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Psychosocial stress affects energy balance in mice: Modulation by social status

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Summary Stress has been associated to changes in eating behaviour and food preferences. Moreover, psychosocial and socio-economical challenges have been related with neuroendocrine-autonomic dysregulation followed by visceral obesity and associated risk factors for disease. In the current study, we provide a model of body weight development, food intake, energy expenditure of subordinate and dominant mice under psychosocial stress either in the presence of a standard diet or of a high palatable diet. When only standard chow was available stressed animals consumed more food in comparison to the control counterpart. Moreover, subordinate mice, at the end of the stress period were heavier in comparison to dominant animals. This last result was due to a decrease in the caloric efficiency of dominant animals in comparison to subordinates. Confirming this, the results of the experiment 2 showed that dominant mice significantly increase their energy expenditure at the end of the chronic psychosocial stress procedure in comparison to subordinate mice, as measured by indirect calorimetry. When a palatable high fat diet was available subordinate animals became heavier in comparison with both dominant and control animals. No differences in the caloric intake were found between groups. Subordinate mice ingested more calories from fat than controls, while dominant animals ingested more calories from carbohydrates. These results suggest that psychosocial stress can be a risk factor for overeating and weight gain in mice. However, social status influences the extent to which an individual keeps up with adverse environment, influencing the vulnerability toward stress related disorders.
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1. Introduction

A critical issue for contemporary research on stress is to explain why some individuals that experience adverse life events develop disease while others do not. Epidemiological and clinical studies

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demonstrate that a variety of factors such as early experience, genetic aspects, and social support modulate individual responses to stress (Kessler and McLeod, 1984; Kessler et al., 1985; Kendler et al., 1995; Troisi, 2001). In humans, chronic psychological stress can increase the risk of several diseases or increase the severity of pre-existing diseases. Psychosocial and socio-economical challenges such as low income, low education and divorce have been associated with perturbed cortisol secretion and neuroendocrine-autonomic dysregulation followed by visceral obesity and associated risk factors for disease (Rosmond and Bjorntorp, 2000). Moreover, stress has been associated to overeating in both men and women (Van Strien et al., 1986; Greeno and Wing, 1994) and stress-driven eaters typically prefer foodstuff rich of fats and high in palatability (Laitinen et al., 2002). However, chronic stress in humans does not inevitably lead to weight gain. For example, recent severe adverse life events were among the commonest identifiable antecedent of anorexia nervosa (Rastam and Gilberg, 1992).

Animal studies on the effects of chronic stress on the energy homeostasis have failed to model the complex variability found in humans. Although the observation that a acute stress can trigger a composite interaction among weight gain phenotype, diet and stress responsivity (Michel et al., 2003), in general, data concerning the effects of repeated stress in animal models report a long lasting decrease in body weight and energy efficiency in stressed animals, regardless the type of stress, often but not always, paralleled by a decrease in food intake (van Leeuwen et al., 1997; Rybkin et al., 1997; Harris et al., 1998; Michel et al., 2005). For instance, it has been reported that repeated immobilization decreases body weight in rats and mice (Harris et al., 1998; Ricart-Jane et al., 2002). Similar results were obtained in subordinate rats following repeated defeats in a model of social stress (Haller et al., 1999). In more naturalistic settings such as the visible barrow system (VBS) it has been shown that while dominant rats maintain their body weight and controls gain weight, subordinate animals show a rapid and considerable weight loss (Blanchard et al., 1995; Tamashiro et al., 2004). The basis of these dissimilar results obtained from animal models and humans remains largely unexplained. Interestingly, in a recent study (Bartolomucci et al., 2004, 2005) it has been observed that in male mice both social status and territory ownership are factors associated to individual vulnerability to chronic psychosocial stress-induced effects on body weight and fat deposition. In this study, only resident mice that had lost their territory and had become subordinates showed an increase in their body weight, but not in food intake. By contrast, dominants showed a significant decrease of body weight throughout the psychosocial stress procedure. This was independent from corticosterone levels that indeed were increased in both subordinate and dominant mice (Bartolomucci et al., 2001). These data suggest that, in male mice, the loss of acquired resources and social status actually interact to drive the effects of stress toward dysmetabolism.

In rodents, stress and glucocorticoids increase palatable food intake (Dallman et al., 2003; Pecoraro et al., 2005). It has been proposed that stress induces or maintains higher incentive salience toward high palatable/high caloric food compared to chow as an adaptive coping mechanism acting to relieve the symptoms associated to the stress related increase in the HPA axis activation (Dallman et al., 2003; Pecoraro et al., 2004). Despite this, so far most of the data available on animal models of stress-induced weight gain have been collected in animals offered standard chow. There are only few studies where palatable food has been provided and in these studies animals underwent repeated immobilization, a procedure known to dramatically reduce food intake (Marti et al., 1994). Thus, the consumption of a high fat diet resulted in an attenuation of the typical body weight loss, rather than a body weight gain, in the restrained group fed on palatable food (e.g. Pecoraro et al., 2004).

The present study is an attempt to model in mice the individual differences in stress-related effects on body weight and food intake found in humans. Food intake, body weight, circulating triglycerides (TGC), esterified free fatty acids (NEFA), glucose (GLU) (Exp 1) and energy expenditure (Exp 2) were monitored in mice under chronic psychosocial stress. In the experiment 3, we tested the hypothesis that the availability of a high palatable food could exacerbate the effects of psychosocial stress on body weight and food intake regulation.

2. General methods

2.1. Subjects

Outbred sexually naive mice of the NMRI strain were used in this study. Mice were born and reared in a colony room at the Institute of Neuroscience CNR of Rome originating from breeders purchased from

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