FATTENED BY FAT: A FACT… OR ONLY A HYPOTHESIS?

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There is a general agreement in the medical and scientific communities, media and population, to consider that dietary fats are the main contributors of our body fat stores and, more specifically, that the growing prevalence of obesity is caused by the excessive consumption of fats. This is mainly due to the results of retrospective and prospective epidemiological studies conducted in the last 30 years. However, a closer look to these studies raises concern about these conclusions and leads to be more cautious about this inference.

First, descriptive epidemiological studies are not the best approach to determine the role of a macronutrient in a multicomponent mechanism such as the one leading to obesity. Macronutrients are consumed in foods and the way foods are associated, modalities of their consumptions, and more generally healthy eating habits, may lead a particular macronutrient to have different effects on adiposity.

Second, the level of the correlation factors linking dietary fats and obesity in multivariate predictive models is low i.e., ≈ 0.20, and this value is spectacularly consistent across studies although their methodologies are diverse and the tested factors heterogeneous.

Third, the level of this relation is often much lower than the ones reported for factors such as physical activity or the fiber content of the diet, being often only a modulatory factor of their effects on body weight. Thus, it seems that dietary fats may contribute to adiposity only when fiber intake is high or physical activity low. Moreover, recent studies suggest that this may depend on the soluble or insoluble nature of the fibers, comforting the idea that focusing on a macronutrient may lead to erroneous conclusions. This becomes more complex since the polymorphisms of some genes involved in obesity may determine the effect of dietary fats. This has been demonstrated with the fat mass and obesity-associated (FTO), the Apolipoprotein A5 (APOE5) and the Peroxiredoxins (Prxs) genes. The percentage of the population to possess the obesity-prone variant will therefore determine the influence of fats on obesity. This may partly explain the failure of most epidemiological studies to previously report a strong effect of dietary fats on obesity in populations expressing the obesity-prone and obesity-resistant variants of these genes.

Fourth, these studies are not relevant to children since in this population, the relation has often been reported to be negative, a lower consumption of fat being associated with a higher obesity rate, and factors such as eating pattern more contributive than macronutrients *per se*. Since a significant part of adult obesity started during childhood, this should moderate the conclusions about the role of dietary fats in obesity.

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Another point is the specific contribution of saturated fatty acids (SFA) to this supposed dietary-fat induced adiposity. Here again, epidemiological studies seem to indicate that SFA explain most of the reported adiposity-increase effect of the various dietary fats of our diet. But, an exhaustive analysis of results shows that rarely SFA are the only fats involved in the increased obesity rate. Moreover, when various SFAs are considered, only the stearic acid was found to be associated with an increased weight gain. Recent studies suggest that the effect of the percentage of SFAs in the diet may depend on genetics. For example, they seem to explain, more than total dietary fats, the role of various obesity prone variants of genes such as the FTO and, more recently, the Signal transducer and activator of transcription 3 (STAT3). Once again, the frequency of the obesity- prone variant in the population will determine the consequence of this gene-nutrient interaction.

Physiologically, it must be remembered that there are some hormonal conditions for dietary fats to be stored efficiently in adipose tissue, and first of them, an appropriate insulin level. Since insulin secretion is almost entirely under the control of insulin-secreting carbohydrates, focusing on dietary fats may miss the point. Thus, a more reasonable approach should focus on the modalities of the glucose-fat association leading to the most efficient fat storage. Studies show that dietary approaches based on reducing high-glycaemic index foods of the diet are similarly or even more effective than based on low fat intake to decrease body weight.

On a public health plan, it may seem an interesting approach to target dietary fats for achieving a general reduction of adiposity in the population. However, being probably a co-factor of more potent determinants of obesity, the efficiency on body weight loss is always very modest and the preventive effect of low-fat diets in the epidemic of obesity is still to demonstrate. Moreover, it seems the level of dietary fats necessary to reach for observing a significant effect on body weight and adiposity, is much lower than the one that can be reasonably expected. In the present dietary environment and due to the strength of the early conditioning of eating preferences, it is rather unrealistic to expect a dramatic and sustainable reduction of fat intake. This is all the more concerning that some genetic polymorphisms involved in obesity lead to an increased selection of high-fat foods, and therefore may cause great difficulties to lower dietary fats prescription.

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