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OBESITY UPDATES: UNDERSTANDING OBESITY AS A DISEASE AND INTERMITTENT FASTING

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ABSTRACT

Obesity is now recognised as a chronic disease that needs chronic treatment to treat or prevent obesity-related complications. This article discusses the biology of weight regulation as a basis to understanding obesity as a disease, and to appreciate the complex and multifactorial nature of the obesity problem. Finally, the article highlights the dietary approaches as part of the multi-pronged approach to treating obesity and gives a brief update on intermittent fasting.

Keywords: Obesity. Chronic Disease. Body Weight Regulation. Intermittent Fasting.

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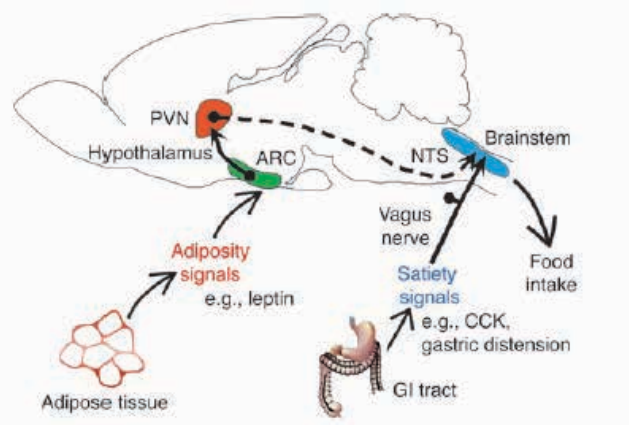
INTRODUCTION

Over the last 40 years, the prevalence of obesity has risen substantially in almost all regions of the world, such that there are now more than 600 million people with obesity worldwide.[1,2] This increasing burden of obesity affects all regions, including Singapore.[1] The National Health Survey (NHS 1992-2010) reports that 10.8 percent of adult Singaporeans were obese in 2010, more than double the prevalence in 1992.[3] Results from recent national health surveys, the National Population Health Survey (NPHS 2017-2020), suggest a continuing of this trend after a brief period of stabilisation from 2010 to 2017.[4]

BIOLOGY OF WEIGHT REGULATION

The body's adipose tissue represents energy stores to survive energy-scarce conditions. Hence, it would not be surprising that that body weight (or more accurately, adipose tissue in the body) is tightly regulated by an extremely complex neuroendocrine energy balance circuitry, which is composed of specific nuclei in various brain regions, most prominently the hypothalamic arcuate nucleus (ARC), the paraventricular nucleus, the lateral hypothalamic area, and the nucleus of solitary tract of the hindbrain[5,6] (**Figure 1**). Under relatively constant environmental conditions, this regulatory system senses and processes various metabolic signals regarding the current energetic status and adjusts the metabolic responses to maintain a stable weight without conscious control.[5] This homeostatic regulation of body weight is similar to the regulation of other physiologic parameters, such as body temperature, blood pressure, or blood glucose, where a "set point" seems to exist and deviation from this "set point" elicits a compensatory response in an opposite direction to restore this body weight "set point". Therefore, weight regain after weight loss is physiological[7,8] and not necessarily due to a failure of conscious efforts (to lose weight).

Figure 1: Model for regulation of the hindbrain response to satiety signals by hormonal input from the ARC[6]



Additionally, there exists a different set of neuroendocrine signals that guides food intake based upon the reward value of the food, also known as the reward or "hedonic" system.[5,9] The brain regions responsible for this reward system are dispersed in the corticolimbic structures, and a primary characteristic of this system is its ability to override the signals from the homeostatic circuits as described.[5] Hence, the reward system is non-homeostatic with regard to energy balance. This system integrates basic midbrain and hindbrain functions with more complex cortical functions involving arousal at the sight of palatable food items and the procurement of food, mediating the "liking" (level of pleasure or reward) and "wanting" (the motivation or drive to consume food), which are subconscious processes.[5] In human studies, functional MRI (fMRI) studies have shown overactivation of reward-encoding brain regions and/or deficiency in cortical inhibitory networks in people with obesity.[5]

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OBESITY AS A DISEASE: PATHOPHYSIOLOGY AND HEALTH CONSEQUENCES

With the understanding of the biology of weight regulation, obesity (defined as a disproportionate body weight for height with an excessive accumulation of adipose tissue[10]) is now understood to signify an abnormal physiological state whereby there has been a surplus intake of energy and an elevated body weight set point is now defended.[5,11] The factors known to cause this are complex and multiple, and they range from genetic to environmental to emotional factors, which are well-known to be potent modulators of appetite.[9] Twin, family, and adoption studies show that the rate of heritability of BMI is high, ranging from 40 to 70 percent,[12] demonstrating a major genetic component. In addition to syndromic and monogenic forms of obesity, genome-wide association studies (GWAS) have identified more than 700 independent loci associated with BMI and/or obesity.[13-15] Environmental and lifestyle factors favouring a positive energy balance and weight gain include increasing per capita food supplies and consumption, particularly of highly processed, energy-dense and palatable foods that are often served in large portions; decreased amounts of time spent in occupational physical activities and displacement of leisure-time physical activities with sedentary activities such as television-watching and use of electronic devices; the growing use of medicines that have weight gain as a side effect; stress; and inadequate sleep.[12] More recent studies have identified a potential role for the microbial content of the gut in determining a broad range of metabolic abnormalities, including obesity.[16,17] The evidence supporting causation includes animal studies that show that obesity, as a phenotype, is transmittable via the transfer of gut microbiota from the obese (mice/humans) to germ-free mice,[18,19] and mechanistic studies that demonstrate the possible mechanisms linking the gut microbiota with obesity.[16,20]

Obesity is not benign. The failure of adipose tissues to continually expand leads to pathological changes in the adipose tissue, which is characterised by macrophage invasion and/or increased release of pro-inflammatory adipokines and decreased release of anti-inflammatory adipokines such as adiponectin[10] (**Figure 2**). In addition, this failure to further expand and act as a "metabolic sink" results in harmful ectopic fat deposition in lean tissues such as the heart, liver, pancreas, and kidneys.[10] These two phenomena contribute to a pro-inflammatory and insulin-resistant milieu, giving rise to metabolic complications such as type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD), and cardiovascular disease (CVD).[10,21] Additionally, the physical forces as a result of excessive adipose tissue can give rise to biomechanical consequences (such as Obstructive Sleep Apnea (OSA) and low back pain), and obesity as a condition has been associated with various psychosocial issues, impacting on mental health.[22] All these adverse consequences affect quality of life, increase healthcare costs, and finally, increase mortality.[23] Therefore, based on the current knowledge that the development of obesity results from established pathophysiology, with attending health consequences (complications, morbidities and mortality), obesity fulfils the criteria for a disease state and is now determined to be a disease,[11] rather than just a lifestyle risk factor. Several associations and organisations, including the World Health Organisation (WHO), have now declared obesity as a disease (**Figure 3**), and this is an important first step in tackling the problem of obesity, which has emerged as an epidemic that poses an unprecedented public health challenge.[11]

Figure 2: Pathological changes in adipose tissue[10]

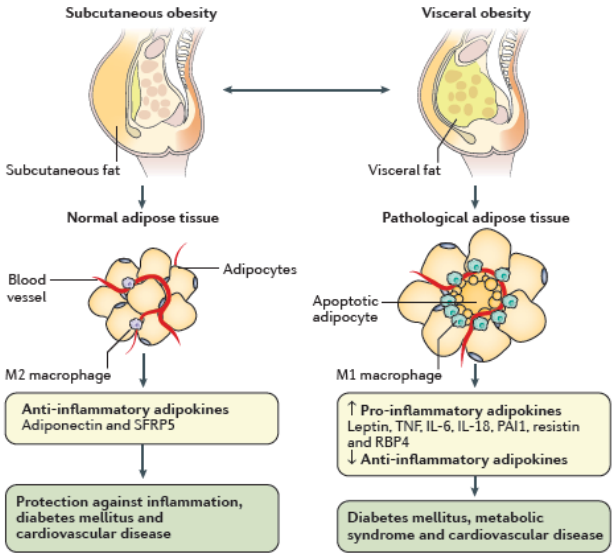

Figure 3: Associations or organisations that have declared obesity is a disease[11]

Box 1
Associations or organizations that have declared obesity is a disease

- National Institutes of Health
- US Food and Drug Administration
- Federal Trade Commission
- American Medical Association
- World Health Organization
- American College of Physicians
- American Association of Clinical Endocrinologists
- American College of Cardiology
- The Endocrine Society
- American Academy of Family Physicians
- Institute of Medicine
- The Obesity Society
- World Obesity Federation
- American Heart Association
- American Diabetes Association
- American Academy of Family Physicians
- American Society for Reproductive Medicine
- American Urologic Association
- American College of Surgeons

Data from Kahan S, Zvenyach T. Obesity as a disease: current policies and implications for the future. Curr Obes Rep 2016;5(2):291–7; and Bray GA, Kim KK, Wilding JPH. Obesity: a chronic relapsing progressive disease process. A position statement of the World Obesity Federation. Obes Rev 2017;18(7):715–23.

SEVERITY OF OBESITY AND TREATMENT APPROACH TO OBESITY AND ITS COMPLICATIONS

As with any disease state, the management of it requires an understanding of how severe the disease is. For obesity, management guidelines have slowly moved from a BMI-centric approach, where the goal of therapy is to lose a given amount of weight (e.g., 5-10 percent), to a complications-centric approach, where weight is no longer the major determinant of appropriate treatment but now based on the risk, presence, and severity of obesity-related complications.[24] For example, at least 10 percent weight loss is needed to significantly improve NAFLD and OSA.[24] Hence, for a person with multiple complications, which include NAFLD and OSA, modest weight loss (defined as 5-10 percent weight loss) may be inadequate, and more aggressive treatment options, effecting more than modest weight loss, need to be considered. Although more aggressive treatment may involve higher risks, the benefits of treating the various obesity-related complications should outweigh these risks. Therefore, the main goal of therapy now is to treat or prevent obesity-related complications, rather than lose weight per se. In line with this paradigm shift, the Working Group on Obesity, Diabetes and the High-risk Patient from the European Society for Hypertension, and the European Association for the Study of Obesity published a consensus document that discusses the mechanisms of obesity-induced hypertension, diabetes, and dyslipidemia, and highlights practice guidelines for the treatment of these conditions.[25] Essentially, this document calls for treatment of the underlying obesity in people with obesity-induced hypertension, diabetes, and dyslipidemia. Hence, for some of these people, the first medication may not be an anti-hypertensive medication (for example) but an anti-obesity medication. The other important point is that medications to treat these conditions should not worsen the underlying obesity, hence awareness of the potential weight effects when selecting pharmacologic agents for the treatment for these conditions is important. For example, sulphonylureas promote weight gain and should be avoided in patients with obesity as far as possible.

IMPORTANCE OF A MULTI-LEVEL AND INDIVIDUALISED MULTI-PRONGED APPROACH TO TREATING OBESITY

It is now known that the simple calculations underlying the traditional adage of "eat less, exercise more" are fatally flawed.[26] Aiming for a 500 kcal deficit (energy expenditure more than energy intake) per day, cumulating to 3,500 kcal per week (equivalent to ~0.5 kg of fat) will not result in a 0.5 kg/week weight loss indefinitely, because this calculation does not consider the homeostatic mechanisms that will resist further weight loss, and in fact, will conspire to regain weight to restore the original "set point".[7,8,26] Additionally, it is important to note that the same diet and exercise plan (often prescribed once in the beginning) will not suffice to maintain that 500 kcal deficit per day as a declining weight will mean declining energy expenditure.[26] Nonetheless, the point here is that asking all people with obesity to just "eat less and exercise more" overly simplifies the obesity problem. An understanding of the biology of weight regulation and the appreciation of the complex and multifactorial nature of how this regulation can go wrong resulting in obesity would indicate that there is no one-size-fits-all intervention or solution[27] and would necessitate a multi-level and individualised multi-pronged approach to treating obesity. Multi-level, apart from the individual, would include the social and community, physical (environment), and economic levels of interventions,[27] while a multi-pronged approach at the individual level would encompass not just the lifestyle and behavioural modifications but also the possible combination with pharmacologic and even bariatric surgical procedures based on individualised risk-benefit assessment.[10,24]

DIETARY APPROACHES TO OBESITY TREATMENT

Lifestyle management remains a cornerstone in a multi-pronged approach to the treatment of obesity, and dietary modification is foundational in this management. General dietary advices that have Randomised Controlled Trials (RCT) level of evidence include decreasing sugar-sweetened beverages and portion control (e.g., plate concept),[28] both of which can be routinely advocated. More specific dietary approaches can be broadly categorised into energy-focused (e.g., use of meal replacements, low/very low energy diets), macronutrient-focused (e.g., low carbohydrate diet, low fat diet), dietary pattern-focused (e.g., DASH diet, Mediterranean diet), and dietary timing-focused (e.g., intermittent fasting, time-restricted feeding).[28] These diets usually involve some form of controlled intake and will on average induce weight loss if followed strictly.[10,28] Hence, adherence to diet seems to be key as long-term diet trials have not shown a clear superiority of one diet over another with respect to average weight loss.[10,26]

However, while patient preference plays a part in adherence, the preceding discussion on the biology of weight regulation informs us that homeostatic mechanisms such as increased hunger and cravings will be triggered upon weight loss, posing a challenge to diet adherence in the long term. Therefore, the satiating effect of a diet may become particularly important for long-term dietary adherence and weight maintenance. For example, diets with high amounts of protein (protein has a greater satiating effect compared with carbohydrates and fats), especially when the overall diet has a low glycaemic index, has been shown to be more beneficial for maintaining weight loss,[26] and there is the suggestion that meals containing carbohydrates may have a weaker satiating effect on individuals with impaired glucose metabolism. Hence, obese individuals with impaired glucose metabolism may benefit more from a diet with higher fat and protein.[29,30]

FINALLY: A BRIEF UPDATE ON INTERMITTENT FASTING

Recent years have seen a surge in popularity of timing-focused dietary approaches whereby such eating patterns involve restricting energy intake by varying degrees for a pre-defined period and eating *ad libitum* (i.e., to satisfy appetite) at all other times.[31] These can range from complete (no energy containing foods or beverages consumed) alternate day fasting, to modified fasting regimens that allow the consumption of 20-25 percent of energy needs on scheduled fasting days (e.g., alternate days, two days per week [the "5:2 diet"]), to time-restricted feeding (which allows *ad libitum* energy intake within specific time frames inducing regular, extended fasting periods), and finally, to a variety of fasting regimens undertaken for religious or spiritual purposes.[32] Based on the current available evidence in humans, it appears that almost any intermittent fasting regimen can result in some weight loss and improvements in multiple health indicators including insulin resistance and reductions in risk factors for cardiovascular disease, through multiple pathways including reducing oxidative stress, optimisation of circadian rhythms, and ketogenesis, with no significant harm demonstrated.[32-34] However, human studies have largely been limited to observational studies of religious fasting, cross-sectional studies of eating patterns associated with health outcomes, and experimental studies with modest sample sizes.[32] Hence, large-scale randomised trials of longer duration (>1 year) are needed for more conclusive evidence on efficacy and potential harm so that sound recommendations can be made.[32] In any case, consistent with points made in the preceding sections, intermittent fasting could potentially be a treatment option but unlikely to be that "silver bullet", and the treatment of obesity would still require a multi-level and individualised multi-pronged approach.

CONCLUSION

Obesity is now recognised as a disease and has been described as a complex, chronic medical condition with a major negative impact on human health. Several associations and organisations, including the World Health Organisation (WHO), have now declared obesity as a disease, and this is an important first step to tackling the problem of obesity. An understanding of the biology of weight regulation and the appreciation of the complex and multifactorial nature of how this regulation can go wrong resulting in obesity would indicate that there is no one-size-fits-all intervention or solution and would necessitate a multi-level and individualised multi-pronged approach to treating obesity and its related conditions.

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LEARNING POINTS

- **Obesity is now recognised as a chronic disease that needs chronic treatment to treat or prevent obesity-related complications.**
- **The complex and multifactorial nature of obesity means that there is no one-size-fits-all intervention or solution and would necessitate a multi-level and individualised multi-pronged approach to treating obesity and its related conditions.**
- **Dietary approaches such as intermittent fasting could potentially be a treatment option but it is unlikely to be that "silver bullet".**