

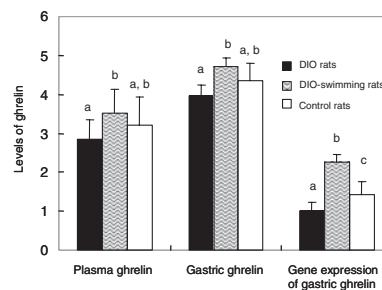
5th International Immunonutrition Workshop, 6–8 April 2011, Puerto Vallarta, Mexico

## Ghrelin expression and effect of exercise in obesity

M. Xiang<sup>1</sup>, W. Liu<sup>2</sup>, L. S. Harbige<sup>1</sup> and H. Ai<sup>2</sup>

<sup>1</sup>Centre for Biosciences Research, School of Science, University of Greenwich, Kent ME4 4TB, UK and <sup>2</sup>Division of Nutrition and Biochemistry, Institute of Sports Medicine, The Third Hospital, Peking University, Beijing, P.R. China

Obesity is a major public health problem associated with morbidity and mortality and continues to increase worldwide<sup>(1)</sup>. Ghrelin is a new growth hormone (GH)-releasing peptide discovered by Kojima *et al.*<sup>(2)</sup> in 1999. Ghrelin is produced by stomach cells and is a potent circulating orexigen, controlling energy expenditure, adiposity and growth hormone secretion<sup>(3)</sup>. Exercise is part of a healthy lifestyle and frequently is an important component in combating chronic diseases such as obesity. Understanding the molecular events initiated by regular exercise can be studied in laboratory animals such as rodents<sup>(4)</sup>. The model of diet-induced obesity (DIO) in rats has many features in common with human obesity and can serve as a useful model to study the pathogenesis and treatment of obesity. The present study determined the effect of exercise on weight loss, plasma ghrelin and gastric ghrelin levels at the gene and protein levels in DIO and DIO-swimming rats. Male Sprague–Dawley rats (*n* 110) at 3 weeks of age were randomly assigned into groups. Ten rats were fed rat chow during the study as the control and 100 rats were fed both rat chow plus high fat diet (HFD, 5.58 kcal/g (23.34672 kJ/g) with 66.5% as fat/lard) *ad libitum* for 14 weeks. At the end of this period, forty-four rats developed DIO which was assessed by the Lee obesity index<sup>(5)</sup>. The Lee obesity index was calculated by dividing the cube root of body weight (g) by body length (cm) × 1000<sup>(5)</sup>. The DIO rat's Lee obesity index was more than the average Lee obesity index plus 2 SD of the chow-fed control rats. At week 15, ten chow-fed control rats were kept on the rat chow, eleven DIO rats were kept on the rat chow plus HFD *ad libitum* and another eleven DIO rats were also kept on the rat chow plus HFD *ad libitum* and trained to swim at 28–30°C for 40 min/d and 6 days/week for 5 weeks (DIO-swimming). Other rats were used in other studies (data not presented). It was found that the body weight of DIO-swimming rats after the swim-training for 5 weeks was significantly less than that of DIO rats before the swim-training and that of DIO rats, however, the body weight of DIO-swimming rats after the swim-training was significantly more than that of chow-fed control rats. DIO-swimming rats had significantly lower perirenal, epididymal and total fat pads weight than DIO rats, but, had significantly higher fat pads weight than chow-fed control rats. The plasma ghrelin concentration and the level of gastric ghrelin in DIO-swimming rats were significantly higher than those of DIO rats, although, was not different compared with chow-fed control rats (Fig. 1). Furthermore, the gene expression of gastric ghrelin in DIO-swimming rats was significantly higher than that of DIO rats and chow-fed control rats (Fig. 1). The body weight and the abdominal fat depots weight were negatively correlated with the levels of plasma ghrelin, gastric ghrelin and gene expression of gastric ghrelin. These results indicate that the elevated ghrelin at the gene and protein levels during weight loss caused by swimming may be the physiological basis of resistance to weight loss in obesity.



a,b,c, Mean values with unlike superscripts were significantly different ( $P < 0.05$  or  $P < 0.01$ ).

1. Dhillon WS (2007) *Thyroid* **17**, 433–445.
2. Kojima M, Hosoda H, Date Y *et al.* (1999) *Nature* **402**, 656–660.
3. Dixit VD, Schaffer EM, Pyle RS *et al.* (2004) *J Clin Invest* **114**, 57–66.
4. Ghosh S, Golbidi S, Werner I *et al.* (2010) *Clin Sci* **119**, 57–74.
5. Bernardis LL & Patterson BD (1968) *J Endocrinol* **40**, 527–528.