

Evaluating the Benefits of Canine Carbohydrate Restriction:

Are Carbohydrates More Fattening For Dogs Than Other Macronutrients?

Evidence Review
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Abstract— Recently it has been suggested that, “calorie-for-calorie,” carbohydrates are more fattening than other nutrients for dogs (as well as other non-ruminant mammals) due to the unique hormonal changes wrought by carbohydrate consumption. This white paper evaluates the strength of this thesis and concludes that it is supported by persuasive (though non-dispositive) experimental evidence and explained by a coherent and compelling pathophysiological model. In light of these findings, we suggest that veterinary nutritional authorities reconsider the received wisdom that, when it comes to the nutritional management of canine body composition, “a calorie is a calorie.”

EVIDENCE REVIEW

The notion that “a calorie is a calorie” has long been considered an article of faith in the professional understanding of small mammal obesity. It is endorsed (both implicitly and, at times, explicitly) in leading veterinary nutrition reference books and research compendia, in the great majority of academic papers on the subject of obesity, in the regulatory framework governing the sale of pet foods in the United States, and in effectively every other professional setting in which the topics of small animal adiposity and obesity are being discussed.

But in recent years there has emerged a sizable (and growing) body of evidence that challenges the assumption that all macronutrients have an equivalent impact on overall body fatness and instead suggests that carbohydrates are particularly fattening nutrients, at least for humans. This research has been bolstered by the development of a theoretical model (the so-called “Carbohydrate-Insulin Model of Obesity”) that folds this body of research into the broader understanding of the pathophysiology of obesity.

The Carbohydrate-Insulin Model (CIM) posits that obesity is fundamentally an endocrine disorder, one which results from chronically elevated levels of the anabolic hormone *insulin*. The three conceptual building blocks of CIM are simple and

uncontroversial. Essentially, the model provides as follows: (1) consumption of dietary carbohydrates causes blood glucose levels to rise, (2) elevated blood glucose levels induce an attendant pancreatic insulin response, and (3) elevated blood insulin levels increase the rate at which glucose and other nutrients are drawn into and retained within adipose tissue, causing fattening. Carbohydrates beget insulin and insulin begets fattening, or so the theory goes.

Over the past ten years, CIM has been endorsed by several leading figures in the field of human nutrition. But to date it has received little attention from their veterinary colleagues. This is surprising to us, considering how well CIM seems to fit the biology of small mammals such as dogs and cats. As explained elsewhere in this white paper series, unequivocal evidence demonstrates that carbohydrate consumption is the primary nutritional driver of canine blood glucose levels. And equally definitive research confirms that, in dogs as in humans, blood glucose levels are the primary drivers of pancreatic insulin production. As one French research team recently described the matter, “the same factors account for postprandial glycemic and insulinemic responses in both human beings and dogs” (Nguyen et al. 1994).

It should come as no surprise, then, that feeding studies tend to find that carbohydrate-rich diets make cats and dogs fatter than isocaloric low-carbohydrate diets. Our review of the research has uncovered six experiments published in the last 15 years in which relatively low-carbohydrate diets were found to reduce the fat mass of dogs or cats more effectively than isocaloric high-carbohydrate diets (Dietz et al. 2002; Berier et al. 2004; LaFlamme et al. 2005; Hoenig et al. 2007; Vasconcellos et al. 2009; des Courtis et al. 2015). Indeed, at times these fat-loss differences have been truly profound. In the experiment featuring the largest study group (Bierer et al. 2004, 39 dogs), for instance, the low-carbohydrate population lost an average of *six times as much body fat* over 12 weeks as the isocaloric high-carbohydrate population.

There are, however, at least two reasons to approach such findings with at least some measure of caution.

First is the fact that at least one recent study (Bergman et al. 2003) potentially constitutes competing evidence. There, a group of six dogs were shown to gain a small amount (less than 1 kg) of body fat over a twelve week study period during which 110g of their usual high-carbohydrate diet was swapped for a supposedly isocaloric amount of pure bacon fat each day. A closer look at the study reveals, however, that the experimental diet was not truly isocaloric in design, despite being labeled that way by the authors. The data suggest instead that the experimental diet added approximately 5,000 calories to the dogs' diets over the twelve week study period, a caloric surplus that would fully explain the <1kg increase in fat mass.

(It should also be noted that the pair of experiments conducted by Dale Rosmos and colleagues in the 1970s, often cited as evidence that carbohydrates are actually *less* fattening than other nutrients for dogs calorie-for-calorie, in fact have little or no bearing on the subject whatsoever. This is because neither experiment was isocaloric in design—dogs were fed *ad libitum* in both cases. And, in both cases, the researchers reported the unremarkable finding that dogs which ate more calories also gained more weight. Such a study design clearly makes it impossible to evaluate the per-calorie impact on adiposity of carbohydrates, fats, and proteins, respectively.)

A second reason to temper enthusiasm for the sizable body of experimental evidence supporting CIM in small mammals is the fact that an alternative (and somewhat less controversial) physiological mechanism might also be used to explain the underlying findings. There is strong evidence that the “thermic effect” of dietary protein (i.e., the amount of energy required to digest and metabolize the ingested nutrients) is some 10-15% higher than the thermic effect of either dietary carbohydrates or fats. Thus, all else being equal, one should expect a high-protein diet to induce at least somewhat greater weight loss than an isocaloric diet in which some of the protein is swapped for an equivalent amount of carbohydrate. In our view, however, such a small incremental increase to overall metabolic rate is unlikely to be the sole explanation for the significant fat loss observed in the studies discussed above. It is more likely that some other mechanism—such as CIM—is contributing as well, and likely playing the more dominant role.

OVERALL SUPPORT FOR PRIMARY THESIS

9/10: Strong and consistent evidentiary support, belied to a more limited degree by competing evidence and counterarguments.

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