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ORIGINAL ARTICLE

Serum TNF-α, GTH and MDA of high-fat diet-induced obesity and obesity resistant rats

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KEYWORDS

High-fat diets;
Obesity;
Obesity resistant rats;
Serum TNF-α;
MDA;
Glutathione

Abstract *Objective:* Mechanism of high fat diet-induced obesity is analyzed and serum tumor necrosis factor, malondialdehyde and glutathione levels of obesity resistant rats are effectively analyzed. *Methods:* 120 male SD rats were grouped into obesity group and control group, each group with 60 rats. Obese rats were fed with high fat diet, while control rats were fed with ordinary fodder. After six months of feeding, growth degree of two groups of rats is observed , and the rats are divided into obesity group and obesity resistant group based on extent of growth. Then glutathione, tumor necrosis factor-α and MDA content in bat serum are detected with enzyme-linked immunosorbent assay. *Results:* The content of tumor necrosis factor α in obese rats and obesity resistant rats is far higher than that in control group ($P < 0.05$), there exists no statistical significance ($P > 0.05$) in tumor necrosis factor α in obesity group and obesity resistant group, glutathione level of obesity group rats and obesity resistant group rats is significantly increased ($P < 0.05$) compared with that of control group, and also serum MDA level of the two groups has statistical significance compared with that of normal control group ($P < 0.05$). *Conclusion:* Among rats fed with high fat diet, in comparison with weight of obesity resistant rats and control group rats, there is no statistically significant difference, ($P > 0.05$). However, high fat diet will impact mechanisms in vivo in rats, which then induces oxidative stress response and inflammatory response in rats.

1. Introduction

At the end of the last century, the World Health Organization introduced relevant provisions on obesity, regarding that obesity is a disease. In recent years, with the continuous improvement of people's living standards and change in dietary structure, incidence of obesity has shown an increasing trend and obesity has become a serious health killer. Relevant research shows that obesity is often accompanied with chronic inflammation and emergence of oxidative stress in patients. Tumor necrosis factor-α is the major protein associated with obesity, which plays a very important role in regulating body fat metabolism (Suo and Wang, 2015). Obesity can cause serious increase in vivo tumor necrosis factor α content in patients. Research at this stage considers that this phenomenon is mainly related to low-grade inflammation

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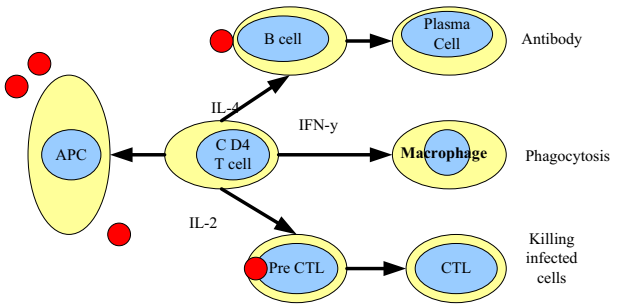

Figure 1 Structure chart of tumor necrosis factor α.

and natural immunity (Liu and Liu, 2012). The author conducted meticulous research on 120 male SD rats, and applied rat test results in human clinical therapy (Song et al., 2014). The structure chart of tumor necrosis factor α is shown in Fig. 1.

2. Materials and methods

2.1. General information

The subjects selected in this study are SD rats born after three weeks, with delactation of 3 days, 120 healthy male rats. After three-day adaptive feeding of rats, the rats were randomized, namely high-fat group and control group, respectively 60. The rats received eating and drinking on their own, and were housed in separate ventilated cages. Each rat was recorded once a week (Jiang and Jiang, 2014). After raising a total of six months, rat weight is observed. Based on different weight of different rats, 60 high-fat fed rats are divided into obesity and obesity resistant groups (Hao et al., 2010).

2.2. Preparation of samples

High fat diet is used for a period of six months of feeding. After obese rat model is successfully established, let rats eat 12 h, anesthesia disposal of rats is applied with 2% intraperitoneal injection of pentobarbital, and femoral vein of rats is removed , venous blood serum is collected, to be stored at a temperature of minus 20°. Perirenal fat tissue, omental adipose tissue and tissue surrounding testis of rats are weighted. The total weight of rat fat is equal to the sum of the three (August et al., 2016; Wang et al., 2014). The fat tissue surrounding testis is shown in Fig. 2.

2.3. Level determination

Tumor necrosis factor α, MDA and glutathione levels were measured and the method chosen is enzyme combined immunization method. Specific steps follow relevant operating instructions. The MDA formula is shown in Fig. 3.

2.4. Statistics

Relevant data obtained in this study were tested with package SPSS15.0. Measurement data obtained in the study are denoted by ($\bar{x} \pm s$) and tested with t test. Experimental data

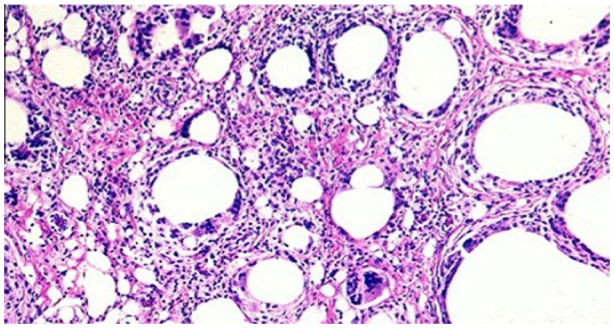

Figure 2 Fat tissue surrounding testis.

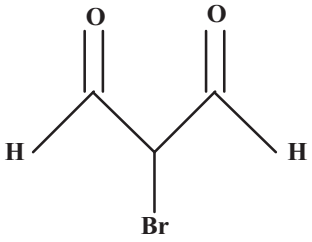

Figure 3 MDA formula.

obtained by the two sets of statistics are $P < 0.05$, which indicates that effect after group therapy is with significant difference.

3. Results

3.1. Total weight of rats and fat

During the study, the total weight of overall fat of SD rats was observed. It can be found that, weight of obesity group rats is markedly increased compared to that of control group, and there was a significant increase in total weight of fat compared with control group, but without significant statistical difference. The Comparison of body weight of control group, obesity group and obesity resistant group rats is shown in Table 1.

3.2. Comparison of content of serum tumor necrosis factor α, malondialdehyde and glutathione

Serum tumor necrosis factor-α levels of obesity and obesity resistant group rats are significantly increased compared to normal control group rats. There is no statistical difference between obesity and obesity resistant group (Xu et al., 2011; Aldea et al., 2016; Huang et al., 2012). There exists no statistical significance in content and level of malondialdehyde and

Table 1 Comparison of body weight of control group, obesity group and obesity resistant group rats ($\bar{x} \pm s$).

Item	Control group	Obesity group	Obesity resistant group
Weight	401.29 ± 116.87	665.21 ± 170.08	387.19 ± 51.39
Fat weight	19.99 ± 14.29	65.71 ± 28.69	31.98 ± 12.98