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Relation between visfatin as a novel adipokine and AMP-activated protein kinase in obesity

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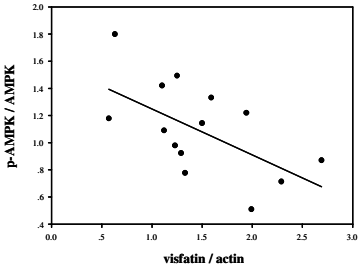
Obesity is a serious public health problem because it increases the risk of chronic diseases such as diabetes mellitus, cardiovascular disease, stroke and some cancers[1,2]. Visfatin, known as pre-B cell colony-enhancing factor (PBEF) and nicotinamide phosphoribosyltransferase (Nampt), is regarded as a novel adipokine that improves glucose tolerance and may play a role in the development of obesity-associated insulin resistance and type 2 diabetes mellitus (T2DM). Visfatin/PBEF/Nampt is preferentially expressed by visceral adipose tissue compared with subcutaneous fat[3]. AMP-activated protein kinase (AMPK) is a major regulator of energy metabolism at both the cell and the whole body level. Many studies have suggested a role for AMPK in the physiological regulation of fatty acid and glucose metabolism, and in the regulation of appetite and of body weight. Therefore, AMPK is considered as 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 model to study the pathogenesis and treatment of obesity. In Wistar rats, a half of the rats became hyperphagic and developed DIO, whereas the rest were diet-resistant (DR) when the rats were fed with a high fat (44% fat) diet[5]. The present study investigated the relationship between visfatin and activated and inactivated AMPK (p-AMPK:AMPK) in DIO and DR rats. Male Wistar rats ($n = 20$) at 4 weeks of age were fed rat chow for 3 days and were switched to high fat diet (3.98 kcal/g with 44% as fat) for 10 weeks. At the end of this period, 7 highest and 7 lowest abdominal fat pads (% body weight) gainers were retrospectively identified as DIO and DR, respectively. It was found that plasma visfatin in the DIO rats increased by 18% compared to the DR rats. Furthermore, visfatin of the visceral adipose tissue in the DIO rats was significantly higher than that of the DR rats. However, AMPK of the visceral adipose tissue in the DIO rats was significantly lower than that of the DR rats. A significant negative correlation was found between the visfatin and AMPK of visceral adipose tissue ($r = 0.58$, $P<0.05$; Fig.). These results indicate that the elevated visfatin expression and lowered AMPK activity in visceral adipose tissue may be related to the development of obese condition in rats. Visfatin may play a role in obesity development by down-regulating AMPK activity.



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