

Glucose but Not Protein or Fat Load Amplifies the Cortisol Response to Psychosocial Stress

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Received September 1, 2001; revised November 10, 2001;
accepted November 16, 2001

We previously reported that glucose intake amplifies cortisol response to psychosocial stress and smoking in healthy young men, while low blood glucose levels prevented the stress-induced activation of the hypothalamus pituitary adrenal (HPA) axis. However, it remains unknown whether this modulation is specific for glucose load or a more common effect of energy availability. To elucidate this question, 37 healthy men, who fasted for at least 8 h before the experiment, were randomly assigned to four experimental groups, who received glucose ($n = 8$), protein ($n = 10$), fat ($n = 10$), and water ($n = 9$), one h before their exposure to the Trier Social Stress Test (TSST). Blood glucose levels were measured at baseline and following stress, while salivary cortisol was assessed repeatedly measured before after the TSST. The results show that both absolute cortisol levels and net cortisol increase were greater in the glucose group in comparison to the other groups ($F_{3,33} = 3.00$, $P < 0.05$ and $F_{3,33} = 3.08$, $P < 0.05$, respectively). No group differences were observed with respect to perceived stress and mood. Furthermore, the cortisol response was positively correlated with blood glucose changes ($r = 0.49$, $P < 0.002$). In conclusion, the results suggest a central mechanism responsible for regulation of energy balance and HPA axis activation, rather than peripheral mechanisms. We thus recommend controlling for blood glucose levels when studying HPA axis responsiveness.

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Key Words: stress; glucose; calories; saliva; hormones; human; lab stressor.

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INTRODUCTION

HPA axis activity is associated with systems responsible for caloric flow in the organism (Akana, Strack, Hanson, and Dallman, 1994; Dallman, Akana, Strack, Hanson, and Sebastian, 1995; Dallman, Strack, Akana, Bradbury, Hanson, Scribner, and Smith, 1993; Tempel and Leibowitz, 1993). Hypoglycemic states after five days of fasting have been shown to increase cortisol secretion (Bergendahl, Iranmanesh, Evans, and Veldhuis, 2000; Bergendahl, Vance, Iranmanesh, Thorner, and Veldhuis, 1996) and alter pulsatile cortisol release (Vance and Thorner, 1989) in humans. On the other hand, caloric intake modulates cortisol secretion, since diurnal cortisol peaks are related to meals (Follenius, Brandenberger, and Hietter, 1982; Rosmond, Holm, and Bjorntorp, 2000). The fact that both fed and fasted states enhance the activity of the HPA axis, suggests different underlying mechanisms and/or timing in the interaction between caloric flow and profile of cortisol levels. In rats with a nocturnal cycle of activity, animals fed *ad libitum* during night showed lower basal activity of the adrenocortical system and a higher stress responsiveness in the morning than overnight fasted rats, which exhibited high basal corticosteroid levels and decreased responsiveness to stress (Choi, Horsley, Aguila, and Dallman, 1996).

In a previous study we could show that decreased energy availability after an eight-h fasting period in the morning, led to a blunted cortisol response to psychosocial stress and pharmacological stimulation, which could be restored by glucose administration (Kirschbaum, Gonzalez Bono, Rohleder, Gessner, Pirke, Salvador, and Hellhammer, 1997). However, the

mechanism involved in this modulation of the stress-response remained unclear.

Several mechanisms are discussed to explain these results: On the one hand, glucose-specific or central mechanisms may account for modulation of the HPA axis response by caloric load. One possible explanation could be the stimulatory action of enhanced serotonin synthesis on the HPA axis. In nondiabetic subjects, glucose load produces an increase of insulin that, in turn, favors tryptophan transport into the central nervous system, which in turn increases serotonin synthesis. Choi *et al.* have proposed another mechanism for regulation at the hypothalamic level: high glucose and insulin levels, which are seen shortly after glucose load stimulate activity of the ventromedial nuclei (VMN). Activity of the VMN seems to be an important permissive input to the paraventricular nuclei (PVN), which mediate HPA activation, since inhibition of the VMN by colchicine is able to disrupt HPA axis responsiveness to fasting (Choi *et al.*, 1996; ter Horst and Luiten, 1986). Low glucose levels may also inhibit the VMN and consequently the PVN, and thereby mediate the attenuated HPA axis response.

On the other hand, if nonspecific or peripheral mechanisms were involved, the citric acid cycle as an energy provider to the organism could be an important candidate to modulate HPA responsiveness. In an acutely stressed organism, oxidative processes enhance ATP requirements and sugars, lipids and proteins all fuel the citric acid cycle efficiently. Thus, each of them might be able to modulate the stress-induced cortisol response.

To further investigate the mechanisms responsible for calorie-induced enhancement of HPA activity, we randomly administered glucose, protein, fat, and water to healthy men after an eight-h fasting period before exposure to a standardized psychosocial stress test (TSST). If the underlying mechanism responsible for HPA response modulation is located at a central level, modulation of the cortisol stress response should be limited to increases in blood glucose levels. Otherwise, if the citric acid cycle is responsible for HPA axis modulation by caloric load and fasting, other fuels like proteins or fat should also be able to produce this effect.

MATERIALS AND METHODS

Sample

Thirty-seven healthy men were randomly assigned to one of four experimental groups: glucose load ($n =$

8); protein load ($n = 10$); fat load ($n = 10$); or water ($n = 9$). All of them were nonsmokers and free from medication. The groups were matched for age and body mass index (BMI; mean age 23.22 ± 0.43 SEM; mean BMI 23.02 ± 0.32 SEM). Participants were required to fast for at least 8 h before the experiment started. The experimental procedure was approved by the Ethics Committee of the University of Trier and written informed consent was obtained from the volunteers.

Procedure

All tests were performed between 1600 and 1900 h. Subjects arrived at the laboratory after an eight-h fast, and baseline glucose levels were measured in capillary blood (puncture of finger tip; Reflolux S, Boehringer Mannheim, Mannheim, Germany). Five minutes later, subjects ingested the respective caloric load or water. The glucose group ingested 75 g dissolved in water in a total volume of 300 ml (Dextro OGTT, Boehringer Mannheim). The fat group ingested 200 grams of avocado, which has an estimated content of 80 g fat and less than 5 grams carbohydrates and proteins, respectively. The protein group drank 83 g of proteins dissolved in 300 ml of mineral water (Proteindrink, Formula 80+, Multipower, Hamburg, Germany). Finally, the water group drank 300 ml of mineral water. Forty-five minutes after intake, a second blood glucose reading was obtained. Immediately after this measurement, all subjects were exposed to the psychosocial stress test (TSST) and a third glucose determination was performed thereafter. Five saliva samples were collected before TSST and five samples afterward, all of them using the Salivette device (Sarstedt, Rommelsdorf, Germany). The aim of the first five samples was to obtain information about the response profile of cortisol after caloric/water uptake. Thus, saliva samples were obtained immediately before and 10, 20, 30, and 45 min after ingestion of glucose, fat, protein, or water (samples 1–5). Sample 5 was taken as baseline for the following stress-induced cortisol response. Samples 6–10 (after TSST) were used to assess the cortisol response to psychosocial stress, and were taken 1, 10, 20, 30, and 60 min after the TSST. In order to control for possible differences in the psychological parameters between groups, we evaluated perceived stress and mood changes using self-reports scales, which have been demonstrated to be sensitive to the TSST (Kudielka, Hellhammer, Hellhammer, Wolf, Pirke, Varadi, Pilz, and Kirschbaum, 1998).

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