ENDOPLASMIC RETICULUM STRESS MAY BE ASSOCIATED WITH SEXUAL DIMORPHISM IN RAT BODY WEIGHT

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Males and females differ in body adiposity, sites of fat deposition, and insulin sensitivity. The lack of sexual hormones causes obesity and insulin resistance in both sexes. Obesity is related to ER stress and this stress, in turn, has a central role in insulin resistance. Therefore, the aim of this study was investigate the role of ER stress pathways in body composition differences observed between males and females.

Male Wistar rats were divided into three groups: sham-operated (SHAM), orchiectomized treated with vehicle (OQX) or treated with testosterone propionate (0.1 mg/100g body weight) (OQX+T). Female Wistar rats were divided into three groups: sham-operated (SHAM), ovariectomized treated with vehicle (OVX) or treated with 17b-estradiol benzoate (0.7 mg/100g body weight) (OVX+E). The hormones were administered for 21 days. Body weight and food intake were measured daily. Liver from both sexes were used to determine BIP, eIF2α and IRE1 protein and mRNA levels.

Body weight gain and food efficiency were impaired in OQX male rats in comparison with SHAM and OQX+T animals. Western blot showed that liver from OQX males had 23% more BIP and 51% more eIF2α than SHAM and OQX+T males. The genomic BIP expression was also 35% higher in OQX males. In females, body weight gain and food efficiency were significantly higher in OVX rats and estradiol replacement was able to circumvent this increase. BIP and eIF2α protein expression was 71% and 42% higher, respectively, in liver of OVX than in SHAM females. No differences were detected in the mRNA content of BIP and IRE1 of females.

These results suggest that ER stress susceptibility can be modulated by sexual hormones. However, further experiments must be performed in order to investigate the physiological importance of this difference.

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