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Weight gain as a consequence of living a modern lifestyle: a discussion of barriers to effective weight control and how to overcome them

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Abstract

Objective: The purpose of this commentary is to discuss modern lifestyle factors that promote weight gain and to suggest methods for clinicians to more effectively educate patients about weight management.

Discussion: Most adults in the United States are overweight or obese. Multiple factors related to the modern lifestyle appear to play causal roles. In general, the population maintains sedentary lives and overconsumes calorie-dense foods. In particular, refined carbohydrates negatively impact metabolism and stimulate neural addiction mechanisms, which facilitate weight gain. As adipose tissue mass accumulates, satiation centers in the hypothalamus become resistant to insulin and leptin, which leads to increased caloric consumption. Several behavior issues further augment weight gain, such as eating too quickly, a lack of sleep, high stress levels, and a lack of exercise. Finally, adipose tissue accumulation alters the body weight set point, which leads to metabolic changes that function to resist weight loss efforts. Each of these factors may work together to augment weight gain and promote obesity. Health care providers, such as chiropractic physicians, who educate patients on wellness, prevention, and lifestyle changes are well positioned to address these issues.

Conclusion: People need to be educated about the modern lifestyle factors that prevent effective weight management. Without this knowledge and the associated practical application of lifestyle choices that prevent weight gain, becoming overweight or obese appears to be an unavoidable consequence of living a modern lifestyle.

Introduction

Efforts by the United States Surgeon General and World Health Organization to wage a war against

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obesity are being lost.[1-3] Nguyen et al[4] encapsulate the body weight problem:

> "In the USA, more than two thirds of adults and one third of children/adolescents are considered overweight or obese, a prevalence that has more than doubled since the 1970s. The projected prevalence of overweight individuals is 74.7% by 2015. Greater than one third of US adults is currently obese and 5.9% (~15 million) are morbidly obese with a body mass index (BMI) of 40."

Effectively combating obesity is a known public health concern due to its relationship to chronic disease expression. In addition, from the perspective of chiropractic practice, it is an error to view weight management as an issue that is separate from treating musculoskeletal pain. Obesity management is relevant because it is a known risk factor for low back pain chronicity.[5-7] A study of 3471 twins confirmed the association between excess weight and a variety of painful conditions, including low back pain, tension-type or migraine headache, fibromyalgia, abdominal pain, and chronic widespread pain.[8]

Although more than two-thirds of US adults are overweight, almost one-third of the population does maintain proper body weight; so it is clearly an achievable goal.

The common theme that is accurate and often repeated to the overweight/obese is to stay active and watch one's caloric intake, which allow for proper weight maintenance[9]; however, maintaining this behavior is difficult for a host of reasons. Perhaps, as some scientists suggest, "there is a lack of well designed, theory and evidence-based interventions that focus on weight-management among adults being overweight."[10]

Or perhaps, researchers have already identified factors related to weight gain and obesity that need to be properly understood and used in clinical practice.

Recent research has identified multiple factors associated with the modern lifestyle that promote weight gain and obesity. Maintaining sedentary lifestyles and the overconsumption of calorie-dense refined foods are the primary problems. Although the typical charge against refined foods is that their high caloric content is what leads to weight gain, additional problems have been identified and should be understood. Refined carbohydrates reduce energy expenditure, promote postprandial inflammation and insulin resistance, and stimulate neural addiction pathways. During the weight gain process, the hypothalamus becomes resistant to insulin and leptin, which leads to increased caloric consumption and further adipose accumulation. In addition, the modern lifestyle is associated with several behavioral factors that facilitate weight gain, such as eating too quickly, a lack of sleep, high stress levels, and a lack of physical activity. As adipose mass is accumulated, the body weight set point is altered, which leads to metabolic changes that function to resist weight loss efforts when attempted.

Health care providers, such as chiropractic physicians, who educate the public and patients on wellness, prevention, and lifestyle changes are well positioned to address these issues.[11-15] The purpose of this commentary is to discuss modern lifestyle factors that promote weight gain and to suggest methods for clinicians to more effectively educate patients about weight management.

Brief historical perspective on physical activity and calorie-dense food

In modern society, we work to acquire money so we can pay for food and other luxuries. However, before the modern era, we had to physically labor for food by hunting, foraging, and then farming. Substantial calories were expended during the laboring process for food as well as during the making and maintaining of shelters.[9] During this time, calorie-dense foods such as refined carbohydrates, fast foods, and processed foods were unavailable. Humans subsisted on natural foods, such as wild game, fish, and vegetation.[9] The natural selection process that took place over thousands of years caused humans to be genetically adapted to be a forager or, put more practically for patients, an *exerciser for natural food*. Indeed, "hunter-gatherers were lean, and obesity was virtually nonexistent."[9] Consequently, our modern sedentary lifestyle represents a foreign and unhealthy experience from the perspective of our genetic physiologic needs.[4] That weight gain and obesity would be unavoidable if humans were to transition from an active to a sedentary lifestyle and overconsume previously unavailable refined carbohydrates/lipids would be a reasonable hypothesis, and this is what has happened to modern man (Fig 1).[9,10,16]

The metabolic impact of refined carbohydrates

The current American diet is approximately 20% refined sugar, 20% refined grains, 20% refined oils, 15% to 20% fatty meat, and 10% dairy by calories.[16] Notice that the average American eats virtually no

- Reduced physical activity
- Overconsumption of calorie-dense food
- Overconsumption of refined carbohydrates
- Eating too quickly
- Inadequate sleep
- Psychological stress
- Obesity-induced hypothalamic inflammation
- Obesity-induced changes in bodyweight set-point

Fig 1. Lifestyle issues that promote weight gain and obesity.

vegetables and fruit and the vast majority of calories (40%) come from refined sugar and flour, which are refined carbohydrates with a high glycemic index/load.[16,17] Although the metabolic conversion of excess glucose from refined carbohydrate consumption into fat is a well-known process, there are at least 3 additional ways in which refined carbohydrates promote weight gain and obesity. In a recent study, total daily energy expenditure in participants on 3 different diets that were isocaloric was compared.[18] The very low carbohydrate diet burned 300 cal (1225.2 J) more per day compared with the high–glycemic index/load diet, which suggests that we should view calorie sources differently from a weight management perspective. Although the authors did not identify the physiologic reason for this metabolic effect of refined carbohydrates, this research indicates that refined carbohydrates should be operationally characterized to patients as foods that should be avoided because they promote fat accumulation.

Refined carbohydrates can promote also weight gain by promoting systemic endotoxemia and by shifting the species type within the gut flora. Before the refinement of grains, our gut flora was exposed only to lean meat, vegetables, fruit, tubers, nuts, and thereafter whole grains and legumes. When the gut is exposed to refined carbohydrates and excess lipid, 2 measurable changes take place. Initially, there is no weight gain; however, there is a postprandial inflammatory response caused by the heightened absorption of gut bacterial endotoxin.[19,20] The human gut contains gram-negative bacteria, of which lipopolysaccharide is the primary structural component of the outer cell membrane, which is also referred to as *endotoxin*.[21] The inflammatory response to endotoxin is associated with insulin resistance, which is related to the expression of obesity and diabetes.[20,22-26] In addition to postprandial endotoxemia, over time, refined carbohydrate consumption leads to a shift in the species population of gut flora that may to lead to a greater energy harvest by bacteria and greater absorbed calories.[27,28]

Despite the obvious intellectual reasons for avoiding refined carbohydrates, the clinical/lifestyle challenge to overcome regarding refined carbohydrate consumption is that humans *want* to eat these foods and seem unable to stop based on the growing obesity epidemic. There are measureable neural correlates to this behavior, such that the intellectual capacity to avoid an unhealthy food is overridden by desire. It is now known that the limbic reward/pleasure system is activated by refined carbohydrate in a fashion that is similar to other addictions.[29-32] In other words, we consume highly palatable comfort foods for their hedonic properties, which is the same reason for abusing substances such as alcohol and recreational drugs.[33]

Unless hedonic food-seeking behavior is adequately resisted, weight gain or obesity is inevitable, which leads to identifiable changes in neural circuitry compared with lean individuals. For example, obese individuals "demonstrate increased activation of brain reward circuitries in response to palatable food or food-associated cues."[32] Studies also suggest that there are individual differences in "trait reward sensitivity" such that individuals display variations in limbic activity to images or the presence of food, which make certain individuals more prone to weight gain.[32,34] These individuals have to work more diligently to prevent weight gain.

In summary, refined carbohydrates pose 2 challenges. First, the regular consumption of refined carbohydrates increases the risk for weight gain; and second, all humans to varying degrees have an "addiction" tendency regarding palatable foods. Patients should be educated regarding the nature of strong urges to eat hedonic foods; that is, the urges are normal to have, everyone has them, and they should be dealt with strong mental discipline, as when an alcoholic is avoiding alcohol.

Fat consumption is a more complicated issue,[35] as fat is normally found in healthy whole foods, such as nuts, olives, avocados, lean meat, and cold-water fish, none of which are hedonic foods. Interestingly, the consumption of nuts, a notoriously high-fat food, can be recommended as part of a weight loss/management program.[36,37] Fat consumption should be considered problematic in the context of hedonic foods that consist largely of refined carbohydrates and lipids, such as desserts.[35] In other words, refined carbohydrates are associated with food addiction; and the added lipid increases the caloric burden.[35] However, it is possible that addiction mechanisms may also be involved in

excessive fat consumption, as neural patterns of addiction for lipids have been identified in rats.[38,39]

How the obesity "state" prevents weight loss

In addition to an established addictive neural circuitry that is augmented in obese individuals and leads to the overconsumption of hedonic foods, another body chemistry change occurs that prevents the obese from losing weight. Obese individuals are systemically inflamed, such that circulating levels of inflammatory mediators, such as of high-sensitivity C-reactive protein (hsCRP), tumor necrosis factor, and interleukin-6, are significantly correlated with weight, BMI, waist circumference, hip circumference, and waist-hip ratio.[40] The inflammatory chemistry of obesity is correlated to insulin resistance and the metabolic syndrome,[41-43] which increases the odds of feeling fatigued,[44,45] unwell,[45] depressed,[46-49] and having local or widespread pain.[50-53] When feeling unwell and depressed, our natural tendency is to seek reward; and hedonic food is one way to do this, which only perpetuates the weight gain problem.[54]

In rodent models, systemic inflammation also impacts satiety centers in the brain, such that the hypothalamus becomes insensitive to the satiating effects of insulin and leptin.[55,56] Diet-induced hypothalamic inflammation actually functions to "increase the defended level of body weight."[56] Imaging of obese humans revealed increased gliosis in the hypothalamus that mirrored the changes that occur in animals.[57]

Obesity should thus be viewed as a "state" of food addiction and reduced satiation, which is likely to be coupled with feelings of fatigue, poor health, depression, and pain. Amidst this state of suffering, it is not likely that the average obese person will have the ability to develop the motivation necessary to exercise daily and minimize hedonic food consumption for the *rest of his/her life*. It is no surprise why battling obesity is so difficult and why weight gain after weight loss is so common, especially when obese patients are told to "eat less" and presumably suffer as a consequence.

Refining the "eat less" recommendation to overweight individuals

Suggesting that overweight individuals should eat less is an unclear message and an obviously difficult recommendation to follow, as they are hyperphagic and accustomed to eating large quantities of food. So the accurate message should be to avoid refined carbohydrate/fat foods and eat large quantities of whole food to achieve and maintain proper weight. A study with domestic pigs demonstrated that substantial amounts of food can be consumed without inappropriate weight gain.[58]

Twenty-four weaning piglets were randomly allocated to a grain-based swine feed or a grain-free Paleolithic diet consisting of vegetables, fruit, meat, and a small amount of tubers. After 15 months on the respective diets, several measurements were made, such as body weight, glucose tolerance, postchallenge insulin response, hsCRP, and blood pressure. All markers were better in pigs on the Paleolithic diet. Remarkably, CRP was 4.0 μg/mL in pigs on the Paleolithic diet vs 21.7 μg/mL in the grain-fed pigs that consumed almost 3.5 lb of feed per day for total of 4976 cal (20820 J). In contrast, the Paleolithic pigs ate 14.5 lb of food per day for a total of 4078 cal (17063 J).[58]

Overweight and obese patients need to be encouraged to eat in the fashion of the Paleolithic pigs; that is, patients should be urged to feel full by eating large amounts of low-calorie vegetation and adequate amounts of lean protein and natural fats, and make this their eating lifestyle. For perspective, 6 cups of cooked broccoli, 2 heads of romaine lettuce, 2 cups of blueberries, 1 apple, and 1 orange represent a large volume of food to achieve the feeling of fullness and at a mere 800 cal (3347.2 J). In comparison, going on a starvation diet is psychologically and physically traumatic. This approach to weight loss is unnecessary and impractical, especially when considered in the context of additional weight management issues that must be overcome, which include eating too quickly, inadequate sleep, stress-induce eating, and body weight set point changes.

Eating too quickly and weight gain

Several studies have demonstrated that eating slowly can reduce calorie consumption and management body weight.[59-61] One study demonstrated that eating the same meal over 30 minutes instead of 5 minutes leads to higher concentrations of gut peptides and favors earlier satiety.[59] The authors concluded that "the warning we were given as children that wolfing down your food will make you fat may in fact have a physiological explanation."[59] Overweight individuals need to be made aware of this information and be encouraged to eat more slowly and to respect satiation signals.

Inadequate sleep and weight gain

The Centers for Disease Control and Prevention explain that inadequate sleep is a public health epidemic.[62] The seriousness of this issue should not be underestimated from an over health perspective, such that inadequate sleep promotes systemic inflammation.[63,64] In one study, participants were sleep-restricted to 4 hours per night for 10 days. By day 10, hsCRP levels had risen 4-fold.[65]

Specific to the issue of weight gain, a lack of sleep increases insulin resistance, downregulates the satiety hormone leptin, upregulates the appetite-stimulating hormone ghrelin, and increases hunger and food intake.[66-69] Thus, identifying and properly managing sleep disorders should be incorporated into an effective weight management program.

In one study, the participants included 12 young men (22 ± 2 years) with an average BMI within the reference range (23.6 ± 2.0 kg/m^2) who normally slept between 7 and 9 hours per night.[70] After 2 nights, during which sleep was curtailed to 4 hours, there was a significant shift in hunger hormones and appetite (Table 1).

Efforts to lose weight are compromised in the wake of inadequate sleep. One study identified that lack of sufficient sleep (< 6 hours) may compromise the efficacy of typical dietary interventions for weight loss and related metabolic risk reduction.[71]

Reduced sleep is also implicated in the genesis of depression.[72,73] This is problematic because depression is a risk factor for obesity and, conversely, obesity is a risk factor for depression.[54] An additional risk factor for depression is the consumption of hedonic foods, which can occur independent of BMI.[74]

Stress and weight gain

The American Psychological Association reports that most Americans suffer from moderate stress.[75] This likely adds to the obesity problem, as the notions of "stress eating" and "self-medication with comfort foods" are actually well-studied phenomena.[76-79]

Table 1 Sleep loss, hunger, and appetite[70]

Marker	% change
Leptin	−18%
Ghrelin	+ 28%
Hunger	+24%
Desire for calorie-dense foods with high refined carbohydrate content	+33%-45%

The stress response is typically taught as the fight or flight response. In other words, we would historically engage in intense physical activity when stressed, which is now known to reduce inflammation and the stress response[80,81] and to reduce anxiety and depression.[82-85] Exercise also has an appetite-suppressing effect.[86,87] We tend to feel better after exercise; and interestingly, there is a known hedonic effect of exercise,[88] which, theoretically, may help to reduce the consumption of hedonic foods.

In Paleolithic days, we did not have the option to eat hedonic comfort foods to reduce stress; we instead naturally engaged in physical activity. However, in modern times, we can instead choose to eat hedonic foods rather than exercise. Patients need to be made aware of these relationships and should be encouraged to exercise or engage in vigorous activities.

Weight loss and the body weight set point

Historically, weight gain conferred survival advantages, such that additional body fat could function as an energy source during periods of food scarcity.[89] Consequently, researchers propose that, through natural selection, humans developed a "thrifty phenotype," such that humans have the capacity to amass energy stores more efficiently during periods of abundant food supply.[89] In other words, humans are disposed to gain weight for survival purposes, which was advantageous before the advent of modern society with its abundant availability of energy-dense processed foods. Similarly, weight loss research indicates that body weight set points are modified to physiologically defend accumulated body weight and facilitate additional weight gain.[90-96]

In a recent study, 34 of 50 obese participants completed a 62-week study to identify long-term hormonal responses to weight loss.[90] Hormonal assessments were performed at baseline, week 10, and week 62, which included hunger-suppressing hormones (leptin, peptide YY, cholecystokinin, insulin, amylin) and ghrelin, which stimulates hunger. During the first 8 weeks, the Optifast VLCD (very low calorie diet; Nestlé, Notting Hill, Australia) was used to deliver approximately 500 cal/d (2092 J). Ordinary low-glycemic index foods were introduced during weeks 9 to 10, and long-term diet and exercise recommendations were provided for maintaining the lost weight. Dietary counseling and support involved bimonthly visits and telephone communications.

Participants lost an average of 30 lb in 10 weeks, which represented at least 10% of their original body weight. At weeks 10 and 62, the hunger-suppressing hormones were reduced from baseline; and there was an increase in hunger-promoting ghrelin, which supports "the view that there is an elevated body weight set-point in obese persons and that efforts to reduce weight below this point are vigorously resisted."[90] Self-reported hunger and desire to eat were elevated during the entire study period. At the end of the study, the average weight regain was 17 lb. Such rebound weight gain is physiologically orchestrated and continues to be investigated in animal models. A recent study led to the following conclusion[96]:

> "We propose that long-term diet-induced obesity creates a higher body weight set-point and that weight loss induced by caloric restriction, as seen in the high-fat caloric restriction group, provokes the brain to protect the new higher set-point. This adaptation to weight loss likely contributes to rebound weight gain by increasing peripheral ghrelin concentrations and restoring the function of ghrelin-responsive neuronal populations in the hypothalamic arcuate nucleus."

Although future research may identify and clarify body weight set point mechanisms, available evidence should lead physicians to explain to patients that they will likely spend their entire life battling the body weight set point system. Patients should understand that, independent of what a physician and patient set as a weight loss goal, the body generates measurable physiological signaling that defends against weight loss. Patients should also be made aware that regular exercise appears to lower the body weight set point and should be considered an essential component of effective weight management.[95] A recent study indicates that "exercise directly influences the responsiveness of the CNS circuits involved in energy homeostasis by allowing the defense of a lowered body weight."[95]

As described in this commentary, the available evidence suggests that multiple factors associated with living a modern lifestyle participate in promoting weight gain and obesity. Admittedly, other weight-promoting factors may participate beyond those discussed in this commentary. Furthermore, it should be understood that it is the author's personal experience and impression that addressing these weight gain factors can be useful in helping patients with weight management issues. To date, intervention trials supporting this impression are lacking.

Limitations

This commentary provides one viewpoint into barriers to weight control; thus, it does not include an exhaustive list. Increasing patient awareness of the weight control barriers they must overcome to improve weight management outcomes is theoretical at present. Although this theory is reasonable, the barriers described in this article have not been systematically investigated in a weight management study, such that the efficacy of this approach is unknown.

Conclusion

Unless individuals are educated to actively engage in lifestyle choices that prevent weight gain, becoming overweight or obese appears to be an unavoidable consequence of modern living. Before the modern era, physical activity was a requirement; and now, it is an option. Historically, weight-promoting hedonic foods were not available and thus foreign to our nervous system; and now, they are readily available and activate addiction circuits in the human brain.

Regular consumption of hedonic foods also leads to metabolic changes, such as chronic inflammation, insulin resistance, and an inhibition of hypothalamic satiety centers, which facilitate their further consumption. Based on these changes, a recent article suggests that our perspective on obesity should be reframed and properly viewed as a chronic inflammatory disease,[97] which is perpetuated by lifestyle issues such as inadequate sleep, stress, and a lack of exercise.

The body also appears to view weight gain as an appropriate survival measure, as far as combating food scarcity is concerned. However, in an age where there is an abundance of refined calorie-dense foods, the outcome is an elevation of the body weight set point, which is vigorously pursued and defended, as made evident by long-term hormonal changes that encourage weight gain.

Patients need to understand that achieving and maintaining appropriate weight should be viewed as a daily focus. The presence of the many modern lifestyle issues that directly promote weight gain represents a

constant challenge to overcome to realize effective weight management.

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Concept development (provided idea for the research): DRS
Analysis/interpretation (responsible for statistical analysis, evaluation, and presentation of the results): DRS
Literature search (performed the literature search): DRS
Writing (responsible for writing a substantive part of the manuscript): DRS
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