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Letter to Editor

Response to Dr George Bray's review of *Good Calories, Bad Calories*

Dear Editor,

I would like to thank Dr Bray for recommending *Good Calories, Bad Calories* (*GCBC*) and noting that it contains important information. However, his review did not accurately reflect the contents and arguments of my book, and so may ultimately serve only to confuse the issues discussed, rather than clarify them.

Dr Bray makes several significant errors of fact that will certainly mislead readers as to the book's actual content and arguments. Bray asserts, for instance, that *GCBC* includes 'a number of errors of omission or commission that are important when relating diet to disease'. The first, he says, is that I make 'no mention' of low- or high-density lipoproteins in the diet-heart discussion of the book. These are indeed discussed at great length in that context, and Bray's assertion could have been easily checked by looking in the index (HDL, 139, 154, 161–3, 165, 166–8, 169, 172–3, 182, 184, 185, 187, 196, 223. LDL, 141, 154–6, 160–1, 163, 165–8, 170–6, 184, 187, 193, 196, 200–1, 205n).

Regarding obesity, Bray makes his first critical factual error in the second sentence of his abstract. He asserts that I believe 'that you can gain weight and become obese without a positive energy balance'. This statement implies that I do not believe in the first law of thermodynamics. It could not be further from the truth. In *GCBC*, I point out that the first law – energy conservation – tells us nothing about what causes obesity. It says that an increase (or decrease) in the energy of a closed system – fat mass, in this case – must be associated with an energy intake greater (or less) than that expended. It says nothing about what causes that imbalance. It implies no causality. I then suggest what European researchers concluded prior to World War II: the cause of obesity – and so of the positive energy balance that must accompany increasing body mass – is not gluttony and inactivity, but a metabolic-hormonal drive to accumulate excess fat.

Because Bray's critique confuses many of the issues in *GCBC*, I would like to spend a few short paragraphs clarifying them. They are quite simple. The hypothesis favoured by Bray and a half century of authorities on human obesity is that fat accumulation is fundamentally caused by positive energy balance, which in turn is driven, as Bray writes, by '"environmental" agents, such as tasty, inexpensive food in large portion sizes, inactivity, viruses, toxins, and social interactions, that interact with the genetically susceptible host to produce obesity'.

The alternative hypothesis begins with the fundamental observation that obesity is a disorder of excess fat accumulation and then asks the obvious question, what regulates fat accumulation. This was elucidated by 1965 and has never been controversial. 'Insulin is the principle regulator of fat metabolism', as Solomon Berson and Rosalyn Yalow described it then, and the mobilization of fatty acids from the fat cells 'requires only the negative stimulus of insulin deficiency' (1). This is why George Cahill, a co-editor of a 1965 American Physiological Society Handbook of Physiology dedicated to this research, (2) recently summarized the relevant science as 'carbohydrate is driving insulin and insulin is driving fat' (*GCBC*, p. 393). Thus, the alternative hypothesis: excess fat accumulation is caused fundamentally by the effect of dietary carbohydrates on insulin and of insulin on adipocytes. In this hypothesis overeating and sedentary behaviour – i.e. positive energy balance – are compensatory effects of accumulating excess fat, not causes.

To call positive energy balance the *sine qua non* of obesity, as Bray does, is no more meaningful than to describe a lack of energy as the *sine qua non* of chronic fatigue. It tells us nothing about why the person is in positive energy balance or lacks the energy to function. It provides no information about the cause of the disorder. Bray acknowledges this fact: 'I see nothing inconsistent with the truth of the idea,' he says, 'that a positive energy balance produces obesity and the idea that it does not tell us why this imbalance occurred'. But he then says, 'Let me make my position very clear. Obesity is the result of a prolonged small positive energy surplus with fat storage as the result,' yet another way of phrasing the notion that positive energy balance *causes* excess fat storage.

As I note in *GCBC*, this inference of causality is logically indefensible. Vertical growth, too, if accompanied by increasing body mass, must be associated with positive energy balance. No one, however, (not even Bray, I presume) would state that children grow because they overeat or that their growth 'is the result of prolonged small positive energy balance'. Rather children overeat because they're growing. The causality is reversed. Understanding the true causality is critical to understanding the

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phenomena. The underlying cause of this vertical growth and its accompanying positive energy balance is hormonal – the secretion of growth hormone.

The question posed in *GCBC* is why we rightfully focus on hormonal regulation when discussing growth abnormalities – gigantism, for instance, or dwarfism – but insist on discussing abnormalities of fat accumulation – obesity and anorexia – as though fundamentally caused by eating behaviour without attending to the hormonal regulation of fat tissue.

Much of Bray's critique hinges on his assertion that I believe that obese individuals do not eat more than lean individuals. He quotes a line from *GCBC*, but by doing so out of context directs attention away from the critical observation that must be explained. 'Even if it could be established', I wrote and Bray quotes, 'that all obese individuals eat more than do the lean – which they don't – that only tells us that eating more is associated with being obese'.

The keyword in the sentence, however, is 'all'. It must be the case, as discussed in *GCBC*, that the obese tend to eat more than the lean, because they tend to expend more energy than the lean. This does not mean, however, that all lean individuals expend less energy than all obese individuals of comparable height, sex and bone structure. The distributions of calories consumed overlap, as do the distributions of calories expended. This is the observation that requires explanation. I do not mention doubly labelled water in this context, because the necessary observations were made with calorimeters nearly a century ago (3).

In this context, Bray's statement 'that obese people eat more food energy than do lean people' is either meaningless – is he indeed claiming that it's impossible to find lean individuals who naturally expend more energy on a daily basis than obese individuals of comparable height, sex and bone structure? – or it is indefensible. The relevant point is how greatly energy expenditure and metabolic rate 'might differ between any two individuals of equal weight, or how similar [they] might be among individuals of vastly different weights' (*GCBC*, p. 278).

Bray also consistently confuses associations – the obese eat more than the lean; the obese are in positive energy balance as they fatten – with causes and effects. Do they get fatter because they overeat, as Bray continues to imply, or do they overeat because they're getting fatter. The goal of science is to correctly determine causality. In these two competing hypotheses, the causalities are diametrically opposed.

Bray argues that high-fat diets cause obesity, but in *GCBC* I note that we can find populations that achieve spectacular obesity eating very-low-fat diets – Sumo wrestlers, for instance, whose fattening diets are only 9–16% fat (4). One implication of the co-existence of malnutrition with obesity in impoverished populations – as reported in 1928 in reservation Sioux (5), in the 1960s in Trinidad (on a diet of 21% fat) (6) and Chile (7), in the 1970s in Jamaica (8), and is now a common observation (9) – is that it's possible to develop obesity in cultures that are physically active by modern standards and that subsist on diets lacking significant or 'excess' calories and certainly lacking what Bray calls 'tasty, inexpensive food in large portion sizes'.

One reason why the Atkins diet is scientifically compelling in this context is that it is a very-high-fat diet, and yet leads to weight loss, at least in the short term, not weight gain. The question is why? And if a very-high-fat diet induces weight loss, why would a merely high-fat diet lead to weight gain?

It is true that certain strains of mice and rats get fat, as Bray notes, when large amounts of hydrogenated vegetable oil are added to their chow, but this says precious little about what happens in humans (or even other strains or species of rodents), nor does it tell us whether the added fat or the carbohydrate-rich chow is the obesogenic factor. To the best of my knowledge, experiments have never been done to determine which of the two is critical.

Bray repeatedly dismisses my observation that positive energy balance tells us nothing meaningful about weight regulation by referring to it with the rhetorically loaded phrase 'calories don't count'. He then cites Kinsell's 1964 article – 'Calories Do Count' – as showing 'clearly' that calories, not nutrient composition, play the critical factor in weight loss. Bray neglects to add Kinsell's own observation that carbohydrate-restricted diets inhibit hunger in a way that calorie-restricted diets do not. 'There is a good reason to believe that the satiety value of [carbohydrate-restricted] diets is superior to diets high in carbohydrate and low in fat', Kinsell wrote, 'and hence, may be associated with better dietary adherence' (10). This observation is made repeatedly in the literature, suggesting that macronutrient composition significantly affects hunger and satiety – another observation that must be explained. From *GCBC*, p. 347: 'Even if we could establish that weight loss on [carbohydrate-restricted diets] is universally attended by a decrease in calories consumed . . . we then have to explain why the subjects of these diets don't manifest the symptoms of semi-starvation. If they eat less on these diets, why aren't they hungry? And if they don't eat less, why do they lose weight?' Significant fat loss on carbohydrate-restricted diets, unrestricted in calories, is the kind of paradoxical observation that might actually inform our understanding of the true aetiology of the disorder itself. It should not be dismissed without careful experimentation.

One goal of *GCBC* is to motivate investigators in this field to take a more rigorous, strictly scientific approach to their research, rather than taking critical issues on faith because they agree with their preconceptions. The book attempts to establish that compelling evidence indeed exists for an alternative hypothesis of obesity, and that the disor-

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der is fundamentally caused by the influence of carbohydrates on insulin and insulin on fat accumulation, not by eating too much or sedentary behaviour as has been dogma for decades. Indeed, what we have known since the 1960s about the hormonal/enzymatic regulation of adipose tissue suggests that easily digestible carbohydrate-rich foods *should* make us fat, and this was accompanied until the early 1970s, when Bray and a handful of his colleagues became the authority figures in this field, with more than a century of conventional wisdom that they do. 'Every woman knows that carbohydrate is fattening', as a *British Journal of Nutrition* article noted in 1963 (11).

Finally, I would like to identify one potential conflict of interest on Bray's part that he neglected to mention. In the 1970s, as I discuss in *GCBC*, the hormonal/enzymatic regulation of fat tissue was deemed irrelevant to the cause, cure and prevention of human obesity. I identify Bray as one of two individuals most responsible for this dubious accomplishment, and 'for effectively removing the [century-old] concept of the fattening carbohydrate from the nutritional canon . . .' (*GCBC*, p. 417). Thus, Bray's critique of *GCBC* may be as much a defence of his own career as it is an unbiased assessment of the book. Readers should be aware of this possibility. It would be a shame if obesity researchers based their opinions on Bray's review, rather than the book itself.

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References

1. Berson SA, Yalow RS. Some current controversies in diabetes research. *Diabetes* 1965; **14**: 549–572.
2. Renold AE, Cahill GF. Jr. (eds) *Handbook of Physiology. Section 5. Adipose Tissue*. American Physiological Society: Washington DC, 1965.
3. Benedict FG, Emmes LE. A comparison of the basal metabolism of normal men and women. *J Bio Chem* 1915; **20**: 253–262.
4. Nishizawa T, Akaoka I, Nishida Y, Kawaguchi Y, Hayashi E. Some factors related to obesity in the Japanese Sumo Wrestler. *Am J Clin Nutr* 1976; **29**: 1167–1174.
5. Stene JA, Roberts IL. A nutrition study on an Indian reservation. *J Am Dietet Assoc* 1928; **3**: 215–222.
6. McCarthy C. Dietary and activity patterns of obese women in Trinidad. *J Am Diet Assoc* 1966; **48**: 33–37.
7. Arteaga A. The nutritional status of Latin American adults. In: Scrimshaw NS, Moises B (eds). *Nutrition and Agricultural Development*. Plenum Press: New York, 1974, pp. 67–76.
8. Richards R, de Casseres M. The problem of obesity in developing countries: its prevalence and morbidity. In: Burland WL, Samuel PD, Yudkin J (eds). *Obesity*. Churchill Livingstone: New York, 1974, pp. 74–84.
9. Caballero O. A nutrition paradox – underweight and obesity in developing countries. *N Engl J Med* 2005; **352**: 1514–1516.
10. Kinsell LW, Gunning B, Michaels GD, Richardson J, Cox SE, Lemon C. Calories do count. *Metabolism* 1964; **13**: 195–204.
11. Passmore R, Swindells YE. Observations on the respiratory quotients and weight gain of man after eating large quantities of carbohydrate. *Br J Nutr* 1963; **17**: 331–339.

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