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Why Diets Make Us Fat

Rachel Fox

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BOOK REVIEW

Why Diets Make Us Fat, edited by Sandra Aamodt, New York, NY, Current, 2016.

Sandra Aamodt's 2016 book *Why Diets Make Us Fat* delivers exactly what the title promises: a comprehensive overview of the science behind why deliberate attempts to become thinner strongly predict weight gain over the long term. In other words, if we want to avoid getting fatter, as either individuals or as a population, we must eliminate diets immediately and completely.

This conclusion is the book's biggest strength as well as its biggest weakness. On one hand, the evidence Aamodt presents is impossible to deny. From brain anatomy and gut physiology to nutrient breakdown and the history of food advertising, she makes an airtight case that every argument in favor of dieting is false. On the other hand, however, her message is far more antidiets than it is fat positive. She may advocate for the benefits of exercise regardless of weight and condemn the harmful effects of weight stigma, but the unquestioned goal driving the whole book is to reduce the number of fat people in the world. If diets are bad because they make us fatter, then fatness is still being presented as an abject embodiment, something we cannot do anything to change once it exists, but nonetheless something to be prevented at all costs. This takeaway is especially frustrating given how strongly Aamodt emphasizes that lower weight does not necessarily result in lower mortality risk.

The book begins with Aamodt's own story of dieting failure and her initial foray into intuitive eating. Much like many people who begin this practice, she was shocked to find that her weight stabilized and her relationship to food improved when she stopped trying to lose weight. Inspired by these results, Aamodt began a mission to use her neuroscientific expertise and scientific literacy to figure out why our bodies are so resistant to dieting, why the weight of the average person in the United States increased between 1975 and 2005, and what should be done in light of these discoveries. Her conclusions—that dieting is unhealthy and leads to permanent physiological changes, that dieters almost always regain the weight they lose, and that our environment does much more to cause fatness than individual choices—are not new. Instead, what this book has to offer is a clear and thorough explanation of the physiological and psychological mechanisms behind these phenomena. The science is where this book shines, and as such, this review will focus on that content.

Aamodt starts Part One, "The Trouble with Diets," by laying out the newest iteration of set point theory, defended weight range (see Linda Bacon's [2008] *Health at Every Size* for more detail about set point theory). The defended range is a target body weight that the brain does its best to maintain. Due to evolutionary mechanisms designed to protect against starvation, this range can go up over time, but is extremely unlikely to come down, and any prolonged time spent below this range triggers the brain to initiate a full-body conservation regime to return to it. Regardless of starting weight, quick and substantial weight loss increases both hunger and the rewards of eating by reducing fat stores. This depletes the cells that produce the hormone leptin, which is responsible for satiety. Less circulating leptin tells the brain that there is not enough stored energy available, which then creates the drive to eat. Depleting fat stores also increases the production of the hormone ghrelin, which activates "hunger neurons" in the hypothalamus, leading to "ravenous levels" of hunger (77). As Aamodt points out, many people assume that the brain knows the difference between "a normal-weight person and an obese person when it comes to weight loss," but this evidence indicates otherwise (9).

The starvation response cannot be overcome with willpower alone. Willpower is an executive function, and therefore a finite resource, meaning any willpower we spend on suppressing hunger signals leaves less willpower for other parts of our lives; correspondingly, any time we are distracted by other things, we have less willpower to devote to dieting. Moreover, using willpower to stay in a starvation state leads us to disregard the hunger and satiety signals our bodies naturally send, which helps to explain why dieting usually leads to weight *gain* once we stop starving ourselves.

Thinking it is in a state of starvation also causes a great deal of stress for the body. In addition to increasing hunger, the starvation alarm in the brain lowers metabolism and disincentivizes physical activity—the opposite of what most dieters are looking for. For some dieters, this explains why keeping weight off requires an ever-increasing amount of exercise and an ever-smaller intake of calories. More often, however, this stress causes the brain to enter a state of emergency, causing inflammation and cellular damage in the hypothalamus, which can permanently decrease its sensitivity to leptin. This is one possible mechanism by which the defended range increases—even one starvation episode can lead to leptin resistance (meaning it's harder to tell when we're full), often beginning a self-sustaining cycle of weight gain, stress, inflammation, and more weight gain. The physical stress of starvation is intensified by the social stress of weight stigma. In addition to fatphobia, compounded discriminations, such as racism or sexism, as well as persistently stressful conditions, such as poverty, all raise inflammation and increase levels of the stress hormone cortisol.

In Part Two, “Why We Gain Weight,” Aamodt demonstrates that the conventional wisdom about “calories-in, calories-out” fails because our genes, our environments, our ancestry, our gut bacteria, and thus our metabolisms, are different and unique. Even if most foods had labels with accurate caloric counts (which they do not), the number of calories released in digestion would vary depending on its efficiency. Smaller food pieces and cooked foods take less energy to break down. Different macronutrients (fats, carbohydrates, proteins, etc.) also take different amounts of energy to digest. And that is all without considering our individual gut bacteria, which can determine how efficiently we break down certain foods by providing specific digestive enzymes, changing the rate at which food moves through the intestine, or modifying how nutrients are released into the blood stream. Weight is also controlled through genetics. Body weight variation is 50–70% heritable and body fat distribution, which affects body shape, is 70–85% heritable. This is not due to the work of one master gene, but rather to “a large set of genes with small individual effects” (133). These genes can be influenced by womb conditions, toxin exposure, and even epigenetic modifications passed down through multiple generations. Moreover, due to gene-environment interactions, these genetic predispositions will lead to particular outcomes, such as ease of weight gain or loss, only in certain situations.

On top of these individual factors, the food industry and the diet industry (which are not always separate) have a major presence in the public sphere and in legislation. Food advertising, food processing, and government subsidizing have all increased in the past four decades, essentially ensuring that we consume more calories than prior to these increases. Unsurprisingly, these factors caused an average weight gain of 20 lb/person between 1975 and 2005 (although that gain has plateaued since 2005), but this gain has not been evenly distributed. Instead, people with slender parents have remained slender and people from average-weight families have gained only a few pounds. People from “overweight” families are, on average, about 10 lbs heavier than their parents, which is “often enough to just barely edge them into the obese category. That explains how a twenty-pound average weight gain can change the country's population from 14 percent obese to 35 percent obese” (130). After helping to create the conditions for this weight gain, the diet industry has used these figures to incite mass panic, lobby the government even harder, sponsor “research” in their favor, bribe

doctors to promote their products, and pass the blame for both the average weight gain and the failure of diets onto individuals, all while turning a massive profit.

In Part Three, “A Better Way,” Aamodt answers the question of what should be done with all this information, especially given how counter it runs to popular discourse. In Chapters 11 and 12, she extolls the virtues of mindfulness and intuitive eating, especially in children and teenagers. Chapter 13 lays out the data on the relationship between weight and health, noting that body mass index categories do not line up with mortality risks, weight loss has never been shown to improve health in the long run (because there is no sample of people from whom to measure this data), dieting leads to weight cycling, which is harmful to health, and that physical activity is a better predictor of health than weight. Exercise helps keep visceral fat (stored inside the abdomen) levels low, limiting the circulation of saturated fatty acids which cause inflammation. Visceral fat is much more metabolically active than fat found below the skin, so it is preferentially lost during exercise, leading to health benefits regardless of weight. Although Aamodt does not mention *Health at Every Size* until page 204, these chapters are extremely similar to Linda Bacon’s 2008 book. Her major contribution here is a physiological explanation for why *Health at Every Size* works, not her advice, which is largely unoriginal.

Why Diets Make Us Fat is crucial reading for every health care provider. It is also valuable as a quick resource in arguments about the efficacy of diets. It makes one point especially clear: prescribing weight loss, especially as a health solution, is entirely and completely unethical. Aamodt does a great job of showing that diets—the explicit reduction of caloric intake below what is needed to maintain a stable weight within the defended range—do not, and indeed, cannot, work. Instead, they inevitably lead to worse health, both physically and mentally, and a higher defended range.

That said, the entire premise of the book is that diets are bad because they make us fatter. Although this is probably not Aamodt’s intended message, the implication of this premise is that if diets did make us thinner, they would be okay, or even good. By quoting “obesity” researchers like Yoni Freedhoff, who says a “time machine... would be the world’s best weight-loss program” (128) so we could go back to being cavemen and Rudy Leibel, who instead recommends fast forwarding “to when we figure out how to do this with a pill” (135), Aamodt makes it clear that just because diets do not work does not mean that we should accept fatness. Instead, it means we should stop advising them (because they make people fatter) until we figure out something that does work. This message doesn’t undermine the importance of the information she has compiled, but it does undermine any claims that the book is fat positive. After reading, a doctor or a researcher may stop recommending weight loss, but they probably will not stop supporting research into a “cure” for “obesity.”

Reference

Bacon, Linda. 2008. *Health at Every Size: The surprising truth about your weight*. Dallas, TX: BenBella Books.

Rachel Fox
*Doctoral student and Kroner Family Fellow in Communication and Science Studies at the
 University of California, San Diego, California*
 rafox@ucsd.edu

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