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The weight gain response to stress during adulthood is conditioned by both sex and prenatal stress exposure

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Summary Food intake and weight gain are known to be affected by stress. However, the type and duration of the stress may have variable effects, with males and females responding differently. We report the short-term and long-term effects of prenatal and adult immobilization stress, as well as the combination of these two stresses, on weight gain and food intake in male and female rats and the role of post-pubertal gonadal hormones in this process. No long-term effect of prenatal stress on food intake or weight gain was found in either sex. However, during the period of adult stress [at postnatal day (P) 90; 10 days duration] stressed male rats gained significantly less weight than controls and previous exposure to prenatal stress attenuated this effect (control: 31.2 ± 2.1 g; prenatal stress: 24.6 ± 3.8 g; adult stress: 8.1 ± 3.4 g; prenatal and adult stress: 18.2 ± 3.3 g; $p < 0.0001$). There was no change in food intake in response to either prenatal or adult stress. Adult stress increased circulating corticosterone levels during the initial part of the stress period, in both male and female rats with this rise being greater in male rats. No effect on corticosterone levels was observed on the last day of stress in either sex. No effect on weight gain or food intake was observed in female rats. Following adult stress, male rats increased their weight gain, with no change in food intake, such that 1 month later they reached control levels. At the time of sacrifice (P180), there were no differences in weight or circulating metabolic hormone levels between any of the male groups. Although castration alone modulated body weight in both male and female rats, it did not affect their weight gain response to adult stress. These results indicate that the weight gain response to adult stress is sexually dimorphic and that this is not dependent on post-pubertal gonadal steroids. Furthermore, the outcome of this response closely depends on the time at which the change in weight is analyzed, which could

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help to explain different results reported in the literature. Indeed, weight and metabolic hormone levels were normalized by the end of the study.

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Introduction

Environmental factors during both the prenatal and postnatal periods have been clearly shown to affect metabolic efficiency during adulthood (Fernandez-Twinn and Ozanne, 2006; Torres and Nowson, 2007). Indeed, maternal diet, emotional state and hormonal balance during pregnancy and lactation are known to influence the offspring's future metabolic function (Lesage et al., 2004; Fernandez-Twinn and Ozanne, 2006; Vickers et al., 2008; Tamashiro et al., 2009). Furthermore, the neonate's early dietary intake and emotional wellbeing are also important factors in the determination of their future propensity to become overweight or to develop metabolic imbalances (Levin, 2006; Nathanielsz, 2006; Spencer and Tilbrook, 2009). Thus, it is conceivable that one factor that may contribute to the rapid rise in mean body weight observed in developed countries is increased exposure to prenatal and postnatal environmental stress (Dallman et al., 2004).

Chronic exposure to increased levels of glucocorticoids prenatally, either by hormonal treatment or maternal stress during gestation, is reported to induce adverse effects on metabolism in later life in both humans and rodents (Seckl, 2001; Maccari et al., 2003; Maccari and Morley-Fletcher, 2007; Tamashiro et al., 2009). Prenatal stress results in decreased birth weights and plasma glucose levels of both male and female neonates (Lesage et al., 2004) and modulates the offspring's response to future stresses, including dietary changes or fasting (Koehl et al., 1999; Lesage et al., 2004; Tamashiro et al., 2009). However, adult rats that had been exposed to prenatal stress are reported to have increased basal glucose levels, as well as reduced body weight and food intake (Vallée et al., 1996). In contrast, others have reported that prenatal stress has no significant effect on basal body weight or food intake in adult male rats, but that it increases basal glucose levels and food intake in response to fasting (Lesage et al., 2004). In addition, Tamashiro et al. (2009) recently reported that prenatally stressed animals had no change in glucose tolerance on a normal diet, but had impaired glucose tolerance on a high-fat diet. Thus, although it appears to be clear that prenatal stress affects metabolism in later life, the precise effect is not evident. This is most likely due to the use of different experimental protocols and animal models, as well as to the social situation of the animal under study (Bartolomucci et al., 2009).

The pathophysiological outcome of prenatal stress also differs between males and females, with numerous studies demonstrating sex differences in behavioral modifications and responsiveness of the stress axis in adulthood (Reznikov et al., 1999; Bowman et al., 2004; Tobe et al., 2005; Weinstock, 2007; Mueller and Bale, 2008; Zuena et al., 2008). Structural and neurochemical modifications in the hippocampus, cortex, amygdala and hypothalamus are associated with some of these adverse outcomes and these too are often sexually dimorphic (Reznikov et al., 1999; Bowman et al., 2004; Tobe et al., 2005; Weinstock, 2007; Mueller and Bale,

2008; Zuena et al., 2008). However, whether the effects of prenatal stress on weight gain are sexually dimorphic has not been thoroughly examined. Thus, the aim of this study was to determine whether prenatal stress modulates weight gain, as well as the weight gain response to stress during adulthood. Furthermore, we analyzed whether males and females respond similarly to these manipulations and if the action of gonadal steroids during adult life plays a role in the difference between the sexes in their stress response.

Materials and methods

Materials

All chemicals and reagents were purchased from Sigma or Merck (Barcelona, Spain) unless otherwise indicated.

Animals

All experiments were designed according to the European Union laws for animal care and the study was approved by the local institutional ethics committee. Young adult pregnant Wistar rats were housed individually and maintained at a constant temperature ($22 \pm 1^\circ\text{C}$) and humidity ($50 \pm 1\%$) under alternate constant light (12 h)—dark (12 h) periods and allowed free access to rat chow and tap water.

Experiment 1

Restraint stress was performed daily in pregnant rats during the last week of gestation (gestational days 14–21) by placing them in transparent plastic cylinders (7 cm in diameter, 19 cm long) along with bright light exposure for 45 min, three times a day, as previously described (Ward and Weisz, 1980). Female rats from the control group remained undisturbed in their home cage.

At birth pups remained with the dam with no handling of either the pups or the mothers until postnatal day 21 (P21) at which time they were weaned. Only litters consisting of 9–14 pups were employed in the study. At P21, pups were distributed (four/cage) according to their origin from control or stressed dams, with males and females being housed separately. At approximately P90, 9–10 rats from each of the 2 groups (i.e., control, C or prenatally stressed, PnS) and of each sex were subjected to adult stress (AS). Female rats were subjected to adult stress starting on day 2 of diestrus (as determined by vaginal swab). Adult stress was induced by using a similar protocol to that described for prenatal stress, but lasted for 10 consecutive days. This protocol was based on that described by Chung et al. (2005) where the period of postnatal stress was a few days longer than that used for the prenatal stress. At 180 days of age, all rats were killed by decapitation between 1900 h and 2200 h. Trunk blood was collected, allowed to clot and then centrifuged at 3500 rpm. The serum was separated and stored at -70°C until processed.

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