The Rise and Fall of the Cafeteria Diet: Some Observations

Dear Dr. Hurley:

In a recent article published in THE JOURNAL OF NUTRITION (1), Dr. Barbara J. Moore defended the view that the so-called "cafeteria" diet was not an appropriate tool for the studies of thermogenesis. Her main criticisms were concerned with the lack of reproducible results when using this kind of diet and the fact that diet-induced thermogenesis caused by cafeteria diet was not due strictly to this diet but rather to the macro- and micronutrient imbalance in it. While I agree that some chaos has developed as a result of the use of the term cafeteria diet, I think that this kind of experimental model is, indeed, suitable for thermogenesis studies and it possibly constitutes the nearest animal model to the voluntary hyperphagia encountered in humans.

In 1976 Sclafani and Springer (2) attempted to produce dietary obesity in rats by feeding them an assortment of highly palatable supermarket foods in addition to a nonpurified diet. Although the animals rapidly gained weight, it was later demonstrated that they showed an imbalance between weight gain and energy intake due to enhanced diet-induced thermogenesis (3–4). This kind of diet was called cafeteria diet. Since then, a plethora of studies concerning the metabolic environment of animals fed this diet have been published.

The cafeteria diet offers the animal a wide variety of items of different composition and taste. Usual components of this kind of diet are cookies, pastry, nuts, chocolate, candy, bacon and other meat products, cheese, milk, water and also nonpurified rat diet pellets. Some cafeteria diets, however, differ from the original conception. For example, in some cases the different items are changed regularly (4) and the animal has no chance to develop its feeding preferences. Others offer little variation in taste because they only provide two or three different items and do not allow the animal to have tap water ad libitum (5). It is also essential that this diet contains a mineral and vitamin complement (as a supplement in liquid) to avoid interference from deficiencies. I agree with Dr. Moore that some anarchy has developed regarding the standardization of this kind of diet. Thus, the term cafeteria feeding should be restricted to diets offering the animal nonpurified rat diet pellets plus a variety of snack-type foods normally consumed by humans in addition to milk, water, and vitamin supplements. This diet places the animal in a situation which is very similar to that encountered by humans, providing a model of obesity secondary to chronic voluntary hyperphagia, and can thus be recognized as the closest approximation to the majority of human cases in which obesity is induced by voluntary hyperphagia of energy-rich food. Regarding composition, an animal subjected to such a diet has basically the same protein and carbohydrate intake as a normally fed one but a significantly greater proportion of lipid, thus making the cafeteria diet a high-fat diet, with an adequate intake of both protein and carbohydrate (6, 7). In addition, the animals do not ignore the nonpurified diet pellets and, although some animals prefer one particular item, they also tend to consume other items; their energy intake is independent of the items chosen. If the cafeteria diet were to be discarded just because not all animals eat the same items offered or because the ingredients may have different compositions from batch to batch, then one would have to discard all the studies carried out with humans.

When adequately supplemented, a cafeteria-fed rat does not necessarily have a lower vitamin or mineral intake. If the supplement is given separate from the rest of the items, a deficiency can occur, since the flavor of this kind of supplement does not normally appeal to the animal, and the animal simply does not eat it. However, if the supplement is properly mixed with the milk—one of the cafeteria items—the animal will have no micronutrient restrictions. In any case, a low dietary vitamin or mineral content does not alter thermogenesis unless there is a severe deficiency. Furthermore, essential metal levels are maintained (7) in this kind of diet. In addition, homeostatic ability allows for relatively wide changes in mineral and vitamin intake before deficiencies develop. The lack of abnormal growth or reproductive function supports the view that the animal is not affected by a nutritional deficiency.

Cafeteria rats consume more than twice as much energy as control rats given a pelleted nonpurified diet, and most of the extra energy is liberated as heat and called diet-induced thermogenesis (DIT). Brown adi-

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pose tissue has been recognized to be a site for this kind of thermogenic response. Large inter-strain differences in DIT and energetic efficiency have been observed in cafeteria rats (8), which are dependent on genetic strain but independent of the experimental methods employed. Rothwell and Stock (9) have observed an 84% increase in heat production in very young rats given a cafeteria diet. When these animals were returned to the normal nonpurified diet they showed a lower energetic efficiency than controls continuously fed the nonpurified diet. Thus the metabolic adaptations to the cafeteria diet were still detectable when the animals were eating the same amount of the same diet as controls.

It has been suggested that an alternative way to stimulate hyperphagia in rats is to supplement their diet with sucrose or fat. However, the use of fat-enriched diets promotes stress and protein deficiency and the animal also lacks the state of natural hyperphagia encountered as a result of the cafeteria feeding.

In conclusion, while one has to agree that not all the studies carried out involving cafeteria diets have been performed correctly (mainly because of the lack of variety in the diet), the cafeteria diet is an appropriate tool for studies of thermogenesis that can lead to clean results; and, most importantly, it is a stressless technique for producing hyperphagia and lowered energetic efficiency in experimental animals, which is more similar to some human obesity syndromes than any other model, because in addition to hyperphagia and obesity, it also stimulates the hyperinsulinemia, insulin resistance and glucose tolerance (5) which are observed in human obesity.

Sincerely,

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Reply to the Letter of Dr. Argilés

Dear Dr. Hurley:

I agree with Dr. Argilés concerning the effectiveness of the cafeteria diet in stimulating overeating. There is no doubt that hyperphagia is stimulated when such a diet is offered to rats. This is an interesting and important observation. The question is how to study the phenomenon without sacrificing nutritional control over the experiment.

There are several questions being asked: *1*) What is stimulating the hyperphagia? Is it the *variety* of food offered or is it the nature (composition) of the food offered? *2*) What are the effects of the cafeteria diet (increased thermogenesis, decreased efficiency, etc.)? Are these effects attributable to the hyperphagia per se or are they attributable to the nature (composition) of the diet? Regardless of the question being asked, it is necessary to control the diet. Without such control, the results will be confounded and uninterpretable, regardless of how interesting they may be.

I fear Dr. Argilés's letter is somewhat misleading. When he says, "I agree with Dr. Moore that some anarchy has developed regarding the standardization of this kind of diet," he suggests that I think the diet *could* be standardized. I do not.

Sincerely,

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