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Bioenergetics of Obesity: Is Fat Gain a Problem or a Solution?

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According to the first law of thermodynamics, fat gain is the result of a positive energy balance (i.e. energy intake > energy expenditure) [1]. When an individual experiences a chronic positive energy balance, this can lead to the accumulation of excess body fat, and ultimately obesity. Obesity rates have increased so dramatically among industrialized countries in recent decades that it has been referred to as a “pandemic” [2]. Attenuating these high rates of obesity is a high priority in many countries not only from a population health perspective, but from a health care system's economic perspective [3]. However, reversing this trend is a herculean task and will likely require a major change in societal norms and the values underlying those norms [4,5].

A major challenge of today's world is that our so-called “obesogenic” environment is conducive to the consumption of energy and unfavorable to the expenditure of energy, therefore predisposing to a chronic positive energy balance. The modern, money-oriented, computer-dependent, sleep-deprived, physically-inactive human lives chronically stressed in a society of food abundance. From a physiological standpoint, the excess fat gain observed in prone individuals is perceived as a normal consequence (i.e. biological adaptation) to a changed environment rather than a pathological process [6]. In other words, weight gain is a sign of our contemporary way of living or a “collateral damage” in the struggle for modernity [7].

Body fat, when kept to an appropriate level (probably defined by an individual's genetics), is a necessary and healthy energy reserve. Recent evidence suggests that while fat stays in the fat cells, for which they are designed, there is little cause for concern [8]. Body fat begins to cause metabolic problems only when lipid-intolerant nonadipose organs are not protected against lipid “spillover” during sustained energy surplus [9]. In fact, it is increasingly recognized that it is visceral fat and the fat deposited “ectopically” in organs like the liver, pancreas, heart or skeletal muscles that tends to cause increases in cardio-metabolic risk [10]. Interestingly, the accumulation of fat under the skin (i.e. subcutaneous adipose tissue), which is less lipolytically active than other fat depots and therefore able to safely sequester fat away from ectopic depots, seems to be associated with reduced cardiometabolic risk [11,12].

1. What are the Benefits of Body Fat Gain?

Obesity, at least when operationally defined as exceeding a specific amount of body fat and/or body mass index, is associated with certain health benefits. Examples include the now rarely needed but obvious protection against starvation in times of food scarcity, protection against osteoporosis, fractures, frailty, and premature mortality in the elderly, as well as reduced mortality rates in the face of certain severe illnesses or injuries [6]. The assumption that adiposity per se increases mortality risk is also not well supported by the scientific evidence. Many epidemiologic studies have shown that people who are overweight or moderately obese live at least as long as normal weight individuals, and often longer [13-15]. Additionally, life expectancy increased dramatically during the same time period in which body weight rose, and the World Health Organization projects life expectancy will continue to rise in coming decades [16].

From a physiological standpoint, fat gain is seen as a solution for

maintaining homeostasis and re-establishing energy balance in the current obesogenic environment. The ability of fat cells to produce molecules involved in genuine regulatory processes has been known for decades. During weight gain, many adaptations over time can promote the re-equilibration of energy balance. These include increases in fat oxidation, sympathetic nervous system activity, insulinemia at euglycemia, leptinemia, and overall energy expenditure [6]. The problem related to fat gain as a physiological compensation to chronic unhealthy lifestyle habits is that it cannot occur with the same metabolic efficiency as exercise. Specifically, fat gain relies more on increased concentration of substrates (e.g. free fatty acids) and hormones (e.g. insulin and leptin) to re-establish energy balance by increasing total energy expenditure, which likely underlies the occurrence of the metabolic syndrome which often accompanies obesity. Thus, while increasing fat reserves may help to restore energy balance, it can also lead to increased risk of chronic disease. These observations emphasize the importance of adhering to a healthy lifestyle in order to maintain body weight stability rather than relying on the overuse of regulatory systems.

2. What are the Adverse Effects of Weight Loss?

Although counter-intuitive, many prospective observational studies suggest that weight loss increases rather than decreases the risk of premature death [17-19]. Paradoxically, most short-term weight loss intervention studies do find improvements in many health indicators. However, given that intentional weight loss is generally accompanied by a change in dietary and physical activity behaviors, it is not known whether or to what extent the improvements can be attributed to the weight loss per se. The case of liposuction can certainly provide relevant information about the effects of subcutaneous fat loss in the absence of behavior change. In their study, Klein et al. [20] evaluated the effects of large-volume abdominal liposuction on metabolic risk factors in obese women before and 10 to 12 weeks after liposuction. Although the participants lost 10.5 kg of fat, liposuction did not improve obesity-associated metabolic abnormalities, suggesting that decreasing adipose tissue mass alone (and especially reducing subcutaneous fat stores) without behavior change will not achieve the metabolic benefits of weight loss. In contrast, most health indicators can be improved through changing health behaviors, regardless of whether weight is lost or not. For instance, it is well-known that physical activity participation without weight loss has the capacity to reduce visceral adiposity and substantially improve the cardio-metabolic risk profile [21]. Similarly bariatric surgery, which dramatically changes the way

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that food is consumed and digested, has been shown to result in rapid improvements in glycemic control just days after surgery, long before any appreciable weight loss has taken place [22]. These observations agree with the “fat but fit” [23] and “metabolically healthy but obese” [24] concepts and stress the importance of regular physical activity and a healthy diet as key components in any health promotion and disease prevention strategy, regardless of body weight.

Moreover, attempts to sustain weight loss invoke adaptive responses involving the coordinated actions of metabolic, neuroendocrine, autonomic and behavioral changes that oppose the maintenance of a reduced body weight. The multiple systems regulating energy stores and opposing the maintenance of a reduced body weight illustrate that body energy stores and fat stores in particular are actively “defended” by interlocking bioenergetic and neurobiological systems. The fact that more than over 80% of individuals return to pre-weight loss levels of body fatness after otherwise successful weight loss certainly illustrates this “sad” reality [25], whereas studies of people successful at sustained weight loss indicate that the maintenance of a reduced degree of body fatness requires a lifetime of meticulous attention to energy intake and expenditure [26]. Among the adverse effects of weight loss, it is well-known that body fat loss complicates appetite control, reduces energy expenditure to a greater extent than predicted, increases the proneness to hypoglycemia and its related risk towards depressive symptoms, increases the plasma and tissue levels of persistent organic pollutants that promote hormone disruption and metabolic complications, and increases psychological stress, all of which are adaptations that significantly increase the risk of weight regain [27].

Concluding Remarks

The general perception of obesity today is more pessimistic than optimistic with words like “global epidemic”, “important problem” or “scourge” that are omnipresent in the popular media as well as in the scientific community. The corollary of this widespread message is obviously a greater pressure on obese individuals towards weight loss. However, an accumulating body of evidence suggests that a focus on weight loss as an indicator of success is not only ineffective at producing thinner, healthier bodies, but also damaging, contributing to food and body preoccupation, repeated cycles of weight loss and regain, reduced self-esteem, eating disorders, and weight stigmatization and discrimination [28]. It is thus time to shift the focus away from body weight and center our efforts on the promotion of a healthy lifestyle if we really want to have an impact on the health of the population in the long term and reduce the burden on individuals with obesity.

Our better understanding of obesity, as witnessed by an impressive amount of publications in the field over the last decades, suggests that body fat can be both detrimental and protective. Simplistic messaging that body fat is “bad” and weight loss is “good” for our health can be misleading and ignores the truth about the biological response and side effects of weight loss, as well as the importance of fat gain in maintaining body homeostasis in a “toxic” environment. Fat gain is part of a regulatory strategy that permits the recovery of energy balance and body weight stability in a world that has increasingly added obesogenic factors to our lifestyle over time. In this context, a preventive approach that includes healthy lifestyle habits and that attempts to reduce the obesogenic nature of our society is the only long-term viable solution to maximize our health, even if it does not easily fit within the priorities of an economically globalized world.

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