Study 2 involved magnetic resonance imaging (MRI), and the MIST is designed to induce stress in an MRI scanner. In both studies, cortisol and subjective stress were sampled repeatedly. Both studies were approved by the Faculty of Medicine IRB of McGill University and all participants gave written informed consent.

Sample

We recruited 128 participants in Montreal by advertising on Internet classifieds. Prospective participants first completed an online survey prescreening for inclusion and exclusion criteria. This survey included the Parental Bonding Inventory (Parker et al., 1979) to assess mother-care, and the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) to assess depression. We tested only men to avoid

the confounds of menstrual cycle phase and chemical contraceptives that affect cortisol responsivity (Kirschbaum et al., 1999). Included men were 18 to 35 years old with normal weight (BMI: 18–27). Exclusion criteria were past or current mental illness, prior exposure to a standardized psychosocial stress task, consuming more than three alcoholic beverages per day, using marijuana or tobacco more than twice weekly, or currently taking prescription or other drugs.

Early-life Adversity Classification

ELA was assessed using two retrospective self-report questionnaires— the Parental Bonding Instrument (PBI; Parker et al., 1979) and the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003). The PBI and CTQ demonstrate good validity in healthy adults (Bernstein et al., 2003; Georgieva et al., 2021; Lancaster et al., 2007) and both measures correlate with HPA function (Ali & Pruessner, 2012; Carpenter et al., 2007, 2011; Cook et al., 2012; Engert et al., 2010; Kawai et al., 2017; Kuras et al., 2017; Lai et al., 2020; Pruessner et al., 2004). The PBI measures care and overprotection of both mother and father during the first 16 years of life. We here focused on mother care; participants were classified as high or low on mother-care relative to the test developers' recommended cutoff (Parker & Lipscombe, 1979). The CTQ measures five subscales: physical abuse, emotional abuse, sexual abuse, physical neglect, and emotional neglect. For each subscale, participants were classified as high or low relative to the test developers' recommended cutoff for moderate to severe trauma (Bernstein et al., 2003). We classified participants as high ELA if they were low on mother-care, or high on at least one CTQ subscale. Participants were classified as low ELA if they were high on mother-care and low on all five CTQ subscales.

There are several reasons why we chose to dichotomize ELA rather than use a continuous score. First, statistical simulation studies have found that dichotomizing variables does not increase Type I error unless there is extremely high multicollinearity (Iacobucci et al., 2015). Dichotomous variables can be more robust than continuous predictors when data are skewed or when the independent variable has both linear and nonlinear effects (Farrington & Loeber, 2000; Iacobucci et al., 2015). This advantage is significant for our purposes since Anderson–Darling tests found that CTQ total scores and PBI scores in our sample were significantly skewed even after Box-Cox transformation. Furthermore, several studies suggest the relationship between ELA and HPA function may contain quadratic and other nonlinear components in addition to linear components that, unless corrected for, increase noise (Deer et al., 2021; Ellis et al., 2005; Engert et al., 2010; Gunnar et al., 2009; Hagan et al., 2014). Modeling quadratic and higher-order effects of ELA would help control for these non-linear effects while keeping ELA as a continuous variable. This would, however, greatly increase model complexity and subsequently reduce statistical power, particularly in our situation since we are interested in the interaction between ELA and another variable (death thoughts). Finally, there is a well-established precedent in research for treating ELA as a categorical variable. Many of the studies that used the PBI or CTQ to examine effects of ELA on HPA function

categorized participants as high or low ELA using the recommended cutoffs as we did here (Ali & Pruessner, 2012; Carpenter et al., 2007, 2011; Cook et al., 2012; Kawai et al., 2017; Kuras et al., 2017).

Procedure

Before testing, we told participants to abstain from drugs and alcohol for at least 24 h before testing, and on testing day, to avoid caffeine and physical exertion, but to eat breakfast. To avoid variation due to circadian cortisol rhythms, testing occurred between 12:30 PM – 3:30 PM. The procedure lasted roughly 2.25 h and consisted of habituation, death thought or control condition, psychosocial stress, and recovery as illustrated in Fig. 1.

Habituation

Upon arrival, participants habituated to the laboratory environment for 30 min while completing questionnaires. Baseline cortisol and subjective stress were assessed after habituation.

Death Thought Manipulation

After sampling baseline cortisol and subjective stress, we exposed participants to either a mortality cue or control condition for 10 min. Condition was assigned so that age, BMI, PBI mother-care, CTQ scores, and PHQ-9 depression were balanced between groups.

Mortality Condition We used the Mortality Attitudes Personality Survey (MAPS) (Rosenblatt et al., 1989), the most common method of experimentally inducing death thoughts (Burke et al., 2010). The MAPS instructs participants to write two brief essays. Participants were asked to "briefly describe the emotions that the thought of your own death arouses in you" and to "jot down, as specifically as you can, what you think will happen to you as you physically die and once you are physically dead". We chose this stimulus as a mortality cue because it has been repeatedly shown to acutely induce fast life-history characteristics (e.g. interest in casual sex, risk taking) (Gillath et al., 2011; Hart et al., 2010; Hirschberger et al., 2002a, 2002b; Hirschberger et al., 2002a, 2002b; Kelley & Schmeichel, 2015; Lam et al., 2009; Routledge et al., 2004; Taubman Ben-Ari, 2004; Taubman Ben-Ari et al., 1999; Wisman & Goldenberg, 2005).

Control Condition Participants that did not receive the mortality cue received one of three control stimuli: sleep, dental pain, or non-death related questionnaires, all of which have been used in past experiments manipulating death thought accessibility (Burke et al., 2010). The sleep and dental pain cues were essay questions identical to the MAPS except the word death was replaced with "sleep" and "dental pain", respectively. For instance, the sleep cue asked participants to "briefly describe the

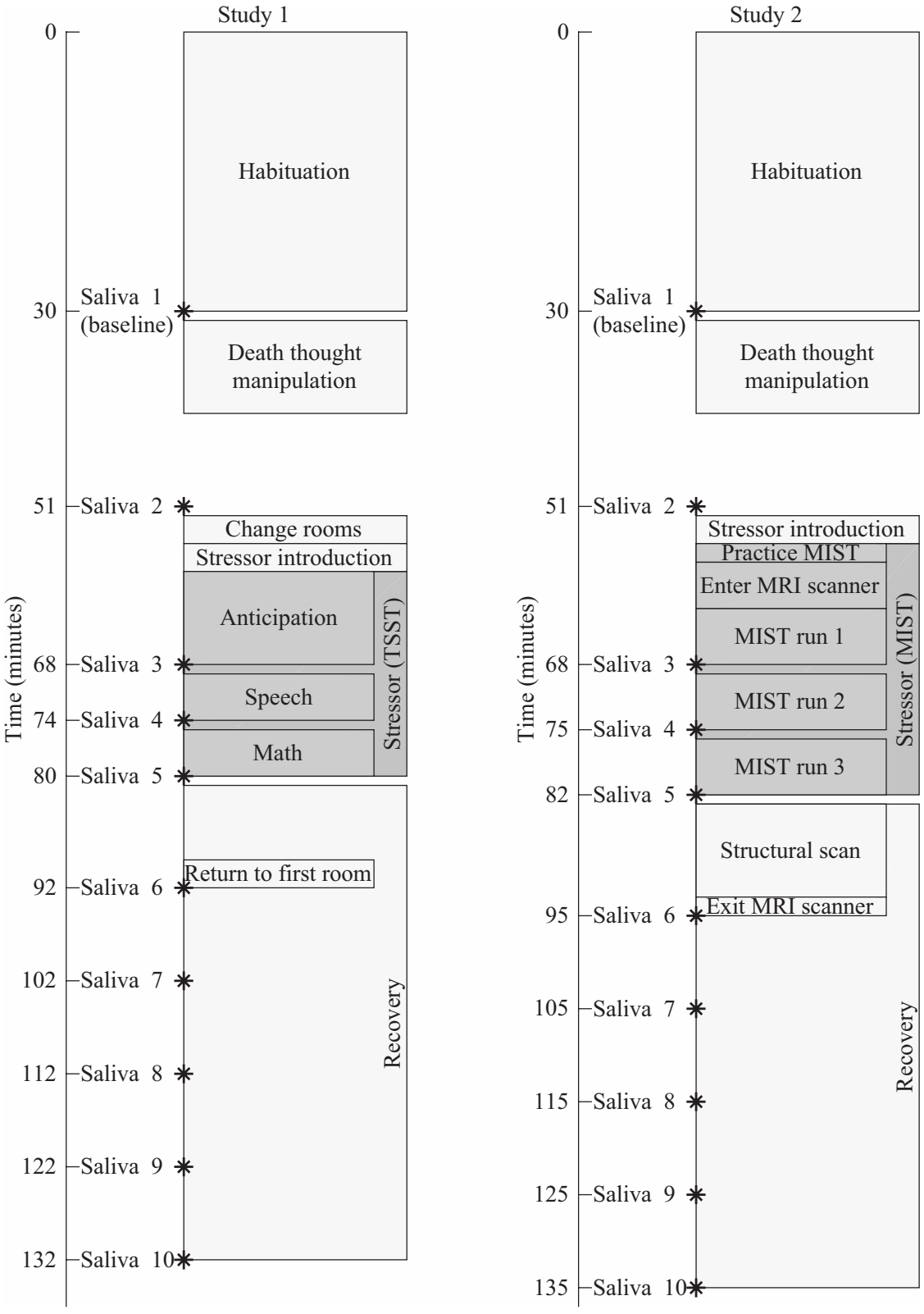

Fig. 1 Salivary cortisol sampled throughout the procedure. Asterisks indicate the time of each saliva sample in minutes relative to participant's arrival for Study 1 and Study 2. Since Study 2 included functional magnetic resonance imaging, saliva samples were taken at slightly different time points relative to Study 1 to accommodate the neuroimaging protocol. In Study 1, momentary subjective stress was measured with each saliva sample. In Study 2, subjective stress was measured with samples 1, 2, 6, 7, 8, 9 and 10 but was not assessed while participants were in the MRI scanner

emotions that the thought of sleeping induces in you", and "jot down, as specifically as you can, what you think happens to you as you fall asleep and once you are sleeping". Participants in the neutral questionnaire condition completed personality questionnaires unrelated to death, sleep, or dental pain. Study 1 control participants completed either the sleep condition or neutral questionnaire condition. Study 2 control participants only completed the dental pain condition. We did not observe any difference in physiological or psychological outcomes between the three control conditions, thus we merged them into one control condition to maximize statistical power.

Psychosocial Stressor

Ten minutes after the death thought manipulation ended, participants were introduced to the stress task. Study 1 used the Trier Social Stress Task (TSST) in a laboratory environment (Kirschbaum et al., 1993). The TSST is used widely in stress research since it elicits robust physiological and psychological responses. The TSST involves anticipation (10 min), public speaking (5 min), and mental arithmetic (5 min). During anticipation, participants prepared an oral presentation for a mock job interview. After anticipation, participants presented their speech and performed mental arithmetic before a panel of two trained confederates of mixed gender.

Study 2 used the Montreal Neuroimaging Stress Test (MIST) in a neuroimaging environment (Dedovic et al., 2005). Like the TSST, the MIST involves motivated performance, cognitive challenge, social evaluation, and uncontrollability, but unlike the TSST, the MIST is compatible with neuroimaging. The MIST is a computer task where participants perform mental arithmetic under time pressure and negative social evaluation. Although participants are urged to do their best, the task covertly adjusts the difficulty level so that participants perform poorly. Participants practiced the MIST for 2 min to habituate to the interface. Once inside the scanner, participants performed the MIST for 18 min (1 min in control mode, 2 min in experimental mode, repeated 6 times). Every 6 min, participants received negative performance feedback from an opposite-sex confederate. There are important differences between the MIST and the TSST; there is no free speech component in the MIST, the subject is not performing before a live audience, and the actual stress lasts much longer. To address these differences, we introduced stressor type as a predictor in our analyses.

Recovery

After the stressor, there was a 1-h recovery period where participants completed questionnaires, colored in coloring books, or read magazines before debriefing.

Salivary Cortisol and Subjective Stress

We collected saliva throughout the experiment using Salivette® collection devices (Sarstedt Inc., Quebec City, Canada). Figure 1 indicates the time of each saliva sample. In Study 1, with each saliva sample, participants rated their current stress levels on a visual analogue scale, placing an "X" on a 10 cm line anchored from

0 (not at all stressed) to 10 (extremely stressed). In Study 2, with each saliva sample (except those taken during the MRI session), momentary subjective stress was assessed using the affect grid (Russell et al., 1989). Participants indicated their current feelings by placing an "X" on a 10 X 10 grid. The grid's horizontal axis is valence (unpleasant vs. pleasant feelings). The vertical axis is arousal (low vs. high). Subjective stress is calculated as arousal minus valence for that time point. Since Study 1 and 2 measured subjective stress on different scales, subjective stress scores were z-transformed within studies. Subjective stress data were missing for two participants. Cortisol levels were measured by time-resolved fluorescence immunoassay with 0.179 nmol/L sensitivity and intra-assay and inter-assay variability below 10% and 12%, respectively (Dressendörfer et al., 1992).

Other Questionnaires

Chronic stress was assessed using the Trier Inventory of Chronic Stress (TICS) (Schulz & Schlotz, 1999). Depression was assessed using the Patient Health Questionnaire (PHQ-9) (Kroenke et al., 2001).

Responsivity to Stress

Cortisol responsivity to stress was calculated with area under the curve with respect to ground (AUC_G; Pruessner et al., 2003). This calculation transforms raw time series to summarize cortisol output. AUC allows for the comparison of cortisol responsivity between individuals, while avoiding some of the problems associated with repeated measures ANOVA (Pruessner et al., 2003). Since we are interested in the effects of death thoughts on cortisol responsivity to the psychosocial stressor, AUC_G calculation was restricted to samples occurring after the death thought manipulation.

Statistical Analysis

Analyses were done with MATLAB 2017 (Mathworks, Inc. Natick, MA, USA). Missing cortisol and subjective stress samples were estimated with linear interpolation. Violations of the normality assumption were checked with Anderson–Darling tests and corrected by Box-Cox transformation. Violations of the equal variance assumption were checked with Brown-Forsynthe tests. First, we performed an ELA (low vs. high) by death thoughts (yes vs. no) by Study (1 vs. 2) chi-squared test of unequal proportions which showed group sizes were not significantly disproportionate $\chi^2[4] = 2.358$, $p = 0.670$. We compared demographic and outcome variables between Studies 1 and 2 to confirm that data were similar enough to merge them. After merging the studies, to control for any differences between studies (e.g. different stress tasks), Study was also included as a categorical predictor.

Before testing our primary hypothesis, to determine whether cortisol levels significantly increased in response to the MIST/TSST we made a repeated measures linear mixed-effects model regressing Box-Cox transformed cortisol onto

$Time^2 + Time + Study + Intercept$, with intercept varying randomly by participant. Time was centered around 15 min after the stress task since cortisol typically peaks around then.

To test our primary hypothesis that death thoughts and ELA interact to affect cortisol responsivity, we set up a general linear model (GLM) with the formula: $AUC_G \sim ELA*Death\ thoughts + ELA + Death\ thoughts + Study + Intercept$. The categorical variables Death thoughts (yes vs. no) and Study (1 vs. 2) were coded so coefficients would sum to 0. Type III sum of squares was specified. A sensitivity power analysis with alpha set to 0.05 and power set to 0.80 was performed with the R "pwr" package (Champely, 2018). The analysis showed that our experimental design, with one degree of freedom in the numerator per term, and 123 residual degrees of freedom is sufficient to detect an effect size greater than $f^2 = 0.064$ which is between small and medium (Cohen, 1988).

Differences in cortisol responsivity may be confounded by other factors such as baseline cortisol, baseline subjective stress, age, BMI, PHQ-9 depression, TICS chronic stress, in addition to positive and negative affect, and subjective stress immediately after the death thought manipulation. For each of these potential confounding variables, we tested separate GLMs, regressing that variable onto the formula: ELA*Death thoughts + ELA + Death thoughts + Study + Intercept.

To supplement our analysis, we examined whether cortisol or standardized subjective stress levels changed after death thoughts (before the stress task). We first performed a mixed design ANOVA on Box-Cox transformed cortisol with death thoughts, ELA, and Study as between-subject factors, and time (sample before vs. sample after death thought manipulation) as the within-subject factor (see Fig. 1 for sample times). We then performed the same ANOVA only with Box-Cox transformed subjective stress as the dependent variable.

Results

Demographic and Baseline Differences Between Groups

Table 2 summarizes sample characteristics. Among low ELA participants (N = 61), 27 were primed with death thoughts and 34 were in the control group. Among high ELA participants (N = 67), 29 were in the death thought condition and 38 were in the control condition. Before testing our primary hypothesis, to assess potential confounds, GLM performed on baseline cortisol and standardized baseline subjective stress revealed no significant main effects or interactions, $F(1,123) < 2.28$, $p > 0.133$, suggesting that basal cortisol and basal standardized subjective stress were comparable across groups. Likewise, additional GLMs showed no significant differences between groups in age, BMI, depression, and chronic stress, though Study 2 had higher CTQ scores and lower mother-care scores.

Table 2 Sample characteristics

Study	Early-life adversity	Death thoughts	N	Age (years)	BMI	TICS chronic stress	PHQ-9 depression	PBI mother-care	CTQ emotional trauma	CTQ physical trauma	CTQ sexual abuse	CTQ total
				M(SD)	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)
1	Low	Yes	12	25.58 (3.92)	22.81 (1.25)	36.25 (16.99)	3.17 (3.13)	33.33 (3.11)	13.08 (2.39)	11.25 (2.05)	5.00 (0.00)	29.33 (3.80)
1	Low	No	20	24.89 (3.83)	22.71 (2.11)	40.00 (12.64)	3.35 (3.31)	31.25(2.29)	13.75(2.77)	11.15 (1.18)	5.05 (0.22)	29.95 (3.35)
1	High	Yes	12	24.10 (4.54)	24.10 (2.76)	46.36 (18.86)	3.17(2.08)	21.92 (6.30)	22.33(7.97)	14.67(5.55)	5.50 (1.17)	42.50 (12.80)
1	High	No	20	24.41(4.78)	22.49 (2.41)	50.87 (12.68)	3.35 (3.45)	24.05 (5.17)	22.45 (7.40)	16.30 (6.14)	6.25 (2.79)	45.05 (12.84)
2	Low	Yes	15	23.65 (2.82)	23.15 (2.44)	39.13 (17.13)	3.93 (3.43)	33.00 (2.17)	13.20 (2.68)	10.87 (0.99)	5.00 (0.00)	29.07 (2.87)
2	Low	No	14	24.56 (3.76)	22.27 (2.29)	36.86 (11.84)	4.07 (3.93)	31.42 (2.77)	13.00 (2.99)	10.86 (1.10)	5.07 (0.27)	28.93 (3.45)
2	High	Yes	17	24.73 (3.77)	22.99 (2.53)	43.35 (14.24)	6.71 (5.69)	16.29 (7.66)	33.50 (9.04)	19.19 (5.97)	6.38(3.16)	57.88 (15.50)
2	High	No	18	24.76(4.26)	23.12(1.75)	46.11(14.42)	9.06(5.86)	18.50(6.84)	29.17(8.59)	18.61(6.87)	5.89(1.64)	53.67(14.45)

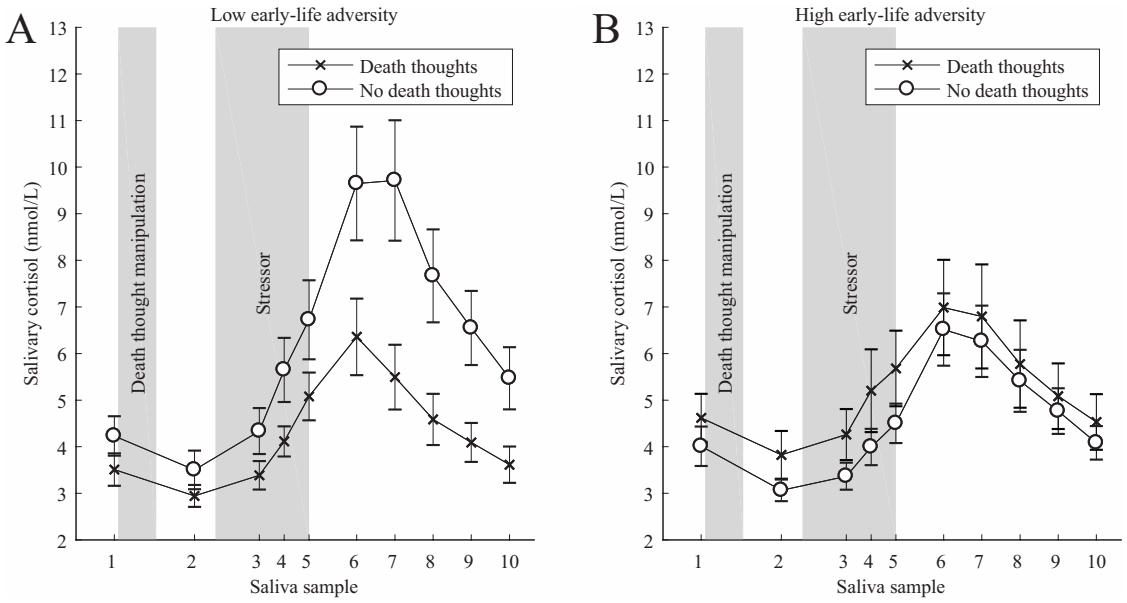

Fig. 2 Change in cortisol levels over time. During the death thought manipulation, participants in the experimental condition ("X" markers) wrote about death, while those in the control condition (open markers) wrote about a non-death related topic. Shortly after the manipulation, participants performed a psychosocial stress task. See Fig. 1 for sample times. Cortisol did not change significantly after death thought induction, however it increased significantly following the psychosocial stressor. Without death thoughts, low ELA participants released more cortisol after the stressor compared to high ELA participants. Inducing death thoughts did not significantly change cortisol responsivity in high ELA participants but blunted responsivity in low ELA participants. Error bars are standard error

Effects of Psychosocial Stress on Cortisol

Figure 2 shows cortisol changing over time. The repeated measures mixed-effects model regressing Box-Cox transformed cortisol onto Time and Time^2 found that cortisol levels significantly increased (Time: $t(1013) = 8.66$, $p < 0.001$, partial $\eta^2 = 0.068$) and significantly peaked 15 min after the stressor (Time^2: $t(1013) = -19.527$, $p < 0.001$, partial $\eta^2 = 0.273$).

Effects of Death Thoughts and Early-life Adversity on Cortisol Responsivity

For our primary hypothesis, we made a GLM regressing cortisol AUC_G onto the formula: ELA*Death thoughts + ELA + Death thoughts + Study + Intercept. Participants in Study 1 showed significantly greater cortisol AUC_G, $F(1,123) = 4.43$, $p = 0.037$, partial $\eta^2 = 0.035$. We further observed a significant ELA by death thought interaction, $F(1,123) = 5.90$, $p = 0.017$, partial $\eta^2 = 0.046$, depicted in Fig. 3. As hypothesized, post-hoc LSD t-tests showed that, without prior exposure to death thoughts, high ELA participants exhibited blunted cortisol AUC_G compared to low ELA participants, $\Delta(123)$(standardized difference) $= 2.31$, $p = 0.023$, Cohen's $d = 0.549$. Furthermore, the difference between high and low ELA participants diminished if participants were primed with death thoughts, $\Delta(123) = 1.23$, $p = 0.223$, Cohen's $d = 0.311$. Death thoughts, relative to the control condition, had

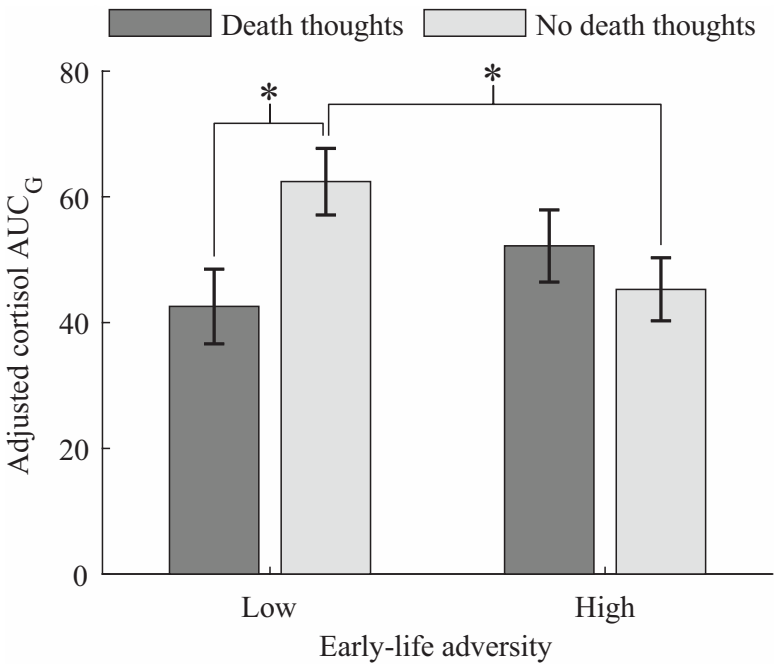

Fig. 3 Group differences in cortisol responsivity to psychosocial stress. Average cortisol responsivity (cortisol AUC_G) for each group is adjusted to control for differences between Studies 1 and 2. Participants in the experimental condition (dark bars) wrote about death, while those in the control condition (light bars) wrote about a non-death related topic. Without death thoughts, low ELA participants released significantly more cortisol after the stressor compared to high ELA participants. Inducing death thoughts did not change cortisol responsivity significantly in high ELA participants but blunted responsivity significantly in low ELA participants. $*=p<.05$. Error bars are standard error

no significant effect on high ELA participants, $\Delta(123)=1.09$, $p=0.279$, Cohen's $d=0.225$. In contrast, among low ELA participants, death thoughts significantly reduced cortisol AUC_G, $\Delta(123)=2.28$, $p=0.024$, Cohen's $d=0.639$.

Effects of Death Thoughts on Subjective and Cortisol Before the Stress Task

To see if cortisol or standardized subjective stress changed after death thoughts (before the stress task), we performed a mixed design ANOVA. There was no significant time by death thought interaction, or time by death thought by ELA interaction (all $F(1,123)<0.15$, $p>0.701$). The same analysis was performed with Box-Cox transformed standardized subjective stress as the dependent variable. Like cortisol, there was no significant time by death thought, or time by death thought by ELA interaction (all $F(1,123)<0.16$, $p>0.689$).

Discussion

This study investigated whether inducing thoughts of one's own death momentarily moderates effects of ELA on HPA responsivity to psychosocial stressors. Cortisol and subjective stress did not increase up to 20 min after death thoughts. These results are in line with other studies reporting no change in stress markers (e.g. heart

rate, negative affect) after death thoughts (Rosenblatt et al., 1989). However, both subjective stress and cortisol increased significantly after exposure to acute psychosocial stress. While death thoughts did not evoke a significant stress response by themselves, they changed how low ELA participants responded to the psychosocial stressor. As hypothesized, inducing death thoughts shortly before a psychosocial stressor significantly reduced HPA responsivity for participants reared in supportive conditions, but did not significantly affect responsivity in high ELA participants. This finding suggests that even subtle non-stressful death reminders presented before a psychosocial stressor can momentarily modulate HPA responsivity to stress, depending on the individual's exposure to ELA.

We believe that the interacting effects of death thoughts and ELA on HPA responsivity are relevant to researchers studying the HPA axis and may partly explain why effects of ELA on HPA responsivity vary across studies in certain situations. If briefly contemplating your own death reduces the cortisol stress response in low ELA participants by more than 50%, any study examining HPA responsivity should account for potential death reminders that may be inadvertently present in the testing environment. For example, many questionnaires routinely used in behavioral studies ask about suicidal ideation to assess depression or inquire into familial death to assess significant life events. If these assessments induce death thoughts, they could potentially blunt HPA responsivity in low ELA participants and may consequently contribute to inconsistent findings across studies. Given the current results, we recommend that future investigations administer death-related assessments either after stress testing, or during a separate testing session.

Extensive research shows that HPA responsivity differs between individuals high and low on ELA. This is the first evidence we know of to show that this difference may be temporarily affected by conditions participants experience immediately before testing. This finding extends past research by our group. We previously found (Engert et al., 2010) that repeated exposure to the TSST blunted cortisol responsivity over time, but only in adults without ELA. Cortisol responsivity in high ELA participants remained blunted across repeated TSST exposures. This resembles what we observed here, only instead of exposing participants to repeated stress, we induced death thoughts before the stressor. One possible explanation is that death contemplation may induce internal processes (evolved conditional adaptive responses) that are similar to those evoked by recurring stress, but without directly activating strong cortisol or subjective stress responses. In other words, inducing death thoughts may partially resemble a stressor without provoking an acute stress response.

The question arises what psychological and physiological processes are initiated by death thoughts. Numerous experiments demonstrate robust effects of death thoughts on risk taking and reproductive preferences (Burke et al., 2010), although it is important to note that certain effects of death thought induction have proven difficult to replicate recently (Klein et al., 2022; Treger et al., 2023). Participants primed with death thoughts desired more offspring (Fritsche et al., 2007; Mathews & Sear, 2008; Wisman & Goldenberg, 2005), were more willing to engage in casual or risky sex (Gillath et al., 2011; Lam et al., 2009; Silveira et al., 2014; Taubman Ben-Ari, 2004), to reduce mate standards (Hirschberger et al., 2002a, 2002b), and engage in other risky behaviors (Hart et al., 2010; Hirschberger et al., 2002a, 2002b; Kelley

& Schmeichel, 2015; Routledge et al., 2004). Interestingly, momentary effects of death thoughts on risk taking and reproductive characteristics are similar to some of the chronic effects of ELA on risk taking and reproductive characteristics (Alley & Diamond, 2021; Kogan et al., 2015; Lovallo, 2013; Simpson et al., 2012; Thomas et al., 2023).

Life-history theory offers a potential explanation for why ELA and death thoughts exert similar effects. While several authors have argued that life-history strategy stabilizes early in life (Simpson et al., 2012), the aforementioned studies on death thoughts and life-history strategy related characteristics suggest that aspects of life-history strategy may change accurately and temporarily in response to mortality cues (along with cues predicting other life-history strategy relevant conditions, such as unpredictability). If inducing death thoughts momentarily shifts life-history strategy, then this may temporally alter the HPA axis. Across species, blunted HPA responsivity facilitates faster life-history strategy, while high HPA responsivity facilitates slow life-history strategy (Carere et al., 2010; Réale et al., 2010). According to the adaptive calibration model (Del Giudice et al., 2011), HPA responsivity changes to adapt life-history strategy to levels of premature mortality (in addition to unpredictability) that individuals detect in their environment.

Results from previous studies suggest that humans avoid thinking about their own death (Hayes et al., 2010). Since death thoughts typically occur in situations with increased mortality (Abdel-Khalek & Lester, 2003; Alaimo et al., 2002; Cox et al., 2012; Robertson et al., 2006; Shakil & Yousaf, 2015), acute death thoughts induced experimentally may also signal increased mortality and subsequently elicit temporary adaptive changes in HPA responsivity and life-history strategy. Our study supported this hypothesis in two key aspects. First, without death thoughts, participants showed greater HPA responsivity if they were reared in supportive conditions, and blunted responsivity if they experienced high ELA. This is consistent with life-history theory (see Table 1), as it claims that high ELA chronically signals increased risk of premature mortality (Belsky et al., 1991; Chisholm, 1996; Chisholm et al., 1993; Del Giudice et al., 2011; Ellis & Del Giudice, 2019; Ellis et al., 2017). Second, low ELA participants also showed blunted HPA responsivity but only when they were primed with death thoughts. Here, death thoughts could have *temporarily* shifted low ELA participants from high HPA responders to low responders to facilitate faster life-history strategy. The interpretation would be that mortality can change acutely as well as chronically. Death thoughts may therefore have similar biobehavioral effects as ELA since both death thoughts and ELA signal higher morality; however, we expect the effects of death thoughts to occur on a much shorter time scale than effects of ELA.

While blunted cortisol responsivity to stress is associated with a range of pathologies (Zorn et al., 2017), it may also have adaptive functions in adverse conditions. From the perspective of life-history theory, blunted cortisol responsivity could be adaptive by facilitating a fast life-history strategy. While the association between life-history strategy and HPA function is complex (Crespi et al., 2013; Duckworth et al., 2023), fast life-history strategy has been associated with blunted HPA responsivity in various species (Carere et al., 2010; Réale et al., 2010). In humans, research has found an association between blunted cortisol responsivity to stress and various

characteristics of fast life-history strategy including short-term mating orientation (Ponzi et al., 2016), younger age at first intercourse (Brody, 2002), more sexual partners (Halpern et al., 2002), impulsivity (Lovallo, 2013), aggression (Gordis et al., 2006), and risk taking (Brown et al., 2016). The relationship between certain life-history characteristics, such as risk taking, and cortisol responsivity may vary between males and females (Wood et al., 2021).

There are likely many ways through which lower HPA responsivity may facilitate faster life-history strategy. One way may involve the effects of the HPA on the hypothalamic-pituitary–gonadal (HPG) axis, which plays a critical role in regulating reproduction. According to Wingfield and Sapolsky (2003), robust HPA activation during stress both acutely and chronically inhibits the HPG thereby suppressing reproduction until conditions are more favorable. Delaying reproduction during stress is only adaptive when organisms have sufficient future reproductive potential. It is not adaptive to delay reproduction when future reproductive potential is low (i.e., when premature mortality is high or unpredictable). Consequently, as Wingfield and Sapolsky (2003) explain, organisms with limited future reproductive potential may increase their reproductive fitness by reducing their HPA responsivity or by altering the cross-talk between the HPA and HPG, thus avoiding inhibition of the HPG.

Beyond life-history theory, there may be adaptive functions of blunted cortisol responsivity in conditions where premature mortality is high. Excessive chronic activation of the physiological stress response systems leads to increased ware and tear on various physiological systems (e.g., chronic suppression of the immune system) (McEwen, 1998). While reduced HPA responsivity may be associated with various costs, it may have certain benefits such as allowing for immulogical responses and preventing the accumulation of allostatic load (Fries et al., 2005).

According to Nettle and Frankenhuis (2020), while a growing number of investigations have applied life-history theory to explain individual differences in humans, it is important to note that life-history originated in the context of ecology and evolutionary biology to describe between-species differences. One should therefore consider other theoretical interpretations for our findings. In addition to life-history theory, terror management theory could also provide some insight into our findings. Terror management theory (Greenberg et al., 1997; Solomon et al., 1991) argues that when individuals are confronted with the thought of their own death, they employ various cognitive defense strategies (terror management processes) such as worldview defense or self-esteem striving. Terror management processes vary between individuals as a function of attachment style and other individual characteristics (Mikulincer & Florian, 2000). It is possible that death thoughts trigger different terror management processes in low and high ELA and terror management processes may have different effects on cortisol responsivity. This claim should be investigated empirically.

Taken together, future studies interested in studying ELA and HPA responsivity should control for situational factors that might induce acute death thoughts. Previous studies from terror management theory provide examples of the vast array of stimuli that can serve this purpose, e.g., images of natural disaster, reading about death, etc. (Hayes et al., 2010). The current study shows that thinking about death

for ten minutes lowered HPA responsivity of low ELA participants to the level of those reporting high ELA.

Our results may also be of interest to researchers studying terror management theory. Recently several studies have struggled to replicate the effects of mortality cues on specific psychological outcomes (self-esteem and worldview defense; Klein et al., 2022; Treger et al., 2023). While we did not examine these outcomes in our study, it is possible that ELA moderates the effects of death thoughts on other outcomes besides the cortisol stress response. Indeed, other studies have found that the effects of mortality cues on reproductive preferences and risk-seeking vary according to ELA (Griskevicius et al., 2011a, 2011b; Griskevicius et al., 2011a, 2011b). While various factors may explain inconsistencies in the effects of death thoughts between studies within terror management theory (e.g., Klein et al., 2022), it may be worthwhile for terror management theory researchers to consider ELA as a potential moderator for the effects of death thoughts, and to control for this variable in future research.

Limitations and Future Directions

Several limitations should be considered regarding the results of this study. First, our findings so far only apply to men – although we are aware of the bias in experimental research favoring men, we were unable to secure the financial and logistical resources necessary to test both men and women. Therefore, we cannot speak to the applicability of these findings to the female population. Since life-history theory proponents (Del Giudice et al., 2011) claim that fast life-history strategy is less feasible for females, this questions the generalizability to women. Several studies found that death thoughts induce fast life-history characteristics (e.g. risk preference) in men, but not in women (Hirschberger et al., 2002a, 2002b; Hirschberger et al., 2002a, 2002b; Mathews & Sear, 2008). Indeed, Byrd-Craven and colleagues (2015) found that death thoughts blunted cortisol responsivity in men but did not affect women. Thus, future research should examine how death thoughts and ELA interact with sex to affect life-history strategy and HPA function.

Another limitation is that our sample under-represents high ELA participants. To control for effects of substance use and mental illness on HPA axis responsivity, we recruited only healthy individuals. Typically, however, individuals high in ELA are more likely to engage in substance use, or experience psychopathology (De Bellis & Zisk, 2014; Levis et al., 2022; Smith & Pollak, 2020). Consequently, our ELA group largely reflects a subpopulation of individuals who are relatively resilient to the harmful effects of ELA. Perhaps latent factors protecting these individuals from the harmful effects of ELA also rendered their HPA axes more resistant to death thought induction. Thus, individuals exposed to ELA with poorer health may respond differently to death thoughts.

Furthermore, while we tested participants at the same time of day, we did not record the time of awakening, which could have impacted their cortisol stress response. Future research should control for awakening time. It is also important to note that we only examined cortisol responsivity to standardized psychosocial

stress tasks. The pattern of results we observed in high ELA participants could reflect habituation of the HPA to real life stressors such that laboratory stressors do not elicit a response. Similar conclusions were reached by Stout et al. (2024) and Wood et al. (2021). Finally, our results combined two different stressors, the MIST and TSST. The MIST and TSST differ in several ways. For instance, the MIST does not require participants to compose a speech and simply involves mental arithmetic. There is also less direct social evaluation in the MIST. We do not expect this to have a significant impact on our results as we controlled for the type of stressor in our analysis.

There are several potential avenues for future research which should be explored. First, it is critical to conduct this experiment with female participants since there are clear sex differences in the effects of ELA on stress responsivity and life-history strategy, as explained above. Secondly, it is also important to note that this study specifically focused on cues of mortality (death thoughts), but premature mortality is only one factor that calibrates life-history strategy and stress response systems; unpredictability (or variability in harshness) is also an important influence on the development of life-history strategy (Del Giudice et al., 2011; Ellis et al., 2009, 2022). Future research should also see whether priming participants with cues signaling unpredictability has similar effects to mortality cues on the cortisol stress response. While unpredictability influences life-history strategy, it also reduces the plasticity of life-history strategy, or specifically the sensitivity of life-history strategy to subsequent environmental cues (Ellis et al., 2022). The idea here is that when levels of premature mortality cannot be reliably determined, it is no longer justifiable from evolutionary perspective to maintain the costly capacity of plasticity (Ellis et al., 2022). While we assessed participants' childhood experiences of parental care, abuse, and neglect, we did not measure the level of unpredictably or instability in their early-life environments. Future attempts to replicate this study should specifically measure early-life unpredictability to see if this is a reason why individuals exposed to ELA are less affected by mortality cues.

Conclusions

This study showed how high ELA interacts with acute exposure to mortality cues in adulthood (death thoughts) to affect HPA function under stress. Future studies should therefore control for the presence of mortality cues in their testing protocols to avoid contaminating associations between ELA and HPA function. Furthermore, life-history theory may be relevant to explaining both the long-term calibration of HPA function (e.g., due to ELA), as well as short-term changes in HPA function due to acute exposure to mortality-related stimuli.

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Data and code availability Please contact corresponding author.

Authors' contributions E.Z. contributed to the study conceptualization and design, material preparation, data collection, analysis, figure preparation, and writing of the manuscript. R.P.J. contributed to writing. A.C.J. and C.C. contributed to material preparation and data collection. J.C.P. supervised and secured funding for the project and contributed to the writing of the manuscript.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Ethics This study was performed in line with the principles of the Declaration of Helsinki. The study was approved by research ethics board of the McGill University Faculty of Medicine research ethics board (Approval No: A10-B38-06B), Montreal, Quebec, Canada. All participants provided written informed consent to participate and have their data published.

Competing interests The authors declare no competing interests.

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