OPEN ACCESS Freely available online

Journal of Glycomics & Lipidomics

ISSN: 2153-0637

Perspective

High Efficiency Nutrient Utilization of Dietary-Induced Obesity in Animal Models

Carlos Rolo*

Department of Human Physiology, Medical University of Gdansk, Gdansk, Poland

DESCRIPTION

Obesity is a prevalent public health problem that affects people of all ages. Since it affects people's health and quality of life, it has a significant social and economic impact. Basic concepts of obesity include, accumulation of visceral and subcutaneous lipids as well as weight gain that may be harmful to one's health. However, lipid deposition (ectopic fat) in non-adipose tissues, such as the liver, is frequently present in conjunction with it. In order to control body weight and adiposity to prevent obesity, it must maintain a negative energy balance, which includes both a healthy diet and regular exercise. However, the development of alternatives for the treatment of obesity, such as functional foods and bioactive compounds, is becoming more important as a consequence of changes in people's lifestyles, including decreased physical activity and changes in eating habits.

According to the way it is assessed in human beings and in animal models, obesity can be determined by criteria based on body weight gain or the Lee obesity index and an increase in body fat content. However, with exception of BMI in humans, there are no recognized standards for obesity. The body weight (or fat) of the experimental group given a high-fat or energy-dense diet has usually been compared to control animals that exhibit normal growth when given chow or low-fat diets to determine the level of obesity. Failure to adjust fat oxidation to the additional fat in the diet, an increase in adipose tissue lipoprotein lipase activity, increased meal size and decreased meal frequency, overconsumption of energy due to the high energy density of the diet, orosensory characteristics of fats, and poorly satiating properties of high-fat diets are all factors that contribute to obesity.

The hypothalamus, neuropeptides like ghrelin and neuropeptide Y, hormones like insulin and leptin are all thought to play a role in the regulation of body weight and food intake in dietary obesity. The adipose tissue is to be an endocrine organ because it makes cytokines like IL-6 and TNF; As a result, obesity may be considered a chronic inflammatory condition.

When an animal uptake more energy than it uses, it becomes obese, leading to an increase in the amount of energy stored in its body fat, especially in the adipose tissues. A combination of hyperplasia (an increase in the number of adipocytes) and hypertrophy (an increase in their size) is associated with obesity. At first, it was hypothesized that the number of adipocytes was determined in early childhood, and that an increase in adipocyte size was the cause of adult obesity. Hyperplasia is an on-going condition that does not start in childhood. When adipocytes reach hypertrophy at any stage of life, they release growth factors like TNF and insulin-like growth factor to promote adipocyte hyperplasia.

Both the Lee obesity index and an increase in body fat content can be used to evaluate obesity in animal models and in human subjects. However, unlike BMI in humans, standard thresholds for obesity have not been established. The majority of studies evaluate the degree of obesity by comparing the experimental group's body weight (or fat) with that of control animals that grow normally on chow or low-fat diets. Animals in models of dietary obesity are categorized as prone or resistant based on their body fat, body weight gain, noradrenaline concentrations in urine.

CONCLUSION

The pathophysiology of the obese syndrome in humans can be better understood using animal models of diet-induced obesity. Physiological mechanisms like the high efficiency of dietary fat which is being stored in the body, the low satiating effects of fats that lead to overconsumption of fat-rich diets and changes in the hormones involved in energy balance have been revealed through the use of fat-rich diets that mimic the human diet. Fat-rich diets cause hyperleptinemia and hyperinsulinemia, but they also cause leptin and insulin resistance, while fat-rich meals cause a lower suppression of ghrelin secretion. Fat-rich diets are associated with a sensory-specific facilitation of intake, one of the behavioural mechanisms of dietary-induced obesity. The connection between obesity and diurnal feeding in large meals has been established through meal pattern analysis of fat-fed animals and diurnal feeding rhythms.

Correspondence to: Carlos Rolo, Department of Human Physiology, Medical University of Gdansk, Gdansk, Poland, E-mail: carlosrolo5656@gamil.com

Received: 29-Nov-2022, Manuscript No. JGL-23-21502; **Editor assigned:** 01-Dec-2022, Pre QC No. JGL-23-21502 (PQ); **Reviewed:** 15-Dec-2022, QC No. JGL-23-21502; **Revised:** 22-Dec-2022, Manuscript No. JGL-23-21502 (R); **Published:** 29-Dec-2022, DOI: 10.35248/2153-0637.22.11.322.

Citation: Rolo C (2022) High Efficiency Nutrient Utilization of Dietary-Induced Obesity in Animal Models. J Glycomics Lipidomics. 11:322.