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SHORT COMMUNICATION

Resilience and hypothalamic-pituitary-adrenal axis reactivity under acute stress in young men

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Abstract
The present study examined the relationship between resilience (measured using the *Resilience Scale for Adults*) and hypothalamic-pituitary-adrenal (HPA) axis reactivity. We examined the subjective and cortisol responses of 28 healthy young men to an acute stressor (public speech task). Eight saliva samples were collected in order to obtain the response curve (anticipation, reactivity, recuperation) for each subject. ANOVA indicated that highly resilient individuals tended to display less mood deterioration than less resilient individuals (marginal $p_{\text{time} \times \text{group interaction}} = 0.075$). They also revealed that the former tended to secrete less cortisol overall than the latter during the experiment (marginal $p_{\text{main group effect}} = 0.087$) but this effect was not uniform across time ($p_{\text{time} \times \text{group interaction}} = 0.029$). Additional analyses performed to identify the source of this interaction revealed that resilience moderates cortisol secretion in *anticipation* of the stressor (i.e. highly resilient individuals secreted less cortisol than less resilient ones, $p = 0.05$) but that it is not conductive to lower HPA reactivity *amidst* stress (i.e. there was no difference between groups in the increase in cortisol secretion from baseline to peak). The recovery slopes were likewise not statistically different. The implications of these findings regarding health are discussed.

Keywords: *Hypothalamic-pituitary-adrenal (HPA) axis, negative affect, positive affect, resilience, salivary cortisol, trier social stress test*

Introduction

Although all human beings encounter stressful events, they do not respond identically to such experiences. Whereas some individuals adapt flexibly to the changing demands of stressful experiences, others cope far less effectively. The construct of resilience has been proposed to account for this variability. Resilience is defined as the ability to thrive in the face of adversity (Block & Kremen, 1996; Tugade & Fredrickson, 2004). Because this definition targets the final outcome—and not what contributes to this outcome—it leaves little room for prediction (Hjemdal et al. 2006). Measures of resilience therefore encompass the factors known to promote resilience, which fall under three categories: (1) positive dispositional attributes, (2) a coherent and loyal family, and (3) a supportive social network (Werner and Smith 2001). The more features a person possesses (i.e. the higher his/her scores on resilience scales), the more resilient s/he is said to be. Validation studies have confirmed that these factors do promote resilience in that (1) individuals scoring high on resilience scales are less likely to develop mental or somatic disorders and (2) these scales discriminate efficiently between psychiatric and healthy populations (Friborg et al. 2003).

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Investigations of the psychological correlates of this construct indicated that resilient individuals are characterized by relatively high positive affectivity (Block & Kremen, 1996) which they cultivate through acceptance coping (Heiman 2002), constructive thinking (Spaccarelli & Kim, 1995) and reliance on social support (Connor & Davidson, 2003). In contrast with the profusion of behavioural studies, only one study to date (Tugade & Fredrickson, 2004) has investigated the biological correlates of resilience. Using a public speech task as the stressor and measuring both subjective and cardiovascular reactivity, these authors showed that resilient individuals do not differ from their counterparts regarding the magnitude of their responses but they do recover significantly faster. Although very informative, this study relied on indices of autonomous arousal whose interpretation is sometimes ambiguous (heart rate can both decrease or increase following stress; Cacioppo et al. 2000). In contrast, the activation of the hypothalamic-pituitary-adrenal (HPA) axis and the secretion of cortisol represent an unambiguous indicator of stress. Moreover, on account of its ubiquitous functions in the body (Chrousos 1998), cortisol may be considered a potent mediator of the biological consequences of stress that could explain protection of resilient individuals from mental and somatic diseases. The role of cortisol in the regulation of blood pressure, glycaemia, immune and inflammatory functions, bone resorption and cognitive functions is indeed well documented. Numerous studies also suggest that hypercortisolemia may play a role in the aetiology of melancholic depression (Ehlert et al. 2001; Gillespie and Nemeroff 2005) and it is a well-known biological marker of other psychiatric disorders such as anorexia nervosa (Licinio et al. 1996) and alcoholism (Adinoff et al. 2003).

The aim of the present study was to examine the impact of resilience on HPA axis reactivity under stress. Given that individuals scoring high on resilience scales are less likely to experience negative psychological and somatic consequences in the face of stressors (Firborg et al. 2006), we hypothesized that it could be due to lower HPA reactivity. To this end, we subjected participants to a well characterized laboratory stressor and measured both psychological (mood deterioration) and biological (cortisol secretion) responses.

Methods

Sample

Twenty eight non-smoker male students (mean age: 20.86 years, SD: 2.38), recruited through advertisement, participated in the study in exchange for course credit or remuneration. Women were excluded due to possible impact of the female menstrual cycle phase and use of oral contraceptives (Kirschbaum et al. 1999). Students were interviewed, and those who presented somatic or psychiatric illnesses or who received any form of medication were also excluded. The participants were informed that the study was about individual differences in job interviews. They were instructed (1) not to abuse alcohol the day before the experiment and to respect their usual sleeping hours; (2) not to ingest alcohol, caffeine, or soda drinks the day of the experiment; and (3) not to ingest any food or drink 1 h before the start of the experiment.

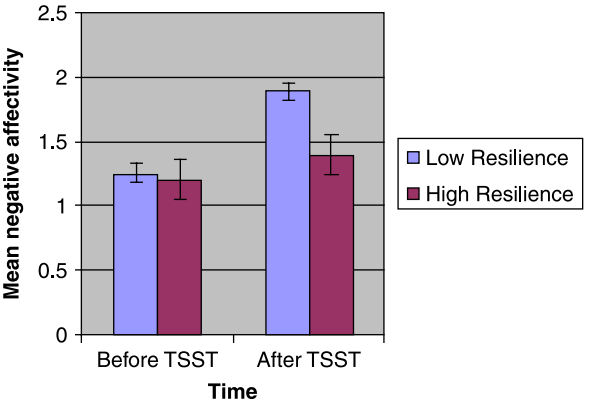

Figure 1. Mood Deterioration as a Function of the Level of Resilience. Note: The "low resilience" group comprises the 14 individuals scoring below the mean whereas the "high resilience" group comprises the 14 individuals scoring above the mean. The Time x Group interaction effect is marginally significant ($p \leq 0.075$).

Procedure

The experiment was conducted in accordance with the Declaration of Helsinki and was approved by the IRB. The effect of circadian hormone rhythms was minimized by conducting all sessions between 14:00 and 18:00 h. After providing written informed consent and a basal sample of saliva, participants underwent a short relaxation procedure (1 min), and then were left alone for 10 min in a comfortable room with several magazines at their disposal. Baseline positive and negative affectivity were then assessed through the positive and negative affect schedule (PANAS). After a second basal sample of saliva was taken, subjects were introduced to the Trier Social Stress Test (TSST; Kirschbaum et al. 1993). This stressor, which has repeatedly been found to induce profound endocrine and cardiovascular responses in 70–80% of the subjects tested, consists of both a public speech (5 min, after a 10–min preparation period) and a cognitive task (5 min) in front of an audience of two people and a video camera. Afterwards, participants returned to the first room, provided a sample of saliva and were re-tested on the PANAS. They spent the rest

of the experiment alone in the room (reading magazines), interrupted only for saliva collection (see Figure 2 legend for exact timing). They were debriefed just before the last sampling and completed the Resilience Scale for Adult (RSA) in a questionnaire session taking place 3 weeks after the experiment.

Measures

Resilience factors. These were measured through the Adult Resilience Scale (RSA; Friborg et al. 2003). The RSA consists of 41 items (responded to on a 7-point scale) targeting 5 factors known to promote resilience: personal competence ("No matter what happens I always find a solution"), personal structure ("I keep up my daily routine even in difficult times"), social competence ("I easily adjust to new social milieus"), family coherence ("In our family, we are loyal towards each other") and social support ("I have some close friends/family members who really care about me"). The Cronbach α was 0.91 in the present sample. The RSA applies for both healthy and psychiatric populations and efficiently distinguishes between them (Friborg et al. 2003). The general norms for men/women and controls/patients were reported in Friborg et al. (2003). The norms relevant to the present sample were communicated by Braun (personal communication, 2007) on the basis of Hjemdal & Braun (in preparation): in a healthy Belgian French-speaking population, the mean for young men (age-group 17–25 years) is 5.13, with a SD of 0.71.

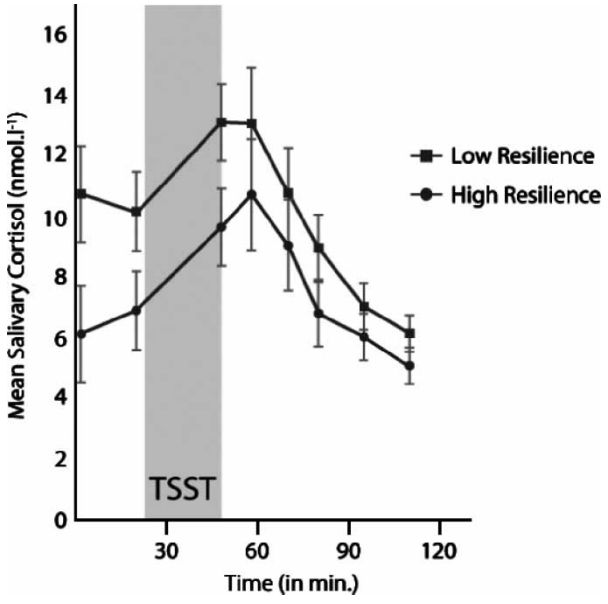

Figure 2. Salivary Cortisol Responses as a Function of the Level of Resilience. *Legend*. N was 14 subjects in each group. Values are means ± SEM. Baseline cortisol concentrations differ significantly across groups ($p \leq 0.05$), the integrated responses (AUCg) differ marginally ($p \leq 0.075$), the increase from baseline to peak (AUCi) does not. The timing for a session was as follows (in minutes): 0–2: Welcome and written informed consent. 2–4: First, salivette. 5–6: One-minute relaxation procedure. 6–16: Free relaxation time (e.g. reading a magazine). 16–21: Positive and negative mood assessment. 21–23: Second, salivette. 23–26: Instructions for the Trier Social Stress Test (job interview). 26–36: Preparation for the job interview. 36–41: Job interview. 41–44: Instructions for the cognitive task. 44–48: Cognitive task. 48–50: Third salivette. 50–58: Positive and negative mood assessment. 58–60: fourth salivette. 60–70: Free relaxation time. 70–72: Fifth salivette. 72–81: Free relaxation time. 81–83: Sixth salivette. 83–96: Free relaxation time. 96–98: Seventh salivette. 98–113: Debriefing. 113–115: Eighth salivette.

Positive and negative affectivity. These were assessed through an extended version of the (PANAS; Watson et al. 1988). In its original version, this questionnaine consists of 20 adjectives rated along 5-point scales, of which 10 measure positive affectivity (PA) and 10 measure negative affectivity (NA). In order to increase the sensitivity of the instrument to the manipulation, the following adjectives were added on account of their particular relevance to our investigation: 'disheartened,' 'incapable,' 'grumpy,' 'disgusted,' and 'tense'. The Cronbach alphas for PA and NA were respectively 0.85 and 0.88 at baseline and 0.90 and 0.89 after stress induction.

Cortisol secretion. Saliva samples were collected using the Sarstedt® Salivettes (Nümbrecht, Germany) and stored at room temperature until completion of the session and at −20°C until assay. The cortisol assays were carried out at the Laboratoire de Biologie Clinique, Cliniques Universitaires Saint Luc (Brussels, Belgium). Saliva was extracted from the cotton swab by centrifugation (1000 g, 2 min) and the cortisol was measured using a competitive polyclonal immunoassay, comprising an electromagnetic separation step followed by electrochemiluminescence quantitation with the Elecsys 1010/2010 analyser (Roche Diagnostics, Mannheim, Germany). The intra- and interassay coefficients are respectively 4.0% and 7.2%. Results are expressed in nmol/l.

Statistical procedures

The effect of resilience on *mood deterioration* was examined through repeated measures ANOVA, with time (two times: before and after the stress induction) as the intra-subject factor and the level of resilience (two categories: below and above the mean, respectively named high and low resilient) as the between-subject factor.

The effect of resilience on *cortisol secretion* was also examined through repeated measures ANOVA, with time (i.e. the 8 saliva collection times) as the intra-subject factor and the level of resilience (from the RSA) as the between-subject factor. In order to identify the source of the effects we also computed

three indicators: (1) baseline cortisol, which was obtained by averaging the first two samplings; (2) increase in cortisol from baseline to peak (i.e. area under the curve with respect to the increase, AUCi), which was computed using the trapezoidal method recommended by Pruessner and colleagues (2003); and (3) the recovery slope, which was obtained by regressing cortisol data for salivette 4 on data for salivette 8 in each group.

Results

The mean resilience score in our sample was 5.13 (SD: 0.73), which is within the norm for this population (see above, *Measures* section). Groups were created via mean-split, resulting in a sample size of 14 individuals in each. They were named low resilience (LR) and high resilience (HR) groups. The means (and SD) were respectively 4.52 (0.30) and 5.73 (0.46). The descriptive statistics for the variables under study are reported separately for each group in Table I.

ANOVA conducted on the *psychological* response revealed a significant effect of time ($F = 16.12$, $p < 0.001$) indicating that NA increased in response to stress. There was also a marginally significant time × group interaction effect ($F = 3.50, p = 0.075$), indicating that highly resilient individuals tended to display less mood deterioration (i.e. less increase in NA) than their less resilient peers (see Figure 1). The latter effect was mostly attributable to two of the resilience construct's factors: personal competence and social competence.

ANOVA conducted on the *endocrine* response yielded a significant main effect of time ($F = 13.24$, $p < 0.001$), indicating that cortisol secretion increased in response to stress and then decreased during the recovery period. There was also a marginal main effect of the group ($F = 3.16$, $p = 0.087$), suggesting that highly resilient individuals tended to secrete less cortisol overall than their less resilient peers. Finally, there was a time × group interaction effect ($F = 5.9$, $p = 0.029$), indicating that the latter effect was not uniform across time (see Figure 2). As this can potentially be attributable to three factors (i.e. baseline, increase or recovery), we performed additional analyses to document the source of the interaction. Analyses on baseline revealed a significant difference between groups ($t = 2.0$, $p = 0.05$), with highly resilient individuals secreting significantly less cortisol than less resilient individuals. Analyses on the increase in cortisol secretion from baseline to peak (AUCi) revealed that there was no statistically significant difference between groups ($t = -1.11, p = 0.28$). The recovery slopes were likewise not statistically different ($t = 1.26, p > 0.10$). It is noteworthy that the effect of resilience on overall cortisol secretion was mostly attributable to the resilience factors social competence and social support. The significant effect on baseline was mostly due to personal competence, family coherence and social support.

Analyses of the relationship between psychological and endocrine indicators of the stress response revealed that they were moderately but significantly correlated (Table II). It is of note that resilience did not moderate the relationship between psychological and endocrine indicators.

Table I. Descriptive statistics for affect and cortisol secretion before and after the trier social stress test in low and high resilience subjects.

	Low resilience ($n = 14$)	High resilience ($n = 14$)
Mean NA before (± SD)	1.26 (0.34)	1.20 (0.21)
Mean NA after (± SD)	1.80 (0.61)	1.40 (0.51)†
Difference in NA (± SD)	0.55 (0.45)	0.20 (0.53)†
Mean AUCg (± SD)	1127 (435.0)	846 (363.9)†
Mean Baseline (± SD)	10.46 (6.94)	6.52 (2.49)*
Mean Peak (± SD)	13.07 (6.90)	10.72 (6.73)
Mean AUCi (± SD)	0	165.4 (373.2)
Recovery slopes	0.88	0.80

NA = negative affectivity, AUCg = Area under the curve with respect to the ground (zero), AUCi = Area under the curve with respect to the increase, Mean at the peak = mean cortisol in salivette 4, Recovery slopes = standardized betas. AUCg and AUCi were computed according to the formulae presented in Pruessner et al. (2003). $^{\dagger}p \leq 0.10$, $^{\star}p \leq 0.05$.

Discussion

The aim of the present study was to examine the impact of resilience factors on psychological and cortisol reactivity to an acute stressor. The results indicated that individuals in the HR group tended to report less mood deterioration than individuals in the LR group, thereby supporting the idea that resilience factors exert a protective effect regarding subjective reactivity to stress. At the biological level, the results revealed that the HR group tended to secrete less cortisol overall during the experiment than the LR group. However, this effect was not uniform across time. Additional analyses performed to detect the source of this interaction revealed that resilience moderates cortisol secretion *before* the stressor, but is neither conductive to a lower HPA reactivity *amidst* stress nor to faster HPA recovery.

Such baseline differences suggest that HR and LR individuals differ in their anticipation of potentially stressful situations (it is of note that the subjects knew from enrolment that the study was about job interviews). The hypothesis that these baseline differences are attributable to anticipation-related differences rather than to differences in the tonic functioning of the HPA axis is especially likely since (1) the LR and HR groups do not differ in baseline cortisol release when at rest (Mikolajczak, Roy & de Timary, unpublished data), (2) HR individuals

Table II. Correlations between negative affect and salivary cortisol measurements before and after the trier social stress test ($n = 28$).

Variable	1	2	3	4	5	6	
1. NA before	1	–	–	–	–	–	–
2. NA after	0.49**	1	–	–	–	–	–
3. Increase in NA	0.01	0.88***	1	–	–	–	–
4. Baseline cortisol	0.47*	0.26	0.05	1	–	–	–
5. Peak cortisol	0.07	0.48**	0.51**	0.21	1	–	–
6. AUCg	0.22	0.51**	0.46*	0.65***	0.87***	1	–
7. AUCi	−0.43*	0.07	0.31	−0.74***	0.42*	−0.005	1

Note: NA = negative affectivity; AUCg = Area under the curve with respect to the ground; AUCi = Area under the curve with respect to the increase; Baseline Cortisol = Mean salivettes 1 and 2; Peak cortisol = salivette 4. *$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$.

appraised an upcoming public-speech task as less threatening than LR individuals in Tugade & Frederickson's (2004) study, and (3) anticipatory cognitive appraisal influences cortisol secretion, with threat appraisal leading to higher cortisol release than challenge appraisal (Gaab et al. 2005). These differences in anticipation of stressful events are of significant importance regarding health because under real life conditions, anticipation often lasts longer than the stress, (e.g. examinations, oral presentations). Moreover, elevations in cortisol secretion have been shown to increase corticotropin-releasing hormone (CRH) mRNA expression in the amygdala, resulting in exaggerated response to fear (Schulkin et al. 1998). Thus, it is plausible that cortisol secretion in response to previous stressful experiences results in exaggerated threatening anticipation of upcoming stressors.

Furthermore, baseline differences could potentially account for the absence of significant differences between groups in the increase in cortisol release from baseline to peak. It is well-known, indeed, that cortisol release feeds back on the anterior pituitary corticotrops to reduce the secretion of adrenocorticotropic hormone (ACTH), resulting in a decrease in the synthesis and release of cortisol from the adrenal cortex. Accordingly, a study in rats showed that the administration of 100 μg/kg corticosterone immediately prior to restraint stress significantly decreased subsequent plasma ACTH responses to restraint (Viau et al. 1993). Thus, it is plausible that the "hyporesponsiveness" in cortisol secretion displayed by less resilient individuals amidst stress may be accounted for by an increased feedback action of elevated baseline (anticipation-related) cortisol levels. Such effective functioning of this feedback loop indicates that the HPA axis is relatively healthy in the less resilient subjects (Gillespie & Nemeroff, 2005). The finding of evident integrity of feedback in the HPA axis of the LR group is not surprising because the range of resilience scores was restricted in our sample since, by excluding participants with mental or somatic disorders, we removed individuals who were likely to exhibit HPA axis hyperactivity and impaired feedback regulation (Evans & Nemeroff, 1983; Gillespie & Nemeroff, 2005; Lammers et al. 1995). However, it cannot be excluded that chronic higher overall cortisol secretion in our "LR" group in the face of stressors ultimately leads to a dysregulation of HPA axis functioning. Hence, in spite of the tendency of LR individuals to release less cortisol than their HR counterparts *amidst* stress, the overall cortisol secretion throughout the experiment was nonetheless greater in the LR group. Considering the number of stressors an individual will face during his/her lifetime along with the ubiquitous functions of cortisol in the body, the cumulative effects of these differences in cortisol secretion may underlie the vulnerability of LR individuals to stress-related mental and somatic diseases, and the protection of HR individuals from these disorders (McEwen 1998; McEwen & Stellar, 1993).

It is noteworthy that the various resilience factors studied did not have the same weight in predicting the subjective and cortisol responses to stress. Four out of five factors (*personal competence*, *social competence*, *family coherence* and *social support*) contributed to the effect of the global score, with their weight and significance depending on the variable under consideration. However, one factor, namely *personal structure*, was not significant at all in the prediction of the stress response. Given that this factor refers to an individual's ability to preserve a certain routine in adverse times, it is plausible that it may be more relevant, and thus have a greater weight, in the face of long-lasting or chronic stressors. The disparity in the contribution of the various factors to the global effect of resilience suggests that, although these factors all contribute to protect the individual, they do not rely on the same mechanisms. This does not weaken the usefulness of the scale, which has proven to be a useful tool to identify vulnerable individuals (Friborg et al. 2003), but it suggests that future studies should investigate separately the mechanisms through which these protective factors exert their effects.

Albeit indicative, the present findings have several limitations. Replications on larger and more heterogeneous samples are necessary. It would also be informative to extend these findings to long-lasting

stressors, (e.g. by examining whether resilience moderates lymphocyte proliferation and interleukin-2 production; see Segerstrom & Miller, 2004 for a meta-analysis of the component of the immune system affected by chronic stress) because the protective effect of resilience may be even more pronounced and detectable amidst long-lasting adversity.

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Disclosure

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References

Adinoff B, Ruether K, Krebaum S, Iranmanesh A, Williams MJ. 2003. Increased salivary cortisol concentrations during chronic alcohol intoxication in a naturalistic clinical sample of men. Alcohol Clin Exp Res 27:1420–1427.

Block J, Kremen AM. 1996. IQ and ego-resiliency: Conceptual and empirical connections and separateness. J Pers Soc Psychol 70:349–361.

Cacioppo JT, Tassinary LG, Bernston GG. 2000. Handbook of Psychophysiology. 2nd ed. New York: Cambridge University Press.

Connor K, Davidson J. 2003. Development of a new resilience scale: The connor-davidson resilience scale (CD-RISC). Depress Anxiety 18:76–82.

Chrousos GP. 1998. Stressors, stress, and neuroendocrine integration of the adaptive response - The 1997 Hans Selye Memorial Lecture. Stress of Life 851:311–335.

Ehlert U, Gaab J, Heinrichs M. 2001. Psychoneuroendocrinological contributions to the etiology of depression, posttraumatic stress disorder, and stress-related bodily disorders: The role of the hypothalamus-pituitary-adrenal axis. Biol Psychol 57:141–152.

Evans DL, Nemeroff CB. 1983. The dexamethasone suppression test in mixed bipolar disorder. Am J Psychiatry 140:615–617.

Friborg O, Hjemdal O, Rosenvinge JH, Martinussen M. 2003. A new rating scale for adult resilience: What are the central protective resources behind healthy adjustment? Int J Methods Psychiatr Res 12:65–76.

Friborg O, Hjemdal O, Rosenvinge JH, Martinussen M, Aslaken PM, Flaten MA. 2006. Resilience as a moderator of pain and stress. J Psychosom Res 61:213–219.

Gaab J, Rohleder N, Nater UM, Ehlert U. 2005. Psychological determinants of the cortisol stress response: The role of anticipatory cognitive appraisal. Psychoneuroendocrinology 30:599–610.

Gillespie CF, Nemeroff CB. 2005. Hypercortisolemia and depression. Psychosom Med 67:S26–S28.

Heiman T. 2002. Parents of children with disabilities: Resilience, coping and future expectations. J Dev Phys Disabil 14:159–171.

Hjemdal O, Friborg O, Tore CS, Rosenvinge JH, Martinussen M. 2006. Resilience predicting psychiatric symptoms: A prospective study of protective factors and their role in adjustment to stressful life events. Clin Psychol Psychother 13:194–201.

Kirschbaum C, Kudielka BM, Gaab J, Schommer NC, Hellhammer DH. 1999. Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. Psychosom Med 61:154–162.

Kirschbaum C, Pirke KM, Hellhammer DH. 1993. The trier social stress test - a tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology 28:76–81.

Lammers CH, Garcia-Borreguero D, Schmider J, Gotthardt U, Dettling M, Holsboer F, et al. 1995. Combined dexamethasone/corticotropin-releasing hormone test in patients with schizophrenia and in normal controls: II. Biol Psychiatry 38:803–807.

Licinio J, Wong M-L, Gold PW. 1996. The hypothalamic-pituitary-adrenal axis in anorexia nervosa. Psychiatric Research 62:75–83.

McEwen BS. 1998. Protective and damaging effects of stress mediators. N Engl J Med 338:171–179.

McEwen BS, Stellar E. 1993. Stress and the individual. Mechanisms leading to disease. Arch Intern Med 153:2093–2101.

Pruessner JC, Kirschbaum C, Meinlschmid G, Hellhammer DH. 2003. Two formulas for the computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. Psychoneuroendocrinology 28:916–931.

Schulkin J, Gold PW, McEwen BS. 1998. Induction of corticotropin releasing hormone gene expression by glucocorticoïds: Implication for understanding the states of fear and anxiety and allostatic load. Psychoneuroendocrinology 23:219–243.

Spaccarelli S, Kim S. 1995. Resilience criteria and factors associated with resilience in sexually abused girls. Child Abuse Negl 19:1171–1182.

Tugade MM, Fredrickson BL. 2004. Resilient individuals use positive emotions to bouce back from negative emotional experiences. J Pers Soc Psychol 86:320–333.

Viau V, Sharma S, Plotsky PM, Meaney MJ. 1993. Increased plasma ACTH responses to stress in non-handled compared with handled rats require basal levels of corticosterone and are associated with increased levels of ACTH secretagogues in the median eminence. J Neurosci 13:1097–1105.

Watson D, Clark LA, Tellegen A. 1988. Development and validation of brief measures of positive and negative affect-the panas scales. J Pers Soc Psychol 54:1063–1070.

Werner EE, Smith RS. 2001. Journey from childhood to midlife: Risk, resilience and recovery. Ithaca, NY: Cornell University Press.