Short Communication
Volume 4:2,2020
DOI: 10.37421/JMA.2020.4.127

Journal of Morphology and Anatomy

ISSN: 2684-4265 Open Access

New Approaches on High Fat Diet Research

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Abstract

Obesity is defined as an excess of body-fat mass. Reliable fat-mass quantitation requires sophisticated tools that are not widely available. BMI is a gross measurement of body fat where it could have an effect at the cellular level. Multiples researches have shown the effects of high-fat diet on behavioral eating patterns, body composition, lipid metabolism and insulin sensitivity, and the role of exercise on these parameters. Also, it is important to consider the positive effects of exercise on adipose tissue metabolism and to understand the mechanisms by which this tissue affects the kinetics of other macromolecules. Some intermittent energy restriction regimens have gained popularity as strategies for achieving weight loss and other metabolic health benefits. However, clinical studies comparing weight loss with intermittent energy restriction regimens to traditional continuous energy restriction in adults with overweight and obesity are limited. Although scientific evidence clearly shows the effects of high-fat diet on behavioral eating patterns, body composition, lipid metabolism and insulin sensitivity; it also important to characterize relationships between these parameters and the role of exercise and some intermittent energy restriction regimens, emphasizing the timing of dietary intake and other aspects of behavior that are linked to intake.

Keywords: BMI • Exercise • High-fat diet • Insulin

About the Study

Obesity is broadly defined as an excess of body-fat mass or the weight that is higher than what is considered as a healthy weight for a given height. Body fatness has been an important psychosocial issue among humans for millennia [1]. In this sense, Body Mass Index or BMI, has been used widely as a screening tool for overweight or obesity. However, reliable fat-mass quantitation requires sophisticated tools that are not widely available and it has hampered efforts to arrive at a more specific definition [1]. Consequently, an elevated body mass index (BMI), which expresses body weight (in kilograms) as a function of body height (in meters^2) as a surrogate measure of body fatness, is the most widely accepted definition of obesity [2].

BMI it seems not to be a reliable clinical tool for assessing individual body fatness, because variations in skeletal muscle and other lean-body-mass components create substantial variations in total body mass. For example, a heavily muscled individual with increased body weight relative to height will have a BMI value that can erroneously place them into the overweight or even obese category. Additionally, there are significant racial/ethnic differences in how BMI associates with adverse medical consequences [2]. In fact, Vázquez-Guzmán, *et al*. has shown that a BMI of 28 kg/m2 is not the best way to diagnose overweight, because it has a very low sensitivity and specificity [3].

In this sense, early on it was recognized that tall people had a lower death rate than did short people with the same weight/height ratio [4-6]. Meanwhile, it also was recognized that a person's height in general and leg length in particular could affect the calculated body mass adjusted for height. Likewise, a person's bone mass, also could affect the interpretation of this ratio [1].

BMI is a gross measurement of body fat. Can it have an effect at the cellular level? Literature shows that it does. Black, *et al*. found that diets relatively high in fat and low in carbohydrate may contribute to increased adiposity associated insulin resistance and β-cell dysfunction in persons who have not yet developed diabetes [7]. Meanwhile, Sikaris shown that BMI had a statistically significant higher correlation with insulin resistance in participants were a 2-hour glucose test was given [8]. Likewise, at the cellular level, an increase in BMI is likely going to increase stored energy and adipocytes, where with 10 kilograms of weight excess, there is a 10% to 30% increase in β-cell mass [9].

Multiples researches have shown the effects of high-fat diet on behavioral eating patterns, body composition, lipid metabolism and insulin sensitivity, and the role of exercise on these parameters [10].High-fat diets could lead to changes in adipose tissue deposition, in mitochondrial functions and in insulin sensitivity. These alterations seem to be very important in the etiology of obesity. In this sense, Schmid, *et al*. studied the principal pathways involved in the response to a high-fat diet in mice [11].

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Received: 7 April, 2020; **Accepted:** 18 April, 2020; **Published:** 24 April, 2020

They proposed that a high-fat diet increases the cell mitochondrial content in brown adipocytes, increasing the capacity of the Krebs tricarboxylic acid cycle particularly. This allows to oxidize the high levels of fatty acids taken up by the cell. Likewise, Santana-Carlos, *et al*. found that deficiency of estrogen and the intake of high fat diet rich inpolyunsaturated fat acids can alone cause the increase of fat depots, adipocytes size, glycemia and insulinemia [12]. But, when the two conditions are together the set of changes worsening the overall condition of the animal and accelerate the onset of metabolic syndrome.

Also, it is important to consider the positive effects of exercise on adipose tissue metabolism and to understand the mechanisms by which this tissue affects the kinetics of other macromolecules. Exercise and the consumption of higher amounts of unsaturated fats, monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) are of primary importance in the treatment of obesity. These two aspects may represent the link between genotypic and phenotypic factors leading to this disease. Motta, *et al*. described the effect caused by exercise interacts with β-cells fat metabolism [13].

They show an abnormal accumulation of fat in the β-cells has been implicated in the pathogenesis of functional islet failure and cell death in type 2 diabetes incident. Further, El-Assaad *et al*. described the fatty acid excess in association with hyperglycemia is toxic topancreatic islets [14]. Moreover, the fatty acids are essential to the insulin secretion stimulated byglucose. In addition, changes in the metabolism of fatty acids may be involved in the physiological adaptations of pancreatic islets in response to the effects caused by physical exercise [15]. In the same way, Motta, *et al*. confirm the HIIT protocol brought benefits in reducing IR and hepatic steatosis where this findings reinforce the importance of HIIT as a non-pharmacological approach to the control of adiposity, insulin resistance, and hepatic steatosis [13].

Some intermittent energy restriction regimens have gained popularity as strategies for achieving weight loss and other metabolic health benefits [16-18]. These paradigms involve recurring periods with little or no energy intake (e.g., 16–48 h), with intervening periods of ad libitum food intake. Studies in rodents have demonstrated that intermittent energy restriction strategies such as intermittent fasting (≥ 60% energy restriction on 2–3 days per week, or on alternate days) and time-restricted feeding (limiting the daily period of food intake to 8–10 h or less on most days of the week) exert beneficial effects on the body composition, energy expenditure, and substrate oxidation. However, clinical studies comparing weight loss with intermittent energy restriction regimens to traditional continuous energy restriction in adults with overweight and obesity are limited.

We have summarized new approaches on high fat diet research explaining the importance of BMI; emphasizing effects of high-fat diet on behavioral eating patterns, body composition, lipid metabolism and insulin sensitivity; the role of exercise on these parameters; the positive effects of exercise on adipose tissue metabolism; and the effects of some intermittent energy restriction regimens. Although scientific evidence clearly shows the effects of high-fat diet on behavioral eating patterns, body composition, lipid metabolism and insulin sensitivity; it also important to characterize relationships between these parameters and the role of exercise and some intermittent energy restriction regimens, emphasizing the timing of dietary intake and other aspects of behavior that are linked to intake.

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How to cite this article: Sandoval C, Santibañez S. "New Approaches on High Fat Diet Research". J Morphol Anat 4 (2020) doi:10.37421/JMA.2020.4.127