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Is there a novel solution to weight-loss failure in America?

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Weight loss is a fairly common concern in contemporary American society. As rates of heart-disease, obesity, diabetes, and similar diseases tied to the conditions of obesity rise, the push by the health-community for citizens to attend to their health with weight-management strategies continues to rise. Despite this, Americans still remain at a high-position within global ranking for nations with the greatest obesity rates. A common complaint that is heard in America is self-reported weight-loss failure, with cries similar to that of "I can't keep the weight off," and "my diet just doesn't work" being common anecdotal complaints by patients to medical practitioners.

A study by Hales, et al. (2018) shows that between 2007 and 2008, the obesity rate in American adults was 33.7%, but by 2015 to 2016, this had climbed to 39.6%, an almost 6% increase across all American adults, making almost four in ten Americans obese. (Hales et al., 2018) At the same time, the proliferation of information freely available through published data on weight loss has only grown since the 2000's, and research into effective weight-loss has only grown in recent years, which begs the question; Why are Americans continuing to report weight-loss failure and grow more obese? Is there an unnoticed, novel reason that may contribute to weight-loss failure? Because obesity has a significant number of well-documented side-effects that severely degrade quality-of-life for its' victims, and because of the steadily rising rate of obesity, this paper seeks to investigate a possible set of extraneous causes for weight-loss failure. Because of the commonality of complaints of difficulty with weight-loss, as opposed to total indifference to suffering obesity, weight-loss failure will be the only-considered aspect of obesity in America.

Investigating the possible extraneous causes of weight-loss failure is a complex issue that

is impacted by numerous disciplines. The primarily accepted method and guidance given to physicians to advise patients on weight-loss is inducing a caloric deficit through diet (Hall & Kahan, 2018). However, this has been disputed in recent years, and a short period of investigation shows a vast wealth of research into theoretical causes for obesity that are disconnected from caloric-deficit via diet as treatment. Some medical practitioners have proposed that this is only one part of an effective co-treatment program (Trujillo-Garrido & Santi-Cano, 2022), and without additional treatments, these methods will not effectively bring about significant or long-lasting weight loss. As a result, it is quite possible that obesity is, in some cases, a condition resulting from multiple comorbidities, requiring multiple or novel treatment methods. Because of this, it is beyond the scope of any one single discipline to find a singular, definitive cause of obesity, but numerous disciplines have provided their own unique insights regarding the causes of obesity, and therefore weight-loss failure.

Because obesity has been a long-standing issue in contemporary America that has persisted extensively over the years, finding a single disciplinary solution has not been able to resolve this issue. Because it has a negative impact on patient quality-of-life that can lead to serious complications, it is important for the purpose of ensuring the public health of American society to determine a more holistic approach to this issue. This paper seeks to help find a probable set of primary, solvable novel causes for obesity.

A cursory investigation of this problem shows numerous relevant disciplines that may contribute their own individual unique, or interconnected insights. These include neurochemistry, gastroenterology, sports medicine, nutritionology, sociology, public health, psychology, endocrinology, and biochemistry. This paper will limit focus to the most relevant disciplines, those being endocrinology, psychology, and public health. Although other disciplines offer

significant insight to the issue, these disciplines show the greatest amount of tangible research and results regarding novel causes for obesity.

Literature Review

Endocrinology provides a unique insight into obesity as caused by hormonal aberrations. Although it provides a diverse, highly complex insight into numerous possible causes and contributing endocrine factors, a set of hormonal influences have been long identified as being serious contributors to obesity in cases of major imbalances in otherwise healthy individuals. Sex-horomones, when imbalanced, show significant correlation with massive weight gain. (Zumoff, 1987) Obese women show signs of having significantly elevated free testosterone, the primary male sex-hormone, and free estradiol, the primary female sex-hormone, levels in their blood when tested. When weight is lost, these hormones rapidly stabilize to normal levels. Conversely, men suffering from obesity, when tested, show signs of having significantly elevated levels of estrone, another female sex-hormone, as well as massively elevated levels of free and total estradiol, but are massively deficient in free and total testosterone. In all cases, these key sex-horomones experience imbalances proportional to the degree of a patient's obesity. After weight-loss is induced, these hormonal imbalances stabilize to normal levels. (Zumoff, 1987)

Additionally, further studies reveal that a significant number of regulatory hormones tied to appetite, especially those that cross the blood-brain barrier, deeply correlate with obesity, or contribute to obesity-inducing symptoms. Leptin, a hormone discovered in 1994 whose production is tied to obesity-inducing genes (Zhang et al., 1994), was found to be mostly produced by adipose, or fatty-connective, tissue, although produced in numerous other tissues, such as bone, muscle, digestive, and brain tissues. Particularly, leptin was found to have a massive influence on behaviors in laboratory mice. According to Susan M., et al, genetically

modified laboratory mice of the *Ob/ob* strain produce no functional leptin. Research shows that leptin, in healthy mice, regulates appetite and activity levels, as well as satiety after feeding. However, mice of the *Ob/ob* strait, without leptin, rapidly gained weight when their diet was unrestricted and they were allowed to consume as much food as they desired. Mice with the *Ob/ob* mutation would thus balloon in weight, reaching more than four times the level of obesity that healthy laboratory mice would when allowed to feed at their own will. Conversely, when the obese *Ob/ob* mice were administered sufficient levels of leptin, these mice would lose this weight. From the studies this data was drawn from, it was evident that leptin played an important role in regulating and controlling appetite.

According to Susan M. et al, concurrent studies showed that this phenomenon was also detectable in human subjects. Human children who tested as being significantly deficient in leptin displayed a massive appetite, and struggled seriously with obesity. However, by administering leptin exogenously to these children, the patients would experience a massive decrease in their appetite. Following this, these children would begin to rapidly lose body-fat, while still maintaining their lean body mass. However, a key caveat to this treatment is that in a number of patient cases, some patients test high for leptin levels in their endocrine systems, which researchers see as a sign of leptin resistance. In these cases, administering exogenous leptin does not induce the usual effects, and fails to reduce and treat the obesity in the specified patients. Although some theories have been postulated regarding reasons for leptin resistance, there is still much research to complete before conclusions can be drawn. It is expected that from the conclusions of the ongoing research into the mechanisms of leptin resistance, a new set of therapeutic options will be available. (Susan et al., 2004)

Another hormone of concern is ghrelin. Conversely to leptin, ghrelin has the opposite effect on patients, stimulating appetite and reducing the body's usage of carbohydrates and fat-stores. In healthy individuals, ghrelin is produced at an accelerated rate before each meal, and then falls to lower levels immediately after finishing consumption. In healthy subjects, ghrelin levels are detected as being high in patients undergoing fasting, and low in obese patients. Because of ghrelin's role as a "counterbalance" to leptin, endocrinologists have suggested the possibility of developing an "antagonist" medication that would inhibit ghrelin production or activity within the body, or a therapeutic mechanism to achieve a similar effect. (Susan et al., 2004)

Public health provides a highly complex set of perspectives to the issue of obesity and weight-loss failure that concurs with the endocrinology's model of hormonal imbalances as a cause of obesity. Historically, public health ascribed to the "calories in, calories out" model of obesity and weight-loss. This model is based on an "economic" view of the function of metabolism with its relation to nutrition, where a specified volume of macronutrients consumed provides the body with a set number of calories, and physical activity over time expends a set amount of calories. (Levine, 2017) This assumption, however, has been recently challenged by new models and proposals within the field of public health. Camacho S. et al. posits that the calorie model not only causes harm beyond immediate health-effects, such as creating social stigma, but that an alternative model may provide a significantly improved success rate in the treatment of obesity. Specifically, a framework of obesity and its causes based on the insulin-dependent regulation of fat generation, as well as the metabolic pathways of degradation of consumed macronutrients, is proposed by Camacho S. et al. as an alternative. (Camacho & Ruppel, 2017)

The field of psychology has offered its own critical insights regarding possible novel primary-causes of obesity, to the degree that a gastroenterology study by Kwong F. et al. posits that cognitive behavioral therapy is an important aspect of a weight-loss regimen (Fock & Khoo, 2013). Recent psychological perspectives have shown that obesity and attempts to treat it are significantly impacted by the expectations and mental health of a patient. According to Kevin H. et al, both patients and physicians often showed a degree of over-optimism regarding potential immediate weight-loss, with patients treated for obesity expecting to lose "20-40% of their starting body weight - amounts that can only realistically be achieved by bariatric surgery," such as a gastrectomy or gastric bypass. Physicians showed a similar set of unrealistic expectations, considering "a loss of 21% of initial body weight" to be an acceptable expectation. However, within the scope of Kevin H. et al, patients showed only a 5-10% average in weight loss, with very few successfully reaching and maintaining their reported "ideal weight." (Hall & Kahan, 2018)

Studies by cognitive psychologists show that "patients' expectations and the degree to which they are met are likely to affect self-efficacy and relapse," causing patients to regain what weight they may have lost. (Foster et al., 1997) Failing to lose the reportedly expected 20-40% of initial weight may disappoint patients to the degree that they may conclude that continuing treatment is not worth the effort, and may discontinue attempting to maintain their weight-loss. (Hall & Kahan, 2018) Conversely, these negative outcomes become far less common in patients who find early success in treatment. Psychologists have found that weight maintenance over long periods of time was more commonly observed in patients that achieved a greater amount of initial weight loss, and who reached a self-determined goal weight in that time. (Elfhag & Rössner, 2005) Additionally, patients who suffered from having poor coping mechanisms, high

stress, or had a history of eating disorders showed reduced success rates in maintaining weight loss. (Elfhag & Rössner, 2005)

Common Ground

The provided evidence shows that obesity may be a symptom of an inherently complex system of morbidities that varies between patients. Endocrinology and public health both concur in a similar sense that the hormonal composition that a patient carries can affect their ability to treat their obesity. The ability of a patient to remain satiated and for their metabolism to process and degrade consumed food is deeply impacted by regulatory hormones such as ghrelin, and can be severely affected by imbalances in sex-hormones in afflicted patients. Although other disciplines have long held the calorie model as the definitive method of weight loss, endocrinology's view of how hormones impact the individual, when contextualized in the public-health model, more effectively communicates the potential issues that an individual patient may face in the pursuit of weight-loss.

By individually modeling the metabolic impact of the hormonal composition of each individual patient, a more comprehensive understanding of the optimal treatment for the patient's case can be obtained. Additionally, this may create opportunity for physicians to develop an individualized, more realistic prognosis for the patient regarding their expectations for weight loss. With this more realistic expectation, the physician may be able to advise their patient more effectively on what a reasonable "goal weight" during the initial period of weight loss may be. The psychological perspective's findings that a patient being able to meet a goal weight during this period significantly improves their chances to maintain this goal weight shows that this advice by a physician may significantly improve rates of weight-loss success.

An additional, novel benefit of considering the hormonal makeup of the patient is how it may impact the psychological perspective of a proposed treatment plan. The impact of a sex-hormonal imbalance on a patient's emotional state may deeply impact their stress levels, as well as require significant coping mechanisms, which a psychological aspect of a proposed obesity treatment may significantly benefit from taking into consideration. Additionally, through regular psychological counseling, patients may potentially be able to be screened for hormonal imbalances in leptin or ghrelin with greater regularity than with regular blood tests by questioning their level of appetite.

In conclusion, this paper proposes a novel solution to weight-loss failure, and by extension obesity. Through usage of a metabolic-pathway framework for treatment as opposed to the calorie model, and compiling initial blood-screening and hormonal testing, physicians will be able to effectively advise their patients on an effective, individualized treatment plan, as well as provide them with more realistic, predictable prognosis, thereby reducing the psychological effect that "failing" to achieve significant initial weight-loss may bring. Undertaking this process offers the opportunity for physicians to find treatable hormonal imbalances, specifically in leptin, that may be solvable by exogenously administering supplementary leptin. Additionally, by screening for ghrelin imbalances, as well as sex-hormone imbalances, physicians may be able to compile a suitable outline of a patient's emotional or mental state that may be able to assist a psychologist partaking in this treatment program in more deeply understanding their patient's needs, raising success rates from the perspective of the application of cognitive behavioral therapy.

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