Higher body fat percentage is associated with increased cortisol reactivity and impaired cognitive resilience in response to acute emotional stress

Article *in* International Journal of Obesity · December 2008

DOI: 10.1038/ijo.2008.218 · Source: PubMed

CITATIONS
38

READS
371

3 authors, including:

Lilianne R Mujica-Parodi
Stony Brook University
95 PUBLICATIONS **2,444** CITATIONS

SEE PROFILE

Marc Taylor
University of California, Irvine
96 PUBLICATIONS **1,725** CITATIONS

SEE PROFILE

International Journal of Obesity (2009) 33, 157–165

0307-0565/09 $32.00
www.nature.com/ijo

ORIGINAL ARTICLE

Higher body fat percentage is associated with increased cortisol reactivity and impaired cognitive resilience in response to acute emotional stress

LR Mujica-Parodi[1,2], R Renelique[1,2] and MK Taylor[3]

[1]*Laboratory for the Study of Emotion and Cognition, Department of Biomedical Engineering, Stony Brook University School of Medicine, Stony Brook, NY, USA;* [2]*Laboratory for the Study of Emotion and Cognition, Department of Psychiatry, Stony Brook University School of Medicine, Stony Brook, NY, USA and* [3]*Stress Physiology Research Core, Department 162, Warfighter Performance, Naval Health Research Center, San Diego, CA, USA*

Objective: Cortisol is elevated in individuals with both increased emotional stress and higher percentages of body fat. Cortisol is also known to affect cognitive performance, particularly spatial processing and working memory. We hypothesized that increased body fat might therefore be associated with decreased performance on a spatial processing task, in response to an acute real-world stressor.
Design: We tested two separate samples of participants undergoing their first (tandem) skydive. In the first sample ($N = 78$), participants were tested for salivary cortisol and state anxiety (Spielberger State Anxiety Scale) during the plane's 15-min ascent to altitude in immediate anticipation of the jump. In a second sample ($N = 20$), participants were tested for salivary cortisol, as well as cardiac variables (heart rate, autonomic regulation through heart rate variability) and performance on a cognitive task of spatial processing, selective attention and working memory.
Results: In response to the skydive, individuals with greater body fat percentages showed significantly increased reactivity for both cortisol (on both samples) and cognition, including decreased accuracy of our task of spatial processing, selective attention and working memory. These cognitive effects were restricted to the stress response and were not found under baseline conditions. There were no body fat interactions with cardiac changes in response to the stressor, suggesting that the cognitive effects were specifically hormone mediated rather than secondary to general activation of the autonomic nervous system.
Conclusions: Our results indicate that, under real-world stress, increased body fat may be associated with endocrine stress vulnerability, with consequences for deleterious cognitive performance.
International Journal of Obesity (2009) **33,** 157–165; doi:10.1038/ijo.2008.218; published online 18 November 2008

Keywords: body fat; cortisol; emotional stress; cognition; body mass index; spatial processing

Introduction

Cortisol, known generally as the 'stress hormone,' is an important endocrine output of the body's hypothalamic–pituitary–adrenal axis response to emotional and physical stress. The impact of stress on cognitive performance appears to be primarily mediated through cortisol, due to cortisol's effects on the hippocampus[1,2] and prefrontal cortex.[3] These effects are particularly associated with executive and spatial processing, as well as working memory, and have been repeatedly demonstrated both when cortisol is introduced exogenously[4,5] and when it is endogenously secreted during emotionally or physically stressful events.[6–11]

Cortisol and visceral body fat are known to have a strong influence upon one another. Unlike peripheral fat, visceral fat allows for much greater blood flow, contains increased glucocorticoid receptors and therefore is sensitive to the fat-accumulating deposits of cortisol and triglycerides. Visceral adipose tissue becomes larger when it encounters cortisol,[12] excessive secretion of cortisol at baseline increases the accumulation of central fat deposits[13,14] and central fat deposits are significantly larger in individuals with diseases that cause cortisol dysregulation, such as Cushing's syndrome[15] and severe depression.[16–19]

Correspondence: Professor LR Mujica-Parodi, Laboratory for the Study of Emotion and Cognition, Department of Biomedical Engineering, Stony Brook University School of Medicine, Health Sciences Center T18, Stony Brook, NY 11794-8181, USA.
E-mail: lmujicaparodi@gmail.com
Received 30 May 2008; revised 29 September 2008; accepted 6 October 2008; published online 18 November 2008

In this study, we wished to address two key gaps in the literature. First, it is known that body fat and acute stress independently cause increases in cortisol and that heightened cortisol can adversely affect cognitive performance (with important caveats described in our Discussion section and addressed more extensively in Lupien *et al*.[20]). Our study was designed to investigate the transitive implication—that is, to ask whether increased body fat might therefore be associated, not only with cortisol reactivity, but also a decline in cognitive performance displayed in response to emotional stress. Second, the relationships reported between body fat and stress reactivity, as well as cortisol and cognitive performance, have generally been observed under two conditions: either laboratory stressors that are experimentally controlled, yet qualitatively, unlike those typically experienced during normal life or naturalistic stressors established less reliably or uniformly through self-report. To most effectively explore the real-world clinical implications of this model, we therefore investigated whether the relationships between all three variables continued to hold outside the laboratory, in response to an acute stressor (first-time tandem skydive) that was significantly more powerful and genuine than those normally induced during laboratory studies, yet also experimentally uniform and tightly controlled across participants.

Methods

Research design

Our experiment was designed to address two questions investigating the relationship between body fat and resilience to emotional stress. First, we wished to test whether individuals with greater amounts of body fat showed greater cortisol reactivity in response to a genuine acute stressor, thereby assessing whether the validity of earlier laboratory findings does in fact extend to stressors in the 'real world.' Second, as cortisol is known (again, mainly within a laboratory context using pharmacologically introduced cortisol) to have deleterious effects on cognitive performance, we wished to test whether individuals with greater body fat therefore also show impaired cognitive resilience to emotional stress.

Acute stressor

We chose to use participants' first-time skydive for our acute stressor for several reasons. First, a first-time skydive provided complete novelty and therefore avoided the prior exposure and personality confounds that can be an issue with social stressors such as public speaking.[21–23] Tandem jumps were chosen to maximize novelty as, unlike solo jumps, they require less than 5 min of training and therefore pose less of a risk for prior exposure and self-selection bias. Second, skydives provide an isolated and experimentally controlled environment that, unlike most stressors outside the laboratory, can guarantee nearly identical time courses for all participants—a critical feature of any study of individual variability. Participants wore digital altimeters (Altimaster Neptune), confirming consistent rise times of 15 min, jumps at 4 km (13 000 ft), free fall lasting 1 min and parachuting for an additional 4 min before landing. Upon landing, all participants reported that their emotional stress peaked at the aircraft door, shortly before exiting the aircraft; thus, we treated the 15-min anticipatory anxiety during ascent to altitude and before the skydive as the acute stress condition, rather than the jump itself. Third, the skydive provided a genuine risk with real consequences, thereby providing a realistic analog to acute stressors experienced in patients' lives. As testing participants in an aircraft shortly before their jumps involved an environment that was experimentally challenging, we chose to conduct a simpler study with greater numbers of participants to address the first question (Cortisol Reactivity Study), as well as to separately conduct a more complex study with fewer participants to address the second question (Cognitive Reactivity Study).

Participants

Participants were recruited in two batches from individuals who contacted a local skydiving school (Skydive Long Island, Calverton, NY) to schedule their first-time skydives. The Cortisol Reactivity Study tested 78 participants (56 men; age 18–50 years, $\mu_{age} = 25$ years, $s.d._{age} = 8$ years), providing body fat, cortisol and state/trait-anxiety measures. The Cognitive Reactivity Study tested 20 participants (14 men; age 18–48 years, $\mu_{age} = 25$ years, $s.d._{age} = 8$ years), providing body fat, cortisol, state/trait-anxiety, cardiac and cognitive measures. All participants were free of endocrine and cardiac illness, and registered with the skydiving school as never having skydived before. Screening on all participants was performed using a clinical interview; in addition, participants participating in the Cognitive Reactivity Study were also screened by a physician with a full medical history and physical examination. Both of our samples showed a full distribution in terms of body fat percentage, from athletic to obese (Cortisol Reactivity Study: male body fat %: 5.94–27.28, $\mu_{bf\%} = 18.21$, $s.d._{bf\%} = 5.31$; female body fat %: 12.01–46.12, $\mu_{bf\%} = 27.93$, $s.d._{bf\%} = 8.65$; Cognitive Reactivity Study: male body fat %: 4.10–29.05, $\mu_{bf\%} = 14.82$, $s.d._{bf\%} = 8.13$; female body fat %: 17.73–26.48, $\mu_{bf\%} = 22.11$, $s.d._{bf\%} = 6.19$).

Structure and timing of Cortisol Reactivity Study

We provide a schematic of the Cortisol Reactivity Study in Figure 1a. Afternoon skydivers provided salivary samples/state-anxiety data immediately before boarding the plane (15 min pre-jump) and stress salivary samples/state-anxiety data 10 min after landing (15 min post-jump). All participants were tested between 1300 and 1700 hours, a period of time during which diurnal variability for cortisol would be

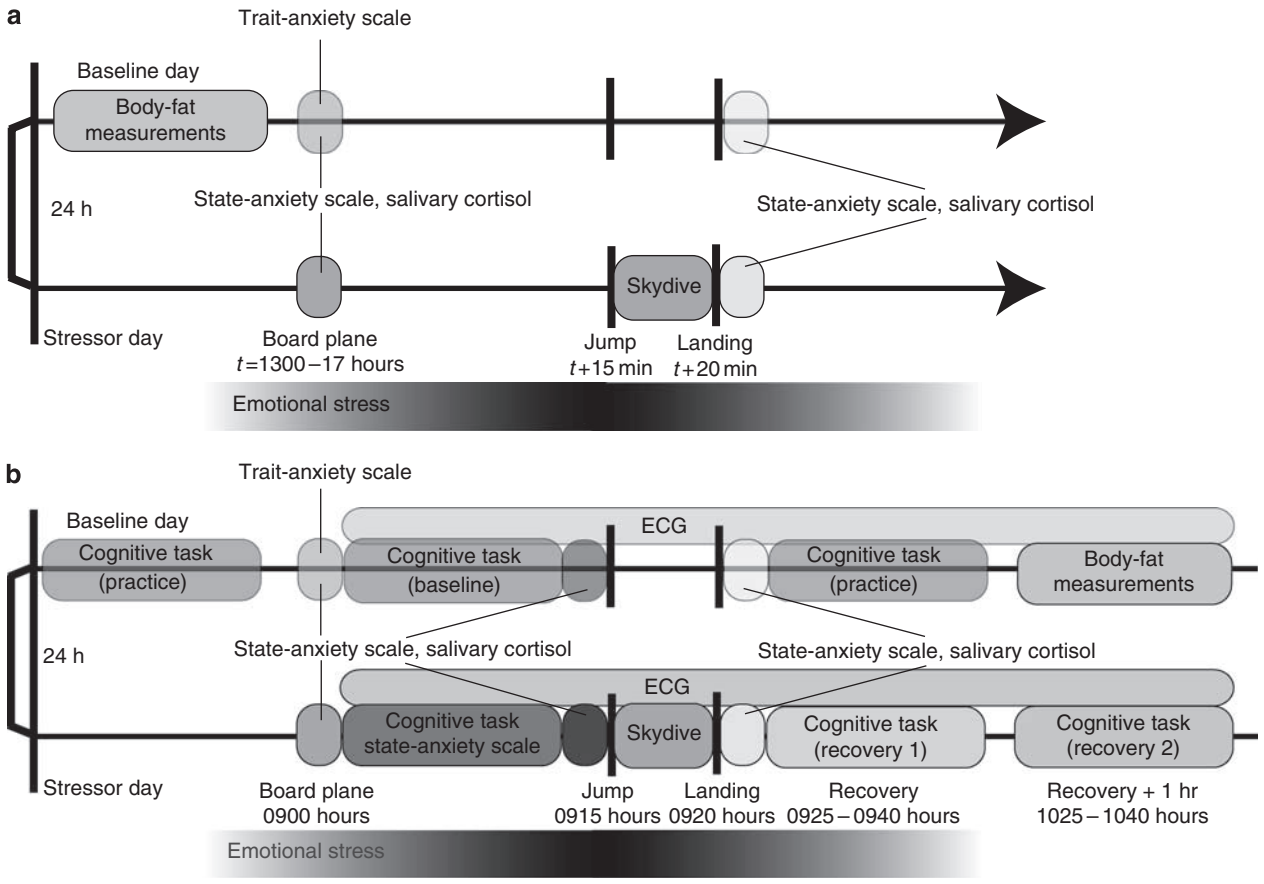

Figure 1 (**a**) Cortisol Reactivity Study. The purpose of this study was to test whether individuals ($N = 78$) with increased body fat percentage also showed increased cortisol reactivity in response to an acute 'real-world' stressor—in this case, a first-time tandem skydive. Individuals participated in 2 days of testing: a 'stressor' day that included the skydive and a 'baseline' day that provided time-matched control measurements. In addition to body fat measurements, participants also provided serial assessments of their state anxiety and salivary cortisol levels before and following the skydive/control. (**b**) Cognitive Reactivity Study. The purpose of this study was to test whether individuals ($N = 20$) with increased body fat percentage also showed greater decline in cognitive performance during an acute 'real-world' stressor—in this case, a first-time tandem skydive. As with the Cortisol Reactivity Study, individuals participated in 2 days of testing: a 'stressor' day that included the skydive and a 'baseline' day that provided time-matched control measurements. In addition to body fat measurements, participants also provided serial assessments of their state anxiety and salivary cortisol levels before and following the skydive/control, as well as completing a cognitive task (Matrix Hidden-Figure Task) before and following the skydive/control. The task was performed multiple times to assess practice effects as well as to ensure complete familiarity before the stressor.

relatively stable over the 20-min time course of the experiment.

Structure and timing of Cognitive Reactivity Study

We provide a schematic of the Cognitive Reactivity Study in Figure 1b. Given the critical role that 24-h circadian rhythms might play in both cortisol production and their potential cognitive effects due to diurnal changes in type 1/type 2 glucocorticoid receptor occupancy,[6] we precisely matched timing between participants on every component of the study, particularly for the morning when cortisol changes most dramatically. All participants had a wake time of 0700 hours, a board time of 0900 hours and a jump time of 0915 hours. Cognitive testing occurred during the 15-min ascent to altitude, and thus the acute stressor was pre-jump anticipatory anxiety rather than in response to the jump itself. Morning skydivers additionally came to the laboratory the day before, to provide baseline cortisol, cognitive and cardiac measurements that were time matched to the stress measurements. For the Cognitive Reactivity Study, skydivers provided salivary samples 24 h before boarding (24 h + 15 min pre-jump) and stress samples 10 min after landing (15 min post-jump). To complement the cognitive data, we analyzed the cardiac data collected while participants completed their cognitive baseline (24 h + 15–0 min pre-jump) and stress (15–0 min pre-jump) tasks.

Body fat measures

Body fat was estimated using the US Navy Circumference Method,[24,25] using measurements taken from the neck, waist, abdomen, hips and height.

Cortisol measures
All salivary cortisol samples were obtained from participants while they were seated, using the passive drool method[26] in which participants allow saliva to collect in their mouths over 2 min and then slowly drain through a straw into a test tube. After collection, the samples were frozen immediately at −30 °F and then subsequently assayed using the Salimetrics Expanded Range High Sensitivity Salivary Cortisol Enzyme Immunoassay Kit.

Subjective report of anxiety
The State–Trait Anxiety Inventory (Mindgarden, Menlo Park, CA, USA) is a well-known and well-validated[27] questionnaire. Forty items assess both chronic (trait) and transient (state) levels of anxiety. To measure subjective perception of the stressor, individuals participating in the Cortisol Reactivity Study filled out the trait and state portions before boarding the plane (15 min before the jump), immediately after landing (1 min post-landing) and on a separate day (baseline) time matched to the jump. Individuals participating in the Cognitive Reactivity Study filled out the trait portion of the State–Trait Anxiety Inventory at baseline (24 h before the jump), as well as the state portion of the State–Trait Anxiety Inventory at baseline (24 h before the jump), immediately before boarding the plane (15 min before the jump), immediately before the jump (1 min before the jump) and three times post-landing (1, 30 and 60 min post-landing).

Cardiac measures
Individuals participating in the Cognitive Reactivity Study wore holter ECGs (Vivometrics Lifeshirt, Ventura, CA, USA) that were attached 1 h before the jump and removed 2 h after the jump. For the analyses presented in this study, we focused on the 15-min ascent to altitude immediately before the jump, a time associated with peak emotional stress. Cardiac data during this period were analyzed for both mean heart rate and autonomic regulation using the well-established power spectrum density method of heart rate variability analysis to quantify sympathetic dominance.[28]

Cognitive measures
To assess the impact of acute stress on cognitive performance, participants completed a laptop-based original test of spatial processing and working memory, the computerized Matrix Hidden-Figure Task (Figure 2), during the 15-min ascent to altitude immediately preceding their jumps. This task is based upon the Hidden-Figure Task first developed by Gottschaldt,[29] requiring that a participant chooses whether or not a simple drawing is 'hidden' within one that is more complex, by detecting a signal within a background of visual noise. Our version modified earlier hidden-figure tasks in several ways: first, it is forced choice to simplify scoring; second, it uses abstract geometric shapes rather than figure drawings to minimize cultural/familiarity bias and third, it introduces noise according to an algorithm that permits a more precise control of task difficulty. The 'matrix' refers to a 9×9 black and white matrix that was used to present the patterns and visual noise. For each presentation of the task, the participant was presented with a series of 96 stimulus pairs. Each stimulus pair presents two shapes, one on the top

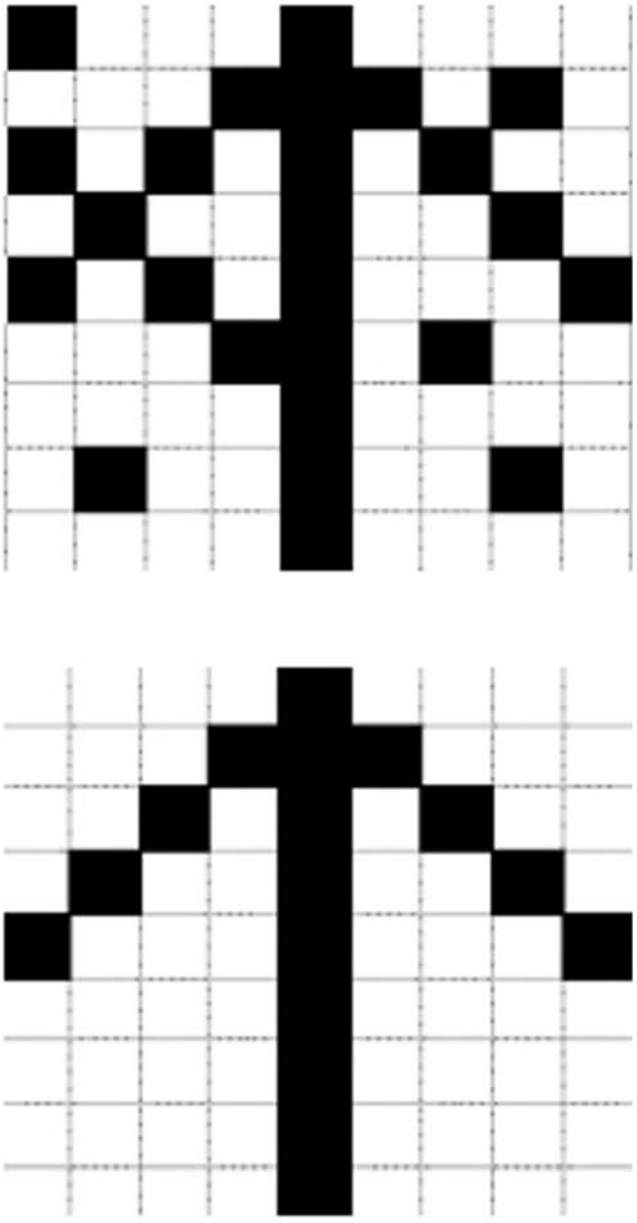

Figure 2 Matrix Hidden-Figure Task. For our cognitive task, we developed a test of spatial processing, selective attention and working memory. In this forced-choice experiment, participants were instructed to determine whether or not the bottom figure was embedded in the top figure, where the top figure included information 'noise.'

of the other, on a white background. The participant was instructed to identify whether the bottom shape is embedded within the top shape by pressing a 'yes' or 'no' button on the response box (Figure 2). The participant's response immediately advanced the task to the next stimulus pair. There were four groups of shapes for the second stimuli: arrows, diamonds, squares and crosses. For each group, there were 24 items: 12 in which the bottom shape was embedded in the top shape and 12 in which the bottom shape was not embedded. Noise increased for each of the 12 levels, with every subsequent level including four more bits of noise than the last (one bit of noise for each quadrant). Items were programmed to present randomly. Although the task was simple and straightforward, participants performed the cognitive task several times before the skydive to assure total familiarity during the stressor: once for practice (24 h + 30–15 min pre-jump), a second time to provide a baseline (24 h + 15–0 min pre-jump), a third time before the stressor (1 h after baseline), a fourth time during the stressor (15–0 min pre-jump), a fifth time immediately after landing (15–30 min post-jump) and a sixth time 30 min after landing (30–45 min post-jump). This task was chosen because it included most components of cognitive function thought to be most affected by stress: spatial processing, selective attention and—as the task requires holding the target image 'online' during search of the noisy image—working memory. To ensure that the cognitive testing environment was free of distractions and as close to identical for all participants as possible, the plane included only the pilot, tandem master and participant for each experiment; all unnecessary conversations between the pilot, tandem master and participant were eliminated during the course of each experiment.

Statistical analyses

All reactivity data measures were calculated by a subtraction of the baseline from the stress conditions. We used both repeated-measures analyses of variance (to assess stress effects over the entire group) as well as partial correlations between body fat and reactivity measures, in both cases controlling for sex as a covariate as body fat distributions are known to be distinct for men and women.

Protection of human participants

We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during this research. This study was approved by the Institutional Review Board of Stony Brook University; all participants provided informed consent.

Results

Validation of the skydive as an acute stressor

As shown in Table 1, the group as a whole showed a marked stress response in anticipation of the skydive, including significant increases in cortisol, heart rate, sympathetic dominance and state anxiety.

Body fat and baseline measures

For the Cortisol Reactivity Study, body fat was positively correlated with baseline state ($r = 0.37$, $P = 0.002$), but not trait, anxiety. For the Cognitive Reactivity Study, body fat was positively correlated with cortisol immediately preceding boarding ($r = 0.56$, $P = 0.007$), but not 24 h before. We found no relationship between body fat and cognitive performance under baseline conditions.

Body fat and stress reactivity

Body fat was significantly correlated with reactivity for cortisol (Cortisol Reactivity Study: $r = 0.33$, $P = 0.003$; Cognitive Reactivity Study: $r = 0.52$, $P = 0.02$) and state anxiety ($r = 0.33$, $P = 0.004$). Body fat was also significantly associated with decline in cognitive performance, for both task accuracy ($r = -0.66$, $P = 0.01$) and response time ($r = -0.62$, $P = 0.02$). Figure 3, which plots the variable body fat and the subtraction of baseline performance from performance during ascent to altitude, indicates that while individuals with less body fat increased performance during the stressor (resulting in positive values for stressor–baseline contrast), participants with more body fat decreased performance during the stressor (resulting in negative values for the stressor–baseline contrast). Using a repeated-measures

Table 1 Mean values for all subjects during baseline and stressor (first-time tandem skydive) for cortisol and cognitive reactivity studies

Variable	μ *(baseline)*	μ *(stressor)*	*F*	P-*value*
Cortisol Reactivity Study (stressor between 1300 and 1700 hours)				
Salivary cortisol (μg per 100 ml)	0.204, s.d. = 0.136	0.504, s.d. = 0.275	117.98	0.000
State anxiety (STAI)	28.29, s.d. = 7.53	40.95, s.d. = 11.08	100.32	0.000
Cognitive Reactivity Study (stressor at 0915 hours)				
Heart rate (b.p.m.)	81.59, s.d. = 12.92	105.76, s.d. = 13.76	70.16	0.000
Sympathetic dominance (heart-rate variability: LF/HF)	5.03, s.d. = 2.74	7.84, s.d. = 4.88	10.21	0.004
Cognitive accuracy	0.94, s.d. = 0.04	0.95, s.d. = 0.04	0.13	0.72
Cognitive response time (ms)	1735, s.d. = 639	1351, s.d. = 434	26.57	0.000

Abbreviations: HF, high frequency (0.15–0.50 Hz); LF, low frequency (0.04–0.15 Hz); STAI, State–Trait Anxiety Inventory.

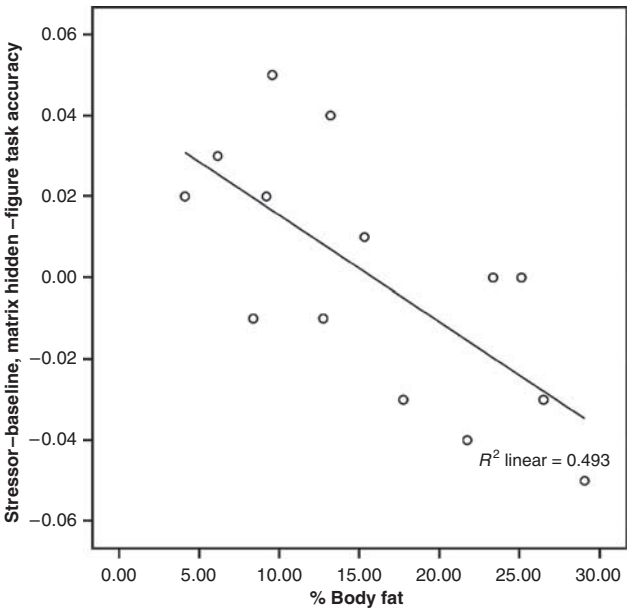

Figure 3 Cognitive reactivity as a function of body fat. Here, cognitive reactivity was defined as subtraction of stressor−baseline values for performance accuracy. Participants with lower body fat increased performance accuracy during the stressor, whereas participants with higher body fat decreased performance accuracy during the stressor.

analysis of variance, with the stress effect on cognition as the within-subject variable and sex as the between-subject variable, we found that arousal affected cognitive performance equivalently for men and for women ($F=0.79$, $P=0.48$). For this analysis, cortisol reactivity was a significant covariate ($F=4.06$, $P=0.04$)—in particular, for accuracy on the cognitive task ($F=8.02$, $P=0.01$). The effect, however, was specific to cortisol and not to generalized arousal: for the same analysis, neither heart-rate reactivity ($F=1.40$, $P=0.33$) nor sympathetic dominance reactivity ($F=0.14$, $P=0.87$) was a significant covariate. Age was not a significant covariate for any of the analyses.

Discussion

This study aimed to answer two questions. First, did laboratory-based experiments indicating that body fat was associated with increased cortisol reactivity continue to hold in response to 'real-world' acute stressors? If so, second, given the critical role that glucocorticoid receptors have been shown to play in cognition (again, mostly in laboratory-based studies with exogenously administered cortisol), did body fat also correlate with decreased cognitive resilience to a 'real-world' acute stressor? Our results suggest that body fat percentage is, in fact, associated with both increased cortisol reactivity as well as decreased cognitive resilience in response to a 'real-world' acute stressor.

The basic and clinical neuroscience research points to cortisol reactivity as the mechanism for our observed cognitive stress vulnerability; this is consistent with our findings, in which participants with increased cortisol also showed decreased performance. However, one should always be cautious in parsing the causal interactions in correlation-based data between neurobiology and behavior in a phenomenon as complex as obesity. For example, one explanation to be considered might be that individuals with low levels of body fat were the more athletic individuals in our sample who had participated in a greater degree in extreme sports and therefore were more habituated to the type of acute stressor presented by the skydive, even if the skydive itself was novel. However, our data do not support this hypothesis. In addition to the State–Trait Anxiety Inventory, participants in the Cognitive Reactivity Study also completed the Sensation-Seeking Scale[30,31] and the Attitudes Towards Risk Questionnaire.[32] Participants' ratings on the scales were uncorrelated with body fat (sensation seeking: $r=-0.02$, $P=0.92$; taking risks: $r=0.06$, $P=0.70$; thinking about risks: $r=-0.03$, $P=0.86$), which suggests that sensation-seeking or risk-seeking behavior between individuals who were more versus less lean was not appreciably different.

Another important question is whether increased body fat is the cause of stress reactivity or rather its consequence. In the evolutionary environment, emotional stress was normally associated with the concrete need for a fight or flight response, and both fighting and fleeing require immediate energy expenditure. In this context, it is therefore easy to see the connection between emotional stress and cortisol release, as cortisol is involved in converting energy to a form in which it can be easily accessed for physical expenditure through gluconeogenesis and lipolysis[33] as well as stimulating appetite to increase energy reserves. For example, in one study, researchers showed that once prednisone was given to cancer patients, their appetites increased dramatically;[34] in another study, a group of healthy men were administered cortisol over a period of 4 days, during which time the cortisol gave them increased energy but also voracious appetites.[35] Macronutrient selection is also altered; women have been shown to prefer high-calorie fatty foods when exposed to emotional stress.[36] In the modern environment, in which emotional stress is not paired with the massive caloric expenditures associated with fighting and fleeing, but for which physiology still prepares during stress as if it were, it makes sense that a tendency toward cortisol hyper-reactivity to stress would lead both to cognitive effects as well as increased body fat. Therefore, upon this model, increased body fat is not the cause of cortisol reactivity and therefore cognitive effects, but rather the consequence in conjunction with the cognitive effects of a common cause: pre-existing cortisol hyper-reactivity. It may also be the case, however, that the relationship between cortisol reactivity and body fat is self-reinforcing: individuals with greater body fat may also show

an increase of their cortisol reactivity, as increased mass is associated with greater caloric needs and therefore may also trigger increased cortisol release to mobilize glucose to meet those caloric needs.

The relationship between cognition and emotional stress is also complex, as emotional stress is not always deleterious to cognitive performance, but depends upon both the time of day (and therefore associated type 1/type 2 glucocorticoid receptor activation ratios[6]) and the type of cognitive task (acquisition versus retrieval, declarative versus working memory), and whether the cognitive task is related to the stressor or unrelated to it. Cognition and stress are known to be related according to an inverted U-type relationship in which a mild arousal is actually beneficial to cognitive performance, whereas a more severe arousal is deleterious.[37] According to Lupien *et al.*'s[20] research, it appears that this pattern is directly associated with the amount of cortisol produced for mild versus severe arousal: stressors experienced in the morning (when baseline cortisol levels and type 1/type 2 activation levels are highest) affect cognition more negatively than stressors experienced in the late afternoon (when baseline cortisol levels and type 1/type 2 activation levels are lowest); as such, our morning testing was deliberately timed to take advantage of the enhanced stress effects on cognition.

The cognitive task itself was unrelated to the stressor, which is an asset when one considers that in modern times most cognitive tasks performed during emotional stress are actually not directly related to approach/avoidance with respect to the stressor itself. Emotional arousal primes the organism for perceived danger by increasing the orienting response, which permits the organism to find and focus on the source of danger. Once oriented to the source of danger, emotional arousal strengthens attention to the source of danger and diminishes attention to irrelevant stimuli, narrowing the amount of peripheral information simultaneously accessible with the target. This two-pronged strategy has both costs and benefits: breadth of cognition is limited, with the individual attending to less information at a time, but is more flexible in terms of the ability to switch attention from one target to another. Under most dangerous conditions in our evolutionary past, these costs and benefits were appropriate for survival: in the presence of a predator, it is adaptive to focus on the predator, to ignore peripheral information and to be able to quickly switch attention between two or more predators that together present a collective threat. Although the cognitive changes associated with arousal in humans are appropriate for predator/prey contexts, most emotional states linked to arousal in modern societies (for example, fear, stress and anxiety) occur under far different circumstances, in which the source of arousal is often not a concrete entity to which one can readily orient. Even individuals in dangerous operational environments, such as tactical aviators in combat, protect themselves by defying their instincts: a fighter pilot needs to attend not only to the enemy 'predator', but equally to the myriad sources of information prerequisite to keep his aircraft aloft and his artillery engaged. Thus, although emotional arousal can, under certain circumstances, benefit cognitive performance by increasing focused attention on a target and decreasing attention to irrelevant information, it can just as easily degrade cognitive performance by triggering the orienting response in the absence of a specific target and by disregarding potentially relevant peripheral stimuli (that is, tunnel vision).

The cognitive results are even more intriguing when one considers that the body fat-associated stress effects occurred in spite of prior training. As our study was designed to investigate individual variability, it was critical to provide conditions as close to identical as possible for all participants; therefore, the timing of each condition was kept constant rather than counter-balancing for order. Our preliminary analysis of this sample, performing the same task over six trials, indicated strong training effects (repeated-measures analysis of variance: $F = 33.94$, d.f. $= 1$, $P = 0.000$); a pair-wise comparison showed significant ($P < 0.01$) increases in performance with each additional trial, stabilizing after the fourth trial. As overall performance for the group increased with repeated practice, our analyses indicate that decline in performance between the second (baseline) and fourth (stress) trials observed in participants with greater body fat was due, specifically, to increased stress reactivity in spite of prior familiarity with the task.

Although obesity is a disease state, the approach taken in this study suggests that the endocrine and cognitive components associated with obesity actually exist along a continuum, in which even small increases of body fat show deleterious effects on stress resilience. Our results indicate that, even among a population of healthy adults of predominately normal weight, body fat has tangible implications for resilience to an acute stressor, in terms of both subjective perception of stress and its cognitive effects. The dissociation of cardiac changes support earlier experimental study specifically tying cognitive stress effects to cortisol, rather than a more general stress response, and indicates that—in our population—the effects were specific to the acute stress response rather than occurring under baseline conditions. This study has direct implications for understanding the role of cognitive control with respect to behavior modification, including cognitive behavioral therapy, in the treatment of obesity,[38–44] and in understanding how the obese may differ cognitively from the healthy population in the application of these techniques under acute stress. Future studies, using larger participant sample sizes, more rigorous body fat indices, tasks capable of differentiating between specific cognitive functions and targeting more directly the obese population, will further enhance our understanding of the relationship between endocrine and cognitive stress resilience as a function of body fat, and will also explore the implications of these studies with respect to chronic, rather than acute, real-world stressors.

Acknowledgements

This research was supported by funding from the Office of Naval Research N0014-04-1-005 (LRMP), the US Army Soldier Systems Center Natick DAAD16-99-C-1033 (LRMP) and the National Institutes of Health 5-MO1-RR-10710 (Stony Brook University General Clinical Research Center).

References

1 Bremner JD, Randall P, Scott TM, Bronen RA, Seibyl JP, Southwick SM *et al*. MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *Am J Psychiatry* 1995; **152**: 973–981.
2 Lupien SJ, de Leon M, de Santi S, Convit A, Tarshish C, Nair NP *et al*. Cortisol levels during human aging predict hippocampal atrophy and memory deficits. *Nat Neurosci* 1998; **1**: 69–73.
3 Wellman CL. Dendritic reorganization in pyramidal neurons in medial prefrontal cortex after chronic corticosterone administration. *J Neurobiol* 2001; **49**: 245–253.
4 Kirschbaum C, Wolf OT, May M, Wippich W, Hellhammer DH. Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. *Life Sci* 1996; **58**: 1475–1483.
5 Newcomer JW, Selke G, Melson AK, Hershey T, Craft S, Richards K *et al*. Decreased memory performance in healthy humans induced by stress-level cortisol treatment. *Arch Gen Psychiatry* 1999; **56**: 527–533.
6 de Kloet ER, Oitzl MS, Joels M. Stress and cognition: are corticosteroids good or bad guys? *Trends Neurosci* 1999; **22**: 422–426.
7 Lupien SJ, McEwen BS. The acute effects of corticosteroids on cognition: integration of animal and human model studies. *Brain Res Brain Res Rev* 1997; **24**: 1–27.
8 Roozendaal B. Stress and memory: opposing effects of glucocorticoids on memory consolidation and memory retrieval. *Neurobiol Learn Mem* 2002; **78**: 578–595.
9 Cahill L, Gorski L, Le K. Enhanced human memory consolidation with post-learning stress: interaction with the degree of arousal at encoding. *Learn Mem* 2003; **10**: 270–274.
10 de Quervain DJ, Roozendaal B, McGaugh JL. Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature* 1998; **394**: 787–790.
11 Roozendaal B. 1999 Curt P. Richter award. Glucocorticoids and the regulation of memory consolidation. *Psychoneuroendocrinology* 2000; **25**: 213–238.
12 Bjorntorp P. Body fat distribution, insulin resistance, and metabolic diseases. *Nutrition* 1997; **13**: 795–803.
13 Epel ES, McEwen B, Seeman T, Matthews K, Castellazzo G, Brownell KD *et al*. Stress and body shape: stress-induced cortisol secretion is consistently greater among women with central fat. *Psychosom Med* 2000; **62**: 623–632.
14 Pedersen SB, Jonler M, Richelsen B. Characterization of regional and gender differences in glucocorticoid receptors and lipoprotein lipase activity in human adipose tissue. *J Clin Endocrinol Metab* 1994; **78**: 1354–1359.
15 Starkman MN, Gebarski SS, Berent S, Schteingart DE. Hippocampal formation volume, memory dysfunction, and cortisol levels in patients with Cushing's syndrome. *Biol Psychiatry* 1992; **32**: 756–765.
16 Lapidus L, Bengtsson C, Hallstrom T, Bjorntorp P. Obesity, adipose tissue distribution and health in women—results from a population study in Gothenburg, Sweden. *Appetite* 1989; **13**: 25–35.
17 Lloyd CE, Wing RR, Orchard TJ. Waist to hip ratio and psychosocial factors in adults with insulin-dependent diabetes mellitus: the Pittsburgh Epidemiology of Diabetes Complications study. *Metab Clin Exp* 1996; **45**: 268–272.
18 Rosmond R, Lapidus L, Marin P, Bjorntorp P. Mental distress, obesity and body fat distribution in middle-aged men. *Obes Res* 1996; **4**: 245–252.
19 Wing RR, Matthews KA, Kuller LH, Meilahn EN, Plantinga P. Waist to hip ratio in middle-aged women. Associations with behavioral and psychosocial factors and with changes in cardiovascular risk factors. *Arterioscler Thromb* 1991; **11**: 1250–1257.
20 Lupien SJ, Maheu F, Tu M, Fiocco A, Schramek TE. The effects of stress and stress hormones on human cognition: implications for the field of brain and cognition. *Brain Cogn* 2007; **65**: 209–237.
21 Fiocco AJ, Joober R, Lupien SJ. Education modulates cortisol reactivity to the Trier Social Stress Test in middle-aged adults. *Psychoneuroendocrinology* 2007; **32**: 1158–1163.
22 Rimmele U, Zellweger BC, Marti B, Seiler R, Mohiyeddini C, Ehlert U *et al*. Trained men show lower cortisol, heart rate and psychological responses to psychosocial stress compared with untrained men. *Psychoneuroendocrinology* 2007; **32**: 627–635.
23 Tyrka AR, Wier LM, Anderson GM, Wilkinson CW, Price LH, Carpenter LL. Temperament and response to the Trier Social Stress Test. *Acta psychiatrica Scandinavica* 2007; **115**: 395–402.
24 Conway TL, Cronan TA, Peterson KA. Circumference-estimated percent body fat vs weight-height indices: relationships to physical fitness. *Aviat Space Environ Med* 1989; **60**: 433–437.
25 Babcock CJ, Kirby TE, McCarroll ML, Devor ST. A comparison of military circumference equations to skinfold-based equations to estimate body composition. *Mil Med* 2006; **171**: 60–63.
26 Gallagher P, Leitch MM, Massey AE, McAllister-Williams RH, Young AH. Assessing cortisol and dehydroepiandrosterone (DHEA) in saliva: effects of collection method. *J Psychopharmacol* 2006; **20**: 643–649.
27 Knight RG, Waal-Manning HJ, Spears GF. Some norms and reliability data for the State—Trait Anxiety Inventory and the Zung Self-Rating Depression scale. *Br J Clin Psychol* 1983; **22** (Part 4): 245–249.
28 Mujica-Parodi LR, Korgaonkar M, Ravindranath B, Greenberg T, Tomasi D, Wagshul M *et al*. Limbic dysregulation is associated with lowered heart rate variability and increased trait anxiety in healthy adults. *Hum Brain Mapp* 2007 November 27. [Epub ahead of print].
29 Gottschaldt K. Über den Einfluss der Erfahrung auf die Wahmehmung von Figuren II. *Psychol Forsch* 1929; **12**: 1–87.
30 Zuckerman M, Link K. Construct validity for the sensation-seeking scale. *J Consult Clin Psychol* 1968; **32**: 420–426.
31 Zuckerman M. *Behavioural Expressions and Biosocial Bases of Sensation Seeking*. Cambridge University Press: Cambridge, 1994.
32 Franken RE, Gibson KJ, Rowland GL. Sensation Seeking and the tendency to view the world as threatening. *Pers Individ Diff* 1992; **13**: 31–38.
33 Bray GA. Autonomic and endocrine factors in the regulation of food intake. *Brain Res Bull* 1985; **14**: 505–510.
34 Willox JC, Corr J, Shaw J, Richardson M, Calman KC, Drennan M. Prednisolone as an appetite stimulant in patients with cancer. *BMJ (Clin Res Ed)* 1984; **288**: 27.
35 Tataranni PA, Larson DE, Snitker S, Young JB, Flatt JP, Ravussin E. Effects of glucocorticoids on energy metabolism and food intake in humans. *Am J Physiol* 1996; **271**: E317–E325.
36 Grunberg NE, Straub RO. The role of gender and taste class in the effects of stress on eating. *Health Psychol* 1992; **11**: 97–100.
37 Yerkes RM, Dodson JD. The relation of strength of stimulus to rapidity of habit-formation. *J Comp Neurol Psychol* 1908; **18**: 459–482.
38 Cresci B, Tesi F, La Ferlita T, Ricca V, Ravaldi C, Rotella CM *et al*. Group versus individual cognitive-behavioral treatment for obesity: results after 36 months. *Eat Weight Disord* 2007; **12**: 147–153.
39 Butryn ML, Phelan S, Hill JO, Wing RR. Consistent self-monitoring of weight: a key component of successful weight loss maintenance. *Obesity (Silver Spring)* 2007; **15**: 3091–3096.
40 Forman EM, Hoffman KL, McGrath KB, Herbert JD, Brandsma LL, Lowe MR. A comparison of acceptance- and control-based

strategies for coping with food cravings: an analog study. *Behav Res Ther* 2007; **45**: 2372–2386.
41 Stahre L, Tarnell B, Hakanson CE, Hallstrom T. A randomized controlled trial of two weight-reducing short-term group treatment programs for obesity with an 18-month follow-up. *Int J Behav Med* 2007; **14**: 48–55.
42 Munsch S, Biedert E, Meyer A, Michael T, Schlup B, Tuch A *et al*. A randomized comparison of cognitive behavioral therapy and behavioral weight loss treatment for overweight individuals with binge eating disorder. *Int J Eat Disord* 2007; **40**: 102–113.
43 Nasser JA, Gluck ME, Geliebter A. Impulsivity and test meal intake in obese binge eating women. *Appetite* 2004; **43**: 303–307.
44 Melchionda N, Besteghi L, Di Domizio S, Pasqui F, Nuccitelli C, Migliorini S *et al*. Cognitive behavioural therapy for obesity: one-year follow-up in a clinical setting. *Eat Weight Disord* 2003; **8**: 188–193.