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Body Fat is Associated with Decreased Endocrine and Cognitive Resilience to Acute Emotional Stress

Running head: Body fat and emotional stress resilience

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Abstract and Keywords

Objective: Cortisol is elevated both in individuals with increased emotional stress as well as with 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 subjects undergoing their first (tandem) skydive. In the first sample (N=78), subjects were tested for salivary cortisol and state-anxiety (Spielberger State Anxiety Scale) during the plane's fifteen-minute ascent to altitude in immediate anticipation of the jump. In a second sample (N=20), subjects were tested for salivary cortisol, as well as cardiac variables (heart rate, autonomic regulation via 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.

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 (HPA) axis response to emotional and physical stress. The impact of stress on cognitive performance appears to be primarily mediated via 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), as well as 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).

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 (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 via self-report. In order 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 subjects.

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 previous laboratory findings do in fact extend to stressors in the "real-world." Second, since 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 subjects' 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 since, unlike solo jumps, they require less than five minutes 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 subjects—a critical feature for any study of individual variability. Subjects wore digital altimeters (Altimaster Neptune), confirming consistent rise-times of 15 minutes, jumps at 4km (13,000 ft), freefall lasting one minute, and parachuting for an additional four

minutes before landing. Upon landing, all subjects reported that their emotional stress peaked at the aircraft door, shortly before exiting the aircraft; thus, we treated the 15 minute anticipatory anxiety during ascent to altitude and prior to 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. Since testing subjects 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 subjects to address the first question (*Cortisol Reactivity Study*), as well as to separately conduct a more complex study with fewer subjects to address the second question (*Cognitive Reactivity Study*).

Subjects: Subjects were recruited in two batches from individuals that contacted a local skydiving school (Skydive Long Island, Calverton NY) to schedule their first-time skydives. The Cortisol Reactivity Study tested 78 subjects (56 males; ages 18-50 yrs, μ_{age}= 25 yrs, s.d.$_{age}$=8 yrs), providing body fat, cortisol, and state/trait-anxiety measures. The Cognitive Reactivity Study tested 20 subjects (14 males; ages 18-48, μ_{age}= 25, s.d.$_{age}$=8), providing body fat, cortisol, state/trait-anxiety, cardiac, and cognitive measures. All subjects were free of endocrine and cardiac illness, and registered with the skydiving school as never having skydived before. Screening on all subjects was performed using a clinical interview; in addition, subjects participating in the Cognitive Reactivity Study also were 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 prior to boarding the plane (15 minutes pre-jump) and stress salivary samples/state-anxiety data ten minutes after landing (15 minutes post-jump). All subjects were tested between 1pm and 5pm, a period of time during which diurnal variability for cortisol would be relatively stable over the 20-minute 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-hour circadian rhythms might play in both cortisol production and their potential cognitive effects due to diurnal changes in Type I/Type II glucocorticoid receptor occupancy (6), we precisely matched timing between subjects on every component of the study, particularly for the morning when cortisol changes most dramatically. All subjects had a wake-time of 7:00am, board-time of 9:00am, and jump-time of 9:15am. Cognitive testing occurred during the 15-minute 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-hours prior to boarding (24 hours + 15 minutes pre-jump) and stress samples 10 minutes after landing (15 minutes post-jump). To complement the cognitive data, we analyzed the cardiac data collected while subjects completed their cognitive baseline (24 hours + 15-0 minutes pre-jump) and stress (15-0 minutes pre-jump) tasks.

Body fat Measures: Body fat was estimated using the U.S. Navy Circumference Method (24, 25), using measurements taken from the neck, waist, abdomen, hips, and height.

Cortisol Measures: All cortisol samples were obtained from saliva using the passive drool method (26).

Subjective Report of Anxiety: The State-Trait Anxiety Inventory (STAI; Mindgarden, Menlo Park CA) 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, subjects participating in the Cortisol Reactivity Study filled out the trait and state portions prior to boarding the plane (15 minutes prior to the jump), immediately after landing (one minute post-landing), and on a separate day (baseline) time-matched to the jump. Subjects participating in the Cognitive Reactivity Study filled out the trait portion of the STAI at baseline (24 hours prior to the jump), as well as the state portion of the STAI at baseline (24 hours prior to the jump), immediately prior to boarding the plane (15 minutes prior to the jump), immediately prior to the jump (one minute prior to the jump), and three times post-landing (one, 30, and 60 minutes post-landing).

Cardiac Measures: Subjects participating in the Cognitive Reactivity Study wore holter ECGs (Vivometrics Lifeshirt, Ventura CA) that were attached one hour before the jump and removed two hours after the jump. For the analyses presented in this article, we focused on the fifteen-minute ascent to altitude immediately prior to 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, subjects completed a laptop-based original test of spatial processing and working memory, the computerized Matrix Hidden Figure Task (Figure 2), during the 15-minute ascent to altitude immediately preceding their jumps. This task is based upon the Hidden-Figure Task first developed by Gottschaldt (29), requiring that a subject choose 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/familitarity bias, and third, it introduces noise according to an algorithm that permits more precise control of task-difficulty. The "matrix" refers to a 9x9 black and white matrix, which was used to present the patterns and visual noise. For each presentation of the

task, the subject was presented with a series of 96 stimulus pairs. Each stimulus pair presents two shapes, one on top of the other, on a white background. The subject 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 subject's response immediately advanced the task to the next stimulus pair. There were 4 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 12 levels, with every subsequent level including 4 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, subjects performed the cognitive task several times before the skydive to assure total familiarity during the stressor: once for practice (24 hours + 30-15 minutes pre-jump), a second time to provide a baseline (24 hours + 15-0 minutes pre-jump), a third time before the stressor (one hour after baseline), a fourth time during the stressor (15-0 minutes pre-jump), a fifth time immediately after landing (15-30 minutes post-jump), and a sixth time 30 minutes after landing (30-45 minutes 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—since the task requires holding the target image "on line" 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 subjects as possible, the plane included only the pilot, tandem-master, and subject for each experiment; all unnecessary conversations between the pilot, tandem-master, and subject 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, controlling for sex since body fat distributions are known to be distinct for men and women.

Protection of Human Subjects: 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 subjects provided informed consent.

Results

Validation of the Skydive as an Acute Stressor: The group as a whole showed a marked stress response in anticipation of the skydive, including increases in cortisol ($F=117.98$, $df=1$, $p=0.000$), heart rate ($F=64.013$, $df=1$, $p=0.000$), sympathetic dominance ($F=4.082$, $df=1$, $p=0.05$), and state-anxiety ($F=8.45$, $df=1$, $p=0.005$).

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-hours prior. 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: 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 variables *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), subjects with more body fat *decreased* performance during the stressor (resulting in negative values for the stressor–baseline contrast). Longer response-times to this task were significantly correlated with increased cortisol levels both immediately at landing (r=0.62, p=0.02) as well as half an hour post-landing (r=0.57, p=0.03). These results were robust after controlling for sex (*accuracy*: r=–0.66, p=0.01; *response-time*: r=–0.62, p=0.02). Body fat was not significantly correlated with the cardiac response, either for heart rate (r=0.55, p=0.10) or sympathetic dominance (r=0.18, p=0.62). Age was not a significant covariate for 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? And 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 subjects 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 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 STAI, subjects in the Cognitive Reactivity Study also completed the Sensation-Seeking Scale (30, 31) and the Attitudes Towards Risk Questionnaire (32). Subjects' 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, since cortisol is involved in converting energy to a form in which it can be easy accessed for physical expenditure via gluconeogenesis and lipolysis (33) as well as stimulating appetite in order 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 four 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 towards 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 their cortisol reactivity, since increased mass is associated with greater caloric needs and therefore may also trigger increased cortisol release in order to mobilize glucose to meet those caloric needs.

The relationship between cognition and emotional stress is also complex, since emotional stress is not always deleterious to cognitive performance, but depends upon both the time of day (and therefore associated Type I/Type II glucocorticoid receptor activation ratios (6)) as well as 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 mild arousal is actually beneficial to cognitive performance, while 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 vs. severe arousal: stressors experienced in the morning (when baseline cortisol levels and Type I/Type II activation levels are highest) more negatively affect cognition than stressors experienced in the late afternoon (when baseline cortisol levels and Type I/Type II 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. While the cognitive changes associated with arousal in humans are appropriate for predator/prey contexts, most emotional states linked to arousal in modern societies (e.g., fear, stress, 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, while 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 (i.e., 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. Since our study was designed to investigate individual variability, it was critical to provide as close to identical conditions for all subjects; 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 ANOVA: $F=33.94$, $df=1$, $p=0.000$); a pair-wise comparison showed significant ($p<0.01$) increases in performance with each additional trial, stabilizing after the fourth trial. Since 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 subjects with greater body fat was due specifically to increased stress-reactivity in spite of prior familiarity with the task.

While 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, both in terms of subjective perception of stress, as well as its cognitive effects. The dissociation of cardiac changes support previous experimental work specifically tying cognitive stress effects to cortisol, rather than a more general stress response, and indicate that—in our population—the effects were specific to the acute stress response rather than occurring under baseline conditions. This work 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 work, using larger subject 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 this work with respect to chronic, rather than acute, real-world stressors.

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Tables

None.

Titles and Legends to Figures

Figure 1a: 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. Subjects participated in two days of testing: a "stressor" day, which included the skydive, and a "baseline" day, which provided time-matched control measurements. In addition to body fat measurements, subjects also provided serial assessments of their state anxiety and salivary cortisol levels prior to and following the skydive/control.

Figure 1b: Cognitive Reactivity Study

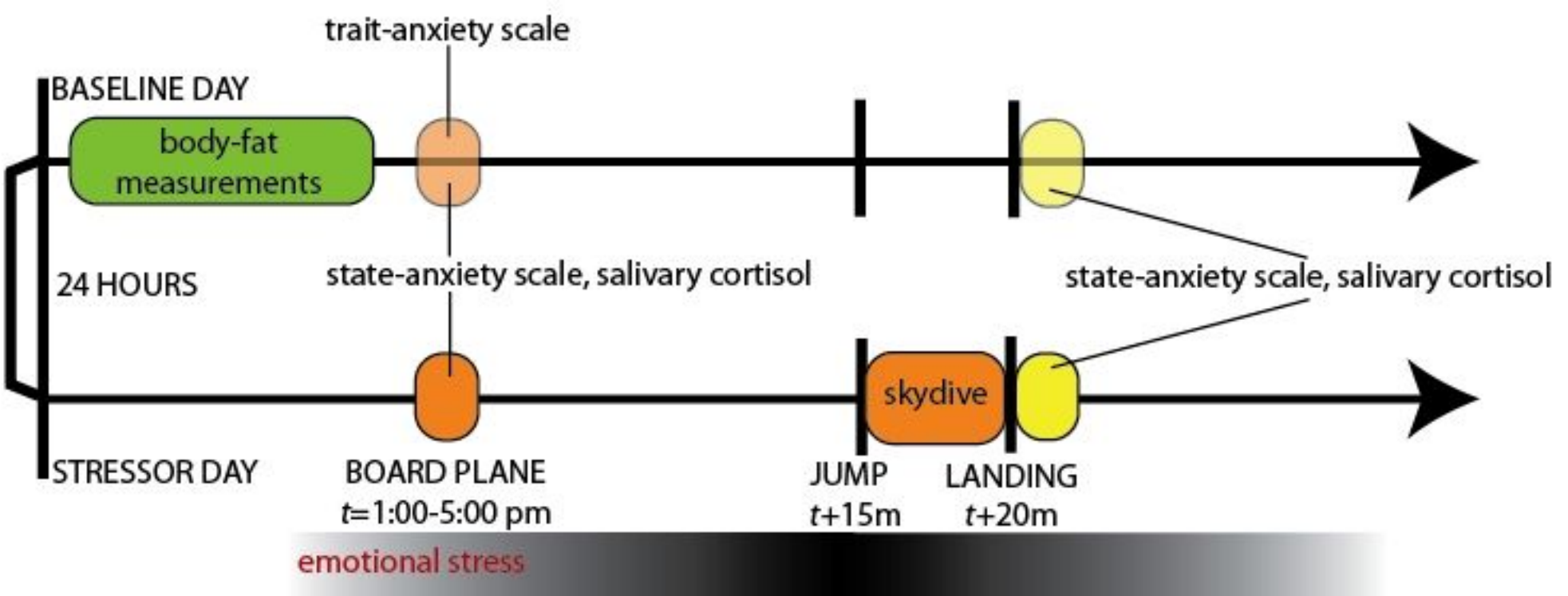
The purpose of this study was to test whether individuals (N=20) with increased body fat percentage also showed greater decline in cognitive performance during an acute "real world" stressor—in this case, a first-time tandem skydive. As with the Cortisol Reactivity Study, subjects participated in two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

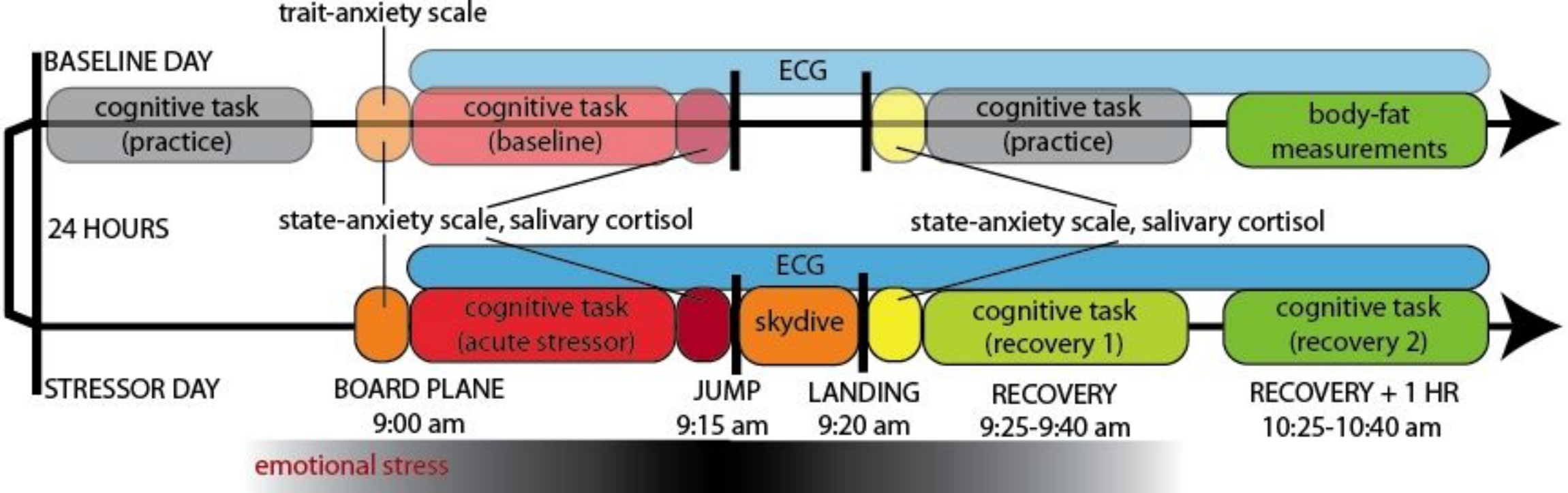
Figure 2: Matrix Hidden Figure Task

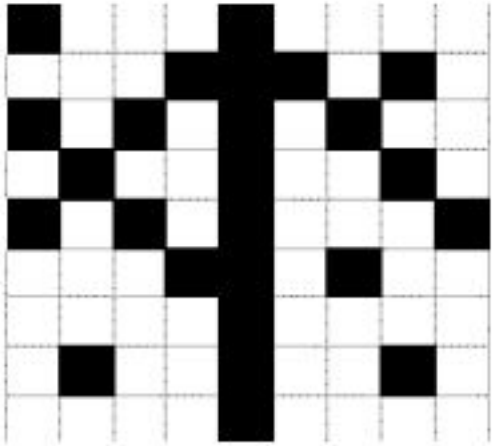
For our cognitive task we developed a test of spatial processing, selective attention, and working memory. In this forced-choice experiment, subjects were instructed to determine whether or not the bottom figure was embedded in the top figure, where the top figure included information "noise."

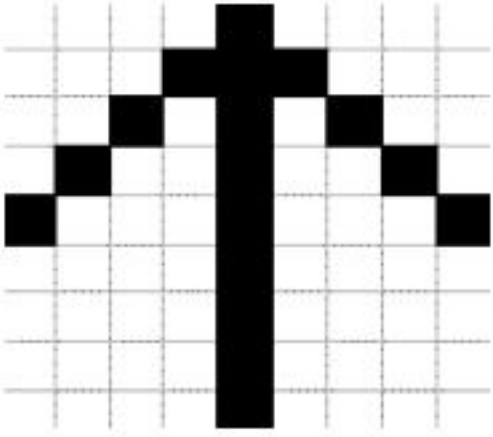
Figure 3: Cognitive Reactivity as a Function of Body Fat

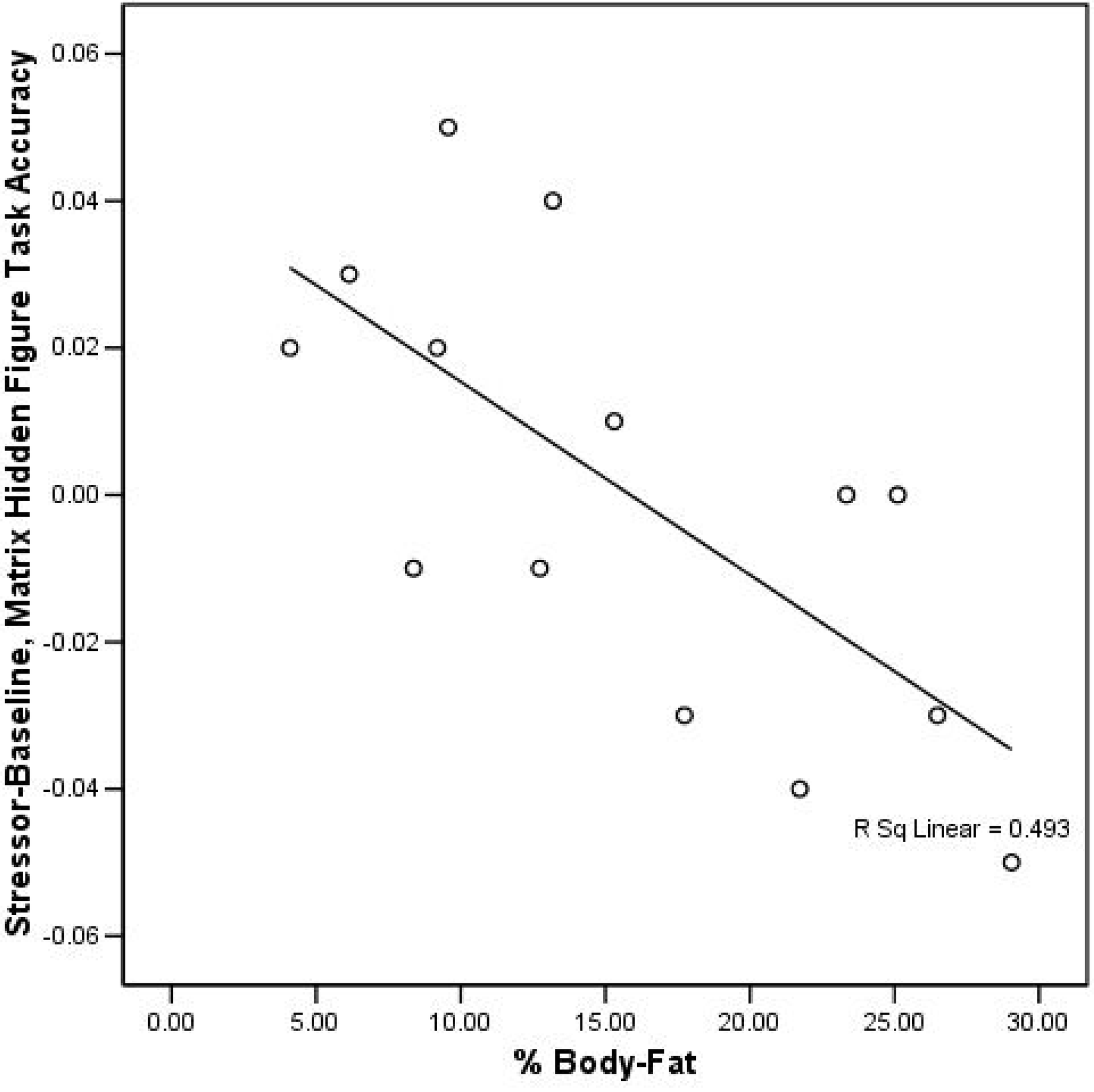
Here, cognitive reactivity was defined as subtraction of stressor–baseline values for performance accuracy. Subjects with lower body fat increased performance accuracy during the stressor, while subjects with higher body fat decreased performance accuracy during the stressor.


trait-anxiety scale
BASELINE DAY
body-fat measurements
24 HOURS
state-anxiety scale, salivary cortisol
state-anxiety scale, salivary cortisol
skydive
STRESSOR DAY
BOARD PLANE
t=1:00-5:00 pm
JUMP
t+15m
LANDING
t+20m
emotional stress


trait-anxiety scale
BASELINE DAY
ECG
cognitive task (practice)
cognitive task (baseline)
cognitive task (practice)
body-fat measurements
24 HOURS
state-anxiety scale, salivary cortisol
state-anxiety scale, salivary cortisol
ECG
cognitive task (acute stressor)
skydive
cognitive task (recovery 1)
cognitive task (recovery 2)
STRESSOR DAY
BOARD PLANE 9:00 am
JUMP 9:15 am
LANDING 9:20 am
RECOVERY 9:25-9:40 am
RECOVERY + 1 HR 10:25-10:40 am
emotional stress






Stressor-Baseline, Matrix Hidden Figure Task Accuracy
0.06
0.04
0.02
0.00
-0.02
-0.04
-0.06
R Sq Linear = 0.493
0.00
5.00
10.00
15.00
20.00
25.00
30.00
% Body-Fat