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A Calorie is Still a Calorie, According to Rigorous New Evidence

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Introduction

Chronic degenerative "diseases of civilization" are the top challenges of all health systems globally[1]. Among them, obesity is the most pernicious because it a) has survived several meritorious lifestyle initiatives undeterred, b) is associated with intractable environmental promotion, c) there is no long-term medical treatment available, but also d) negatively affects several other cardiovascular risk factors, and e) is the leading cause of preventable life-years lost in the United States[2]. The misbelief that an unrestrained, permissive "diet," effortless exercise "program", or magic "diet pill" may exist continues to permeate popular thinking and media pieces. The combination of such fantasies, denial, and rejection of scientifically proven nutrition principles has enabled the triple epidemics of obesity, diabetes, and cardiovascular risk to continue. One of the controversies that recur is the question of whether a low-fat or low-carbohydrate is "healthier"[3]. Some authors argue a special case which, in their view, will help clarify the issue: do dietary calories count and are they equal? Classical experiments, particularly ones that used meticulously-controlled environments and metabolic ward measurements, indicate that calories do count[4]. Usually considered basic physiology, obesity ultimately results from a calorie intake in excess of calorie utilization, and the initial clinical approach is to correct this imbalance[5,6]. When done, it succeeds. Calorie utilization is comprised of the energy cost of resting metabolic processes (REE), physical work performed, and the thermal effect of food (TEF), with the most practical, safe and clinically meaningful target being physical activity.

In contrast with the classical, accepted paradigm of excessive dietary calories as the most important cause of weight gain, a new "carbohydrate–insulin model" has been proposed[7,8]. According to this model, individuals may consume far fewer calories than they need to maintain their weight-according to conventional calculations-and still gain weight[7]. This theory posits that carbohydrate (CHO) intake drives insulin release, which partitions energy storage in adipose tissue, rather than allowing fat oxidation. The brain responds to this internal energy deficit in working cells by increasing hunger and depressing the metabolic rate, furthering fat deposition. Low CHO diets therefore, "release" this partitioning, and allow oxidation of fat by working cells, eg, muscle, leading to weight loss. Hence, the number of calories consumed is unimportant compared to the type of macronutrient, and is *alone* irrelevant in the pathogenesis of obesity. The "metabolic advantage" purportedly associated with a low CHO diet offered is a unique ability to oxidize great amounts of fat. In contrast with prior alternative theories of obesity, the CHO-insulin hypothesis may be verified directly, provided that dietary intakes and energy variables are meticulously controlled and measured.

This bold theory, however, is at odds with much metabolic data collected over the preceding decades, and is not supported by specific new data. For instance, isocaloric overfeeding results in weight gain whether fat or CHO is consumed[9]. Despite much discussion and theoretical debate, followed by screening for outstanding credentialed experimentalists, a series of rigorous experiments was commissioned by proponents, including Taubes, the cofounder of Nutritional Science Initiative (NuSI), and cosponsored by the National Institutes of Health.

In a highly-controlled metabolic ward study, Hall and

coworkers[10] randomly assigned 19 obese female and male subjects to either a diet with a 30% calorie restriction in CHO or a diet with 30% calorie restriction in fat. Participants were exposed to these two diets separated by a wash-out period in a cross-over fashion; RQ, body composition, rates of metabolism, fat oxidation and fat loss were measured along with insulin and other hormones. Despite a decrease in insulin levels associated with the low-CHO diet, there was no corresponding rise in weight loss. Specifically, during the low-CHO diet period, there was an average loss of ~245 g body fat, compared to a higher average loss of body fat, ~463 g, during the low-fat diet period.

Another rigorously-controlled feeding study sought to determine changes in energy expenditure, respiratory quotient (RQ) and body composition using an isocaloric low-CHO ketogenic diet (KD)[11]. Seventeen overweight or obese men were fed a high-CHO baseline diet (BD) on a restricted ward for 4 weeks, and a ketogenic diet (KD) with clamped protein intake for another 4 weeks. Two days weekly, each subject was placed in a metabolic chamber for measurement of energy expenditure (EE), sleeping EE (SEE), and RQ. The average EE expended during the two diet periods, baseline *versus* low-CHO, rose by a biologically insignificant ~151 kcal/d, accompanied by a fall in both the rate of body fat oxidation and loss of fat-free mass.

According to the CHO-insulin hypothesis, lower CHO intakes, as compared with isocaloric amounts of fat, would be associated with increased EE, increased fat oxidation, and loss of body fat. Contrary to the hypothesis, the data in the aforementioned metabolic study demonstrated that the low CHO-KD was associated with barely measurable rises in EE and no increase in loss of body fat. The authors' interpretation was that the absence of physiologically important changes in EE or body using the two isocaloric diets confirms that "a calorie is a calorie." The principle investigator in these two studies subsequently published a review which placed the findings in perspective[12].

Conclusion

Outpatient studies involving nutrients commonly involve insurmountable methodological and/or practical limitations. Even in the two well-designed studies discussed, future replication in larger randomized controlled trials is needed. Nonetheless, these metabolic studies not only provide their own important data, but pointedly add to the already considerable data base[13]. In particular, the carbohydrate–insulin hypothesis predicts a low-CHO diet, as compared with a isocaloric high-fat diet, will lower plasma insulin, cause a release of sequestered fat from adipose tissue, which will then be oxidized and lead to weight loss. These two rigorous studies, which may serve as a model for larger studies, found the opposite. The greater significance is that data which form the basis for public health policy must be of the highest caliber, using objective, repeatable, meticulous design. In weighing merits of hypotheses, Carl Sagan advised "Extraordinary claims require extraordinary evidence."

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