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Visfatin as a novel adipokine in relation to AMP-activated protein kinase in obesity and weight loss

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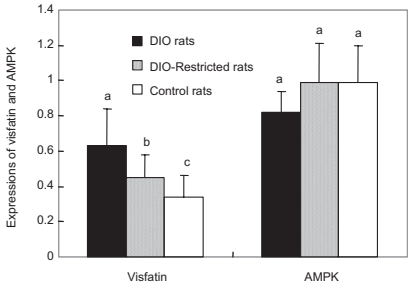
Obesity is a leading preventable cause of death worldwide with increasing prevalence in adults and children. It is one of the most serious public health problems of the twenty-first century[(1)]. Visfatin, known as pre-B-cell colony-enhancing factor (PBEF) and nicotinamide phosphoribosyltransferase (Nampt), is regarded as a novel adipokine and may have a function as a direct proinflammatory mediator of obesity-related vascular disorders with a potential role in plaque destabilisation[(2)]. Visfatin/PBEF/Nampt is preferentially expressed by visceral adipose tissue compared with subcutaneous fat[(3)]. AMP-activated protein kinase (AMPK) is a key molecular player in energy homoeostasis at both cellular and whole-body levels. AMPK has been shown to have a role in the physiological regulation of fatty acid and glucose metabolism, and in the regulation of appetite and body weight. Therefore, AMPK is considered a major player in the development of obesity[(4)]. 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. In Wistar rats, half the rats become hyperphagic and develop DIO, whereas the rest are diet-resistant (DR) when the rats are fed with a high fat (44% fat) diet[(5)]. The present study investigated the relationship between visfatin and AMPK in DIO and DIO-restricted 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) *ad libitum* for 14 weeks. At the end of this period, forty-four rats developed DIO which was assessed by the Lee obesity index[(6)]. The Lee obesity index was calculated by dividing the cube root of body weight (g) by body length (cm) $\times$ 1000[(6)]. 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 the other eleven DIO rats that were designated as DIO-restricted, were restricted on the intake of the rat chow plus HFD for 5 weeks. Other rats were used in other studies (data not presented). Plasma visfatin concentration was significantly lower in both DIO-restricted and chow-fed control rats than DIO rats and was 69 and 70% of DIO rats, respectively. DIO-restricted rats had significantly lower visfatin expression in the visceral adipose tissue than DIO rats, however, they had significantly higher visfatin expression than chow-fed control rats (Fig. 1). Moreover, the activity of AMPK in the visceral adipose tissue tended to be higher in both DIO-restricted and chow-fed control rats than DIO rats (Fig. 1). These results indicate that visfatin and AMPK may play important roles in the development of obesity. The lowered visfatin expression and elevated AMPK activity in visceral adipose tissue in DIO-restricted rats may stimulate fatty acid oxidation, and we hypothesise that inactive visfatin and activate AMPK may be of therapeutic targets in obesity.



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

1. Tilg H & Moschen AR (2008) *Clin Sci* **114**, 275–288.
2. Sommer G, Kralisch S, Kloting N *et al.* (2010) *Obesity* **18**, 1486–1492.
3. Fukuhara A, Matsuda M, Nishizawa M *et al.* (2005) *Science* **307**, 426–430.
4. Lim CT, Kola B & Korbonits M (2010) *J Mol Endocrinol* **44**, 87–97.
5. Xiao H, Pei X, Li X *et al.* (2008) *Chin J Sports Med* **27**, 170–173.
6. Bernardis LL & Patterson BD (1968) *J Endocrinol* **40**, 527–528.