Dietary Assessment Tools and Metabolic Syndrome

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Metabolic syndrome (MS) is associated with a range of chronic diseases, for which lifestyle interventions are considered the cornerstone of treatment. Dietary interventions have primarily focused on weight reduction, usually via energy restricted diets. While this strategy can improve insulin sensitivity and other health markers, weight loss alone is not always effective in addressing all risk factors associated with MS.

Keywords: metabolic syndrome ; carbohydrate ; processed ; meal timing

1. Introduction

The clustering of metabolic disturbances linked to cardiovascular disease was first described by Swedish physician Eskil Kylin in 1923 [1]. In 1947, Jean Vague noted upper-body obesity as the phenotype most commonly associated with the metabolic abnormalities linked to type 2 diabetes and cardiovascular disease [2]. However, it was not until 1975 that the term ‘metabolic syndrome’ (MS) was first used in the medical literature by Hermann Haller [3], with Gerald Reaven later proposing in 1988 that insulin resistance was the common feature of this syndrome [4]. Whilst there are various definitions of MS, the most favoured was developed by the US National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) in 2001. This definition incorporates the key diagnostic criteria of hyperglycaemia/insulin resistance, visceral obesity, atherogenic dyslipidaemia and hypertension, and allows the use of readily available anthropomorphic, hemodynamic and blood assessments to diagnose MS based on the presence of three out of the five aforementioned criteria [5].

The US National Health and Nutrition Examination Survey (NHANES) (1999–2002) estimated the age-adjusted prevalence of MS in United States adults aged 20 years or greater to be between 34.6% and 39.1% [6]. Unpublished data from the AusDiab study (1999–2000) suggested that prevalence of MS in Australian adults was between 23.9% and 26% [7]. More recent NHANES data suggest that the overall prevalence of MS did not significantly increase in the period 2011–2016; however, statistically significant increases in prevalence amongst younger adults (aged 20–39), women and Asian and Hispanic individuals were still noted [8]. Globally, MS is estimated to be 3-fold more common than type 2 diabetes, with prevalence estimated at one-quarter of the world’s population [9]. Individuals with MS have a 5-fold higher risk of developing type 2 diabetes and a 3-fold higher risk of developing cardiovascular disease [10]. In the period 2015–2016, an estimated 8.9% ($10.4 billion) of total disease expenditure in the Australian health system was attributed to cardiovascular diseases and an estimated 2.3% ($2.7 billion) to diabetes [11]. To translate this to the health care costs of an individual, a 2009 report suggested that for each risk factor a person develops (i.e., obesity, high blood pressure, etc.), health care costs can increase nearly 1.6-fold (approx. $2,000 USD). For each additional risk factor, those costs can rise by an average of 24% [12].

2. The Aetiology of Metabolic Syndrome

The aetiology of MS is beyond the scope of this entry and a more detailed discussion can be found elsewhere [13]. Briefly, insulin resistance and the underlying pathophysiological mechanisms that contribute to its development have been identified as major contributors to the development of MS [14][15]. Obesity has historically been regarded as one of the leading causes of MS, however, whether weight gain precedes hyperinsulinemia and insulin resistance or whether hyperinsulinemia drives weight gain and metabolic disease is often debated [16]. Evidence from studies investigating the use of exogenous insulin to manage both type 1 and type 2 diabetes has demonstrated that intensive control of blood glucose through increased insulin use resulted in weight gain [17] even when caloric intake was reduced [18]. Moreover, a recent meta-analysis of 60 longitudinal studies and randomised clinical trials revealed temporal sequencing between fasting insulin, body mass index and systemic inflammation [19]. Their findings showed that changes in fasting insulin preceded changes in weight gain and did not support the assertion that obesity comes first before elevated fasting insulin levels and disease development. Consequently, the authors suggested that hyperinsulinemia may be the driver of adverse

health consequences, rather than weight (obesity) itself [19]. While it is well acknowledged that obesity and elevated body fat levels alone can negatively impact the body's physiology and internal homeostasis, this area of ongoing research does raise questions regarding the most effective lifestyle and dietary approaches for preventing and reversing MS, with weight loss potentially becoming not the only target. Despite this, current guidelines and interventions still primarily focus on weight loss, meaning that moving beyond this would be timely.

3. Diet and Lifestyle Interventions—Moving beyond Weight Loss

The traditional diet and lifestyle interventions used to treat MS involve eating less and moving more. This approach aligns with the current guidelines for the management of overweight and obesity by the Australian National Health and Medical Research Council, which recommends caloric restriction, increased physical activity and behaviour modification to create a 2500 kilojoule energy deficit and achieve a 5–10% weight reduction [20]. While these guidelines can be successful, especially in the short term, prioritising weight loss as the primary goal for treating MS can be limiting. Firstly, these dietary and lifestyle approaches do not take into account the concept that energy input and expenditure are interdependent and complex, rather than 'one simply influences the other'. It is evident that energy balance is controlled by multiple feedback mechanisms that help maintain body weight within a narrow range [21]. Subsequently, many individuals that solely focus on an energy deficit model to lose weight often fail and tend to gain the same (and more) weight back [22].

In addition, the focus on weight loss inherently means targeting those with a higher BMI. This focus could increase the likelihood of overlooking at-risk populations with MS that fall within the normal-weighted BMI category. Identified as metabolically obese but normal weight (MONW), this subgroup of normal-weight individuals displaying obesity related phenotypic characteristics are at higher risk for type 2 diabetes and cardiovascular diseases [23]. A recent analysis of mortality rates in different weight categories amongst 12,047 US adults with and without MS found that the prevalence of MS in normal-weight individuals was 8.6% [24]. Interestingly, multivariate risk analysis demonstrated that these individuals displayed the highest mortality rate [24]. Furthermore, recent data from the UK and US suggest the application of different BMI cut-off points for certain ethnic groups such as South Asian, Arab, Chinese and Black populations that are at higher health risk at lower BMI thresholds [23][25]. Collectively, these observations indicate that MONW individuals should also be targeted for lifestyle intervention.

With new and emerging research in this area, there is growing support for moving the focus away from weight loss, typically by caloric restriction, and directing the attention toward the diet or eating pattern that is most appropriate for treating MS. While this topic is also highly debated, the current standard dietary recommendations for treating MS include promoting low intakes of saturated and trans fats, reducing consumption of simple sugars and increasing intakes of fruit, vegetables and whole grains [26]. Although these eating patterns are typically captured within the current dietary assessment tools, alternative and emerging dietary patterns/strategies that have demonstrated effectiveness for treatment and management of MS such as reducing overall carbohydrate intake, ultra-processed foods and timing of food intake are not [27][28][29].

3.1. Carbohydrate Restriction

The current Dietary Guidelines for Americans [30] recommend that half of calories consumed be derived from carbohydrates in an effort to limit the intake of dietary fat. US government data suggest that, in terms of percentage of total energy intake, consumption of carbohydrates has increased by 30% whilst consumption of fat has decreased by 25% since 1965 [31]. Given the association between MS and insulin resistance, overconsumption of carbohydrates, especially those known to induce large spikes in insulin levels, could be linked to MS development [31].

Contemporary evidence suggests that restricting dietary carbohydrates is an effective means of targeting a range of risk factors associated with MS with a single modification to dietary intake [32]. Clinical trials have shown that diets low in sugar and refined carbohydrates, while high in whole foods and healthy fats, can reduce atherosclerotic cardiovascular disease risk in overweight and obese adults [33]. This approach has demonstrated effectiveness when uniformly restricting carbohydrate intake in short-term studies, e.g., to 12% of total calories [32] and in long-term programs that adapted dietary intake of carbohydrates to suit the personal circumstances of participants [34]. Improvements in clinical features of MS such as elevated blood pressure, hyperglycaemia, weight and lipid profiles have also been shown to be both achievable and sustainable in the long term through moderate carbohydrate restriction (<120 g per day) in community settings through the avoidance of sugary and starchy foods such as breakfast cereals, bread, pasta and rice [35]. A dietary assessment tool that identifies high-starch foods, which might feature in every meal, and high-sugar foods provides the opportunity to discuss options for substituting with lower-carbohydrate alternatives.

3.2. Minimising Ultra-Processed Food

The term “ultra-processed food” (UPF) was coined in epidemiological studies that found an association between UPF consumption and a range of chronic diseases including MS, irritable bowel disease and cancer [36]. The hedonistic qualities of UPF are thought to encourage reward-driven eating and play a role in overriding biological controls of appetite and satiety [37], which impacts weight and health [38]. The extent to which food has been processed plays a part in its nutritional quality, so categorising food by levels of processing provides valuable information beyond defining foods simply by food group [39]. The NOVA food classification system developed at the University of Sao Paulo, Brazil, classifies foods into four categories: unprocessed or minimally processed (e.g., fresh meat and vegetables or pasteurised milk), processed culinary ingredients (e.g., sugar or oil), processed food (e.g., canned fish and fruit in syrup), and ultra-processed food (e.g., sweet or savoury packaged snacks and pre-prepared frozen meals). Critics of the NOVA classification system argue that it simply identifies food that are likely high in sugar, fat and salt [40] and therefore adds nothing new. While this is in part true, identifying and classifying these types of foods are helpful for a number of reasons. Firstly, evidence shows that the highly addictive potential of ultra-processed foods is related to their added sugar content [41]. Secondly, the rewarding properties of high-fat foods and their links to overconsumption appear to occur when fats and carbohydrates are consumed together [42]. Thus, while traditional dietary advice and definitions of a healthy diet have focused on specific nutrients in food, the NOVA classification system focuses on the extent and nature of food processing and identifying such foods or dietary patterns (via dietary assessment tools) could help further mitigate known factors that contribute to the development of MS.

3.3. Meal Timing and Frequency

Dietary patterns are considered central to combating metabolic diseases. However, there is an emerging acceptance that meal timing may be as or more important than the amount or type of food consumed [43], and combining these approaches may elicit greater benefits [44]. Surveys conducted between 1977 and 2006 demonstrated that daily “eating occasions” increased in adults and children, with energy intake, particularly from snacking, increasing and the time between “eating opportunities” decreaseing from 3.5 to 3 h [45]. Circadian rhythms, the 24 h cycles that are part of the body’s internal clock, run in the background to carry out essential functions and processes, including weight regulation. The central clock of the suprachiasmatic nucleus of the hypothalamus controls many circadian rhythms, as well as clocks located in other brain regions and most peripheral tissues [46]. Chronic circadian rhythm disruption, such as shift work or repetitive late night snacking, is a risk factor for metabolic diseases and both human and animal studies have demonstrated that time-restricted feeding can provide protection from circadian rhythm-induced metabolic disturbances [44]. Food is a non-photic stimulus that can reset the circadian rhythm by predominantly influencing the peripheral clocks and the timing of ‘when’ the majority of calories are eaten is an important factor [47]. For example, consuming the majority of calories in the evening or at night is associated with a higher risk of non-alcoholic fatty liver disease, whilst eating main meals earlier in the day is associated with decreased risk for hepatic steatosis [48]. Alternatively, it may involve restricting your window of eating during the day. Indeed, both early time-restricted eating (from 8:00 a.m. to 5:00 p.m.) and delayed time-restricted eating (from 12:00 p.m. to 9:00 p.m.) improved glycaemic response to a test meal, with the early pattern also leading to a decrease in fasting glucose [49]. Given the profound benefits on strategic meal timing/frequency on chronic disease risk factors such as MS, it is perhaps time to consider including such practices within dietary assessment tools, and, in doing so, help facilitate discussion with an individual about which timing strategy may best suit them.

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