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Managing obesity as a treatable trait of obstructive airway diseases★

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A B S T R A C T

Obesity is a non-communicable chronic disease that is growing in prevalence in all regions of the world. In adult populations obesity rates have doubled since the 1990s. Defined as an excess in fat deposits that deleteriously effect health, obesity is complex and leads to multimorbidity, and death from disease. In people with obstructive airway diseases, obesity is even more common and negatively impacts outcomes for people living with these coexisting diseases.

Treatable Traits is a personalised medicine approach to managing obstructive airway diseases, focusing on clinically relevant traits that are identified in individuals followed by the application of individualised interventions. In airway diseases traits are grouped within three domains: pulmonary, extra-pulmonary and behavioural/risk-factors. Obesity is a key extra-pulmonary trait which we purpose to be a super trait due to its impact on individuals living with the disease and the major improvements gained from successful treatment.

In this review we explore the clinical relevance of obesity as a treatable trait of obstructive airway diseases, the optimal approach to measuring and treating obesity, the mechanisms, and the person-centred recommendations for shared decision making. All these areas enable the management of obesity as a treatable trait.

1. Introduction

Obstructive airway diseases including asthma and chronic obstructive pulmonary disease (COPD) are common chronic respiratory conditions causing significant burden to people with these diseases, the health care system and society. Multimorbidity, defined as the presence of two or more chronic non communicable diseases that coexist in any one individual is the norm in people with asthma and COPD, and is associated with poor outcome [1–3]. Multimorbid conditions both compound and confound management and outcomes for people with obstructive airway diseases. Most guidelines for asthma and COPD are single disease focused, which in the most part fail to address the complexity and heterogeneity people are faced with. Single focused disease management is no longer feasible or acceptable, instead we must implement personalised, person-centred disease management for people with airway diseases [4]. An approach that has gained traction over the last 10 years is Treatable Traits [5–13].

1.1. Justification for this review

Obesity is prevalent in obstructive airway diseases and a treatable trait that complicates disease management contributing to increased disease severity and poor outcomes. Whilst these concerns are well recognised in the literature, obesity is usually managed as a background comorbidity in people with asthma and COPD rather than as a key treatable trait. This narrative review is needed to bring obesity to the forefront of disease management in obstructive airway diseases and to provide an approach through the treatable traits paradigm that enables improved recognition, treatment, and outcomes for people with asthma and COPD.

1.2. Aims

The aim of this review is to describe the treatable traits approach and specifically focus on one key trait that is highly prevalent among people with obstructive airway diseases: obesity. The structure of the review will first include an overview of obesity and its pathophysiological impacts, followed by an examination of the three essential criteria for determining a treatable trait. They are a). the clinical relevance of obesity in airway diseases, b). the measurement of obesity in practice, and c). its treatability. We will then address essential elements for person centred management of obesity, a central component of the treatable traits approach.

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1.3. Treatable Traits

Treatable Traits is a model of personalised disease management, first proposed in asthma and COPD, and now in multiple other diseases [14–16]. Treatable Traits moves away from a one size fits all approach to management, instead the approach focuses on identifying and treating specific clinically relevant traits. Traits are identified within three domains: pulmonary, extra pulmonary and behavioural/risk factors [11,13]. The extra pulmonary domain enables the management of multimorbidities like obesity.

Central to the treatable traits approach is that it delivers individualised treatment based on trait identification rather than disease label, it is holistic, going beyond the lungs, and it is person centred ensuing the patient is at the centre of their care [4,11,17]. Due to the complexity of asthma and COPD management care is usually multidisciplinary. A Treatable Traits model of care is an ideal vehicle that enables these components of care delivery [4].

To be defined as a treatable trait, a trait must be clinically relevant, measurable and treatable. In determining clinical relevance traits will associate with disease outcomes, such as symptoms, quality of life, lung function, exacerbation risk, and an increased risk of future events (for example, cardiovascular events and mortality) [18]. For identification there must be a trait identification marker that enables measurement, and for treatable/modifiable there must be an intervention that modifies the trait for improved outcomes.

A more recent concept that has emerged as part of the treatable traits' paradigm is 'Super Traits'. Super traits are defined as traits that are essential to identify and treat in order to effectively manage other traits, or traits that have such a large positive treatment effect that management is crucial, and that once identified and treated improvements occur in seemingly unrelated traits [7,19]. This concept emerged in recognition of the complexity of implementing treatable traits into practice, and the need to prioritise traits in primary care settings and in low to middle income settings [6,7,20–22]. Obesity has been proposed as a super trait as it is prevalent, has a major impact on outcome, and when treated leads to improvements in multiple other traits [6]. A key example of this was demonstrated in a study that aimed to treat obesity in people with COPD by intervening with a low-calorie diet and resistance exercise training. Not only did body mass improve, but there were significant improvements demonstrated in exercise tolerance, quality of life, anxiety and depression, and the BODE index, while maintaining skeletal muscle [23]. Ensuring that obesity is included in treatable traits management approaches is critical to improving outcomes.

2. Obesity

Obesity has been defined by the World Health Organization (WHO) as "a chronic complex disease defined by excessive adiposity that can impair health" [24]. Obesity is a highly prevalent condition, with rates continuing to rise in all parts of the globe [25]. Obesity affects 32 % of the general Australian population, but its prevalence is notably higher among people with asthma (41 %), severe asthma (46 %), and COPD (35–54 %) [26]. In people with obstructive airway diseases obesity can impact symptoms, treatment response and increase risk of exacerbation. Obesity is often refractory to intervention, particularly in clinically severe obesity, however there is now renewed opportunity to improve outcomes for people living with obesity due to new pharmacological therapies.

2.1. Potential mechanisms linking obesity with poorer outcomes

Although we know that obesity is associated with poorer asthma outcomes, the mechanisms responsible are not completely understood. Given that obesity is a complex disorder with varied aetiologies, it is also likely that the mechanisms driving the association between obesity and asthma are similarly complex. There is some evidence to suggest that inflammation, metabolic dysregulation, sex hormone abnormalities, mechanical effects of excess adiposity, poor diet quality and physical inactivity, may all have a role [27].

Our recent systematic review found that obesity is associated with increased airway inflammation in adults with asthma, with an increased proportion of neutrophils in the airway lumen (sputum %neutrophils) and an increased number of eosinophils in the lung tissue [28]. This is in line with hypothesis-free cluster analyses, which consistently demonstrate that obesity is associated with either a neutrophilic or mixed neutrophilic/eosinophilic pattern of airway inflammation [29–32]. Neutrophilic airway inflammation is associated with more severe asthma and the presence of comorbidities [33], and there are limited effective therapeutic options for neutrophilic asthma. The increased presence of neutrophilic asthma in people living with obesity may help explain the reduced efficacy of asthma pharmacotherapy in this population, which may contribute to poorer asthma control and increased exacerbation risk, highlighting the need for new effective therapies and optimal obesity approaches.

There is emerging evidence that metabolic dysregulation is associated with an increased prevalence of asthma, as well as more severe asthma. A cross-sectional study conducted in Sweden found that people with asthma have a higher prevalence of diabetes, dyslipidaemia and hypertension compared with the general population, while a lower FEV_1 %predicted is associated with diabetes and a higher HbA1c [34]. In another Swedish study, there was an association between asthma and type 2 diabetes that remained significant even after adjustment for BMI [35]. Given the apparent relationship between altered glucose metabolism and asthma, it is possible that normalising glucose concentrations may improve asthma.

Sex hormone changes influence asthma onset and severity, while obesity is linked with sex hormone abnormalities in both females and males [36–39]. We have previously shown that females with asthma using the oral contraceptive pill (OCP) have less neutrophilic airway inflammation, better asthma control and use less inhaled corticosteroid medication than females not using the OCP [39]. When analysed by BMI, women with obesity using the OCP have lower sputum %neutrophils than women with obesity not using the OCP. Brown and Carroll [40] have discovered that sex hormones and the OCP modify immune responses and asthma severity in experimental asthma, by modulating glucose transporter (GLUT)1 responses in the lungs [38]. GLUT1 is a dominant glucose transporter in lung cells and GLUT1-mediated glucose metabolism facilitates pro-inflammatory responses in immune cells [41,42], suggesting a potential interaction between sex hormones, glucose metabolism, and inflammation in asthma.

Dyslipidaemia is also associated with poorer asthma outcomes. Liu et al. [43] found dyslipidaemia is associated with an increased risk of severe non-allergic asthma, poorer asthma control, and airway obstruction, in adults with asthma. Statins may serve as a potential therapy, as a systematic review of 12 randomised controlled studies found that statins treatment improved asthma control and reduced sputum %eosinophils and IL-6, however there was no change in lung function and there was also significant heterogeneity in the airway inflammatory findings [44]. A real-world study conducted in Korea found statins were associated with fewer asthma exacerbations (including severe exacerbations) in adults with asthma, when compared with adults who had a normal blood lipid profile but were not using statins [45], suggesting the beneficial effects of statins may extend beyond their cholesterol-lowering effects. This contrasts with a mouse model of asthma, which found a correlation between BALF %neutrophils and serum total cholesterol [46]. Given the lack of certainty regarding whether potential beneficial effects of statins are due to their cholesterol-lowering effects or via another mechanism, further research in this area is warranted.

There is clear emerging evidence that metabolic dysregulation, which is more common in obesity, is associated with increased asthma severity. This raises the question regarding whether it is excess

adiposity, or the metabolic dysregulation that frequently co-exists with obesity, that is driving more severe asthma outcomes. Further research is warranted to better understand the relative contributions of adipose tissue and metabolic health on clinical asthma outcomes and inflammation.

2.2. Adipokines and inflammation

Adipokines are proteins synthesised by adipose tissue that can exert either pro-inflammatory or anti-inflammatory actions. Leptin is a pro-inflammatory adipokine that is higher in people living with obesity, including adults with asthma and obesity [47]. In a cross-sectional study of 90 women with asthma, Kalmarzi observed serum leptin concentration correlated with more severe asthma and worse lung function) (FEV_1/FVC) [48]. This observation is supported by other studies that have also observed leptin concentration is associated with increased asthma severity and/or worse lung function in adults with asthma [49,50]. However, research findings are mixed, with other studies reporting no relationship between leptin concentration and asthma outcomes [51,52].

Unlike leptin, adiponectin is considered an anti-inflammatory adipokine and increases insulin sensitivity. Adiponectin is lower in people with obesity, including adults with asthma and obesity [47]. Epidemiological studies have demonstrated that a low adiponectin concentration is associated with an increased prevalence and an increased incidence of asthma in women [53,54]. In people with asthma, a lower concentration of adiponectin has been shown to be associated with increased asthma severity [55]. However, this is not a universal finding, with one study observing a lower adiponectin concentration was associated with fewer asthma symptoms and lower asthma medication usage in men with asthma, while there was no relationship in women with asthma [56]. Other studies have observed no relationship between adiponectin and asthma outcomes or asthma severity [48,57,58]. Further research is warranted to clarify the potential role of adipokines in driving a more severe presentation of asthma in people living with obesity. These studies should examine whether effects on asthma outcomes vary according to sex.

2.3. Obesity and lung function

Obesity has direct adverse impacts on pulmonary function. This is partly due to physical compression of the chest cavity secondary to obesity which results in the restrictive spirometry pattern frequently observed on people living with obesity [59]. This is important in terms of diagnosis of obstructive airway diseases as it may result in misinterpretation with the obstructive pattern being overlooked. Also observed in individuals with obesity is reduced total lung capacity, expiratory reserve volume, functional residual capacity, PEF and FEV_1. As a consequence of reduced lung volumes, peripheral small airways tend to collapse. Reduced lung volumes may also lead to increased airway hyperresponsiveness which is also increased as a result of obesity and continuing weight gain, leading to increased disease severity in asthma [59,60].

Furthermore, there is a complex interrelationship between obstructive sleep apnoea (OSA), obesity, and obstructive airway diseases [61]. These conditions share common risk factors and comorbidities including, gastroesophageal reflux disease and rhinitis which mutually exacerbate disease severity [62]. Intermittent hypoxia and sleep fragmentation, cardinal features of OSA, may worsen respiratory symptoms and further complicate clinical management. Identifying the overlap between OSA, obesity, and asthma and/or COPD is therefore critical to enabling personalised, treatable-trait–based management strategies [63].

3. Obesity as a treatable trait in obstructive airway diseases

3.1. Criterion 1: clinical relevance of obesity

3.1.1. Asthma

Obesity not only increases the risk of developing asthma [64], but is also associated with a more severe presentation of asthma that is more difficult to manage. Studies demonstrate that the presence of obesity is associated with poorer asthma control and asthma-related quality of life, worse lung function, and more frequent exacerbations and asthma-related hospitalisations (including ICU admission), when compared with people with asthma who do not have obesity [65–68]. In adults with asthma, obesity is associated with an increased risk of all-cause mortality and cardiovascular mortality [69]. Indeed, obesity is estimated to have contributed to almost half of asthma-related deaths globally since 2013 and is a leading risk factor for disability adjusted life years in this population [70].

This increased asthma severity in people with obesity occurs despite increased use of inhaled corticosteroid medications, short- and long-acting bronchodilators, and maintenance oral corticosteroid medication [71]. This may be explained by the fact that asthma pharmacotherapy appears to be less effective in people living with obesity [72–74], which means a higher dose may be prescribed in an attempt to achieve asthma control. Nevertheless, in people with obesity the cornerstones of asthma management need to be applied including inhaled corticosteroids and bronchodilators, asthma self-management education, comorbidity management and harm minimisation through smoking cessation, vaccination, minimisation of oral corticosteroids, and in those with severe asthma, biologics.

Novel biologic medications are now standard care for people with severe asthma, with significant effects on exacerbation reduction and corticosteroid sparing effects. In adults living with obesity [75–79] and severe asthma the positive impact of biologics remains, but the effects may be attenuated. Studies by Sposato [76] and Gu [75] have shown blunted responses to biologic medication in people with obesity. Albers [77] found that mepolizumab reduced exacerbation risk, but had no effect on lung function in people with obesity. Indeed, obesity as a comorbidity in people with severe asthma using biologic treatments is a significant barrier to achieving the new therapeutic goal of asthma remission, according to a systematic review and meta-analysis involving 25 studies of which six studies contributed to a meta-analysis evaluating the impact of obesity on remission (0·41 [0·31–0·54]; $I^2 = 0$ %) [80].

In contrast, Omalizumab, an anti-IgE monoclonal antibody therapy, appears effective in patients with moderate to severe allergic asthma irrespective of BMI. In a pooled analysis of two RCTs involving 995 participants, there were larger reductions observed in exacerbation rate with increasing BMI (normal or underweight, −37.4 % [−69.0 %–26.8 %]; overweight, −52.7 % [−78.4 %–3.7 %]; obese, −71.9 % [−86.9 % to −39.5 %]). However, there were no differences in BMI categories for FEV_1. Oliveira [78] found omalizumab improved asthma symptoms and lung function, as well as reduced exacerbation risk, in people living with obesity, however this improvement coincided with a significant 5 % weight loss [78]. It is therefore not possible to disentangle the effects of weight loss from the effects of biologic medication in this study, but it does highlight the need for future studies to test whether obesity management at the commencement of biologic therapy augments its efficacy in people living with obesity and severe asthma.

Tezepelumab, an anti-thymic stromal lymphopoietin monoclonal antibody for the treatment of severe asthma has been shown to reduce asthma exacerbations irrespective of blood eosinophil count [81], data extracted from the DESTINATION. NAVIGATOR, and PATHWAY RCTs suggests Tezepelumab's effect on annualised exacerbations is independent of baseline BMI [82].

3.1.2. Chronic obstructive pulmonary disease (COPD)

The relationship between obesity and disease outcomes in people

with COPD is complex. While research indicates that obesity is associated with increased dyspnoea and activity limitation in COPD, the literature also suggests that obesity is associated with reduced mortality and a lower risk of COPD exacerbation [83]. This apparent contradiction has been labelled the "obesity paradox" and the mechanism(s) responsible are unclear. A 2024 systematic review including 120 studies and >1 million participants found a U-shaped association between obesity and all-cause mortality in people with COPD, whereby the apparent "protective effect" of obesity is lost once body mass index (BMI) exceeds a threshold of 35.25 kg/m^2 [84]. One possible explanation could be due to differential effects of lean mass versus fat mass. Given that obesity is associated with higher muscle strength [85], and that lower muscle mass is predictive of mortality in people with COPD [86,87], it is possible that the protective effect of obesity at the lower end of the BMI range is being driven by a higher lean mass in this population. On the other hand, once BMI exceeds the threshold of ~35 kg/m^2, the deleterious effects of excess adiposity may take over.

Indeed, Martínez-Luna et al. [88] have demonstrated that the positive relationship between BMI and lung function (FEV_1) is driven purely by lean mass, while there is no relationship between lung function and fat mass. In further support of this hypothesis, Pishgar et al. [89] quantified intramuscular fat and found it to be positively associated with increased mortality in COPD. It is also possible that some people within the normal weight range have experienced unintentional weight loss because of increased disease severity, which would also be associated with a reduced lean mass. Further research is therefore needed to better understand the impact of body composition (fat and lean mass quantity and distribution) on COPD outcomes, with these data collected alongside a patient weight history to better understand the relative contribution of unintentional weight loss. Nevertheless, in those with severe obesity and COPD, obesity is clinically relevant.

3.2. Criterion 2: measurement of obesity

Obesity is typically defined by BMI measurement, calculated as weight in kilograms divided by height in metres squared (kg/m^2). Classifications within BMI categories define the level of obesity-related health risk, with classifications ranging from Class I to Class III. In the United States, the United Kingdom, Canada and Qatar additional classes of Class IV and Class V are recognised [25,90–94].

This definition, operationalized through body mass index (BMI) > 30 kg/m^2, has been widely used in clinical practice and epidemiological studies. Despite its simplicity and utility in population-level screening, BMI has significant limitations [25].

As BMI is calculated as body weight in kilograms divided by height in metres squared, it does not differentiate between fat and lean mass and can both underestimate and overestimate adiposity. For instance, individuals with high muscle mass may be misclassified as obese, while those with normal BMI may harbor excess visceral fat and be at elevated health risk. Moreover, BMI fails to provide information about the functional status of organs and tissues, which is critical for assessing health at the individual level [25].

Recognising these shortcomings, the Lancet Diabetes & Endocrinology Commission advocated for a redefinition of obesity that aligns with clinical principles used in other chronic diseases [25]. They proposed adequacy of BMI-based definitions and a more nuanced framework for diagnosing obesity as a disease. According to the new definition, "obesity is characterized by excessive adiposity, with or without abnormal distribution or function of the adipose tissue."(25) The Commission recommended that traditional obesity measures based solely on BMI should be regarded only as a proxy indicator of health risk in population-level research, epidemiological studies, or initial screening, rather than for clinical diagnosis.

The current clinical assessment of obesity requires confirmation of excess or abnormal fat through one of several recommended methods. Preferably, direct body fat measurement, such as dual-energy x-ray absorptiometry (DEXA) or bioimpedance, should be used. Computer tomography and magnetic resonance imaging (MRI) may also be used to objectively characterise obesity. The advantages of these imaging techniques is that they can quantify compartmental volumes of adipose tissue and muscles. Of these methods MRI is preferred due to the absence of ionizing radiation [95]. However access to these methods for obesity diagnosis in practice is limited. Alternatively, obesity can be confirmed using BMI ≥30 kg/m^2 combined with at least one anthropometric measure – such as waist circumference, waist-to-hip ratio, or waist-to-height ratio, which may be more feasible in the clinic. If a person has at least two anthropometric measures characterising excess of fat, regardless of BMI, obesity can also be confirmed [25].

Following the confirmation of excess body fat, further assessment should be conducted to establish whether an individual has pre-clinical obesity or clinical obesity [25]. Pre-clinical obesity refers to an excess adiposity with preserved organ and tissue function. Individuals in this category may be at increased risk of developing non-communicable diseases such as type 2 diabetes, cardiovascular disease, and certain cancers, but do not yet exhibit signs of illness. In contrast, clinical obesity is defined as a chronic, systemic illness resulting from the direct impact of excess adiposity on organ and tissue function. Clinical obesity is present when there are signs/symptoms of organ dysfunction associated with obesity or if the individual experiences age-adjusted limitations of daily activities. This state may lead to life-altering complications including heart failure, stroke, and renal impairment.

Fig. 1 provides examples of diagnoses based on this updated approach, along with further details on the relevant anthropometric measurements. This new approach addresses concerns about overdiagnosis associated with BMI-centric definitions, which may label healthy individuals as diseased or overlook those with significant health risks.

3.3. Criterion 3: obesity treatment in obstructive airway diseases

The Global Initiative for Asthma (GINA) guidelines recommend weight loss as part of the comprehensive management of people with asthma [96] and obesity, and the Global Initiative for Chronic Obstructive Lung Disease (GOLD) strategy for COPD acknowledges the burden of obesity and the need for individualised nutritional care [97]. Personalising obesity management requires determination of the optimal intervention: behavioural, pharmacological, or surgical, in line with the patient's clinical profile, goals and preferences.

Regardless of the choice of intervention, how obesity is addressed is key in health outcomes. Stigma, both interpersonal and systemic, can discourage people from engaging in care, increase psychological distress and undermine health outcomes. Weight-inclusive, person-centred communication, such as "person living with obesity" instead of "obese patients" is fundamental in nurturing trust, reducing shame and promoting engagement [98,99].

3.3.1. Lifestyle interventions

Lifestyle interventions are generally the first approach in managing obesity as a treatable trait and aim to achieve weight loss through calorie reduction, increased physical activity or a combination of these. In individuals with asthma and obesity, a modest weight loss (5–10 %) is associated with improved asthma symptoms and lung function, a reduction in exacerbations and enhanced quality of life [100]. Scott et al. showed that a 10-week programme combining dietary restriction and exercise led to significant improvements in asthma control and asthma-related quality of life [100]. Likewise a three-month pulmonary rehabilitation programme was associated with improved asthma control and reduced BMI [101], while a 10-week meal plan tailored to individual nutrition goals led to improvements in both asthma control and quality of life [102]. Sharma and colleagues reported the results of a small RCT (N = 36) of weight management regimens in adults with difficult to treat asthma and a BMI ≥30 kg/m^2. The regimen consisted of

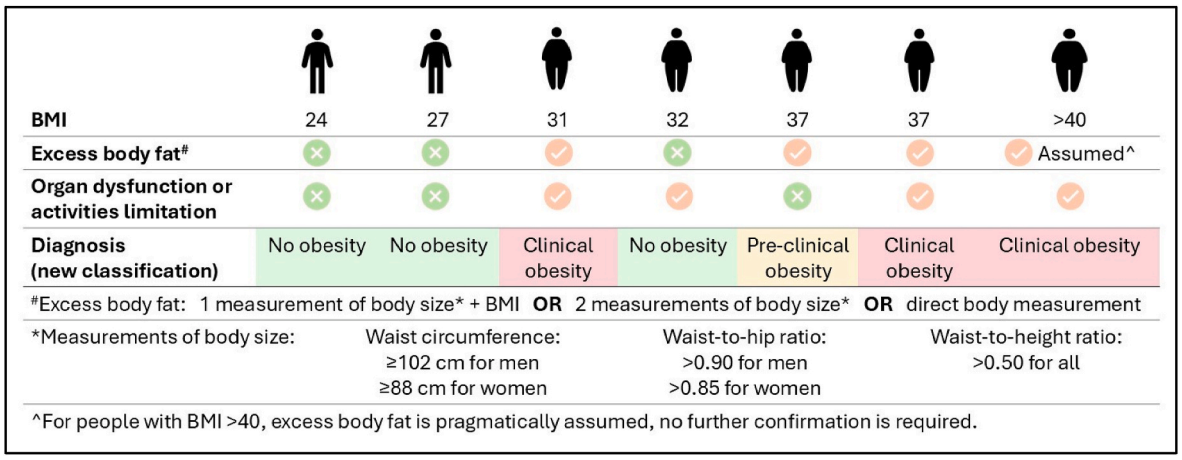

Fig. 1. Diagnosis of pre-clinical and clinical obesity according to the new definition. Values for white Caucasian population. Criteria for other ethnic groups may be different.

dietitian support combined with a low-calorie diet (850 kcal/day) utilising nutritionally complete meal replacements for 12 weeks, followed by food reintroduction and maintenance. The regimen resulted in greater weight change (median, −14 kg [Q1,Q3, −15 to −9 kg]) compared with usual care (median, 2 kg [Q1,Q3, −7 to 8 kg]; P = 0.015) at 52 weeks, and a greater proportion of participants achieving the MCID in the Asthma Quality of Life Questionnaire compared to usual care (71 % vs 6 %, respectively; P < 0.001). However, they found no between-group differences in ACQ-6. While the median exacerbation frequency reduced over 52 weeks from 4 (Q1,Q3, 2 to 5) to 0 (Q1,Q3, 0 to 2) (P < 0.001), no between-group difference was observed [103].

Whilst several RCTs have tested lifestyle interventions in people with asthma and obesity there is observed heterogeneity in the interventions and the effects. To address this Abbasalizad Farhangi and colleagues reported the results of a systematic review of the impact of weight loss on asthma control. Included were 12 RCTs and all but one (which involved diet plus pharmacotherapy), were lifestyle interventions involving diet, physical activity, exercise, and behavioural change, or any combination of these. Overall, weight loss resulted in improved asthma control and FEV_1 [104], however due to the small number of RCTs included and methodological quality further research to determine the optimal intervention in people with asthma is needed.

Although fewer studies exist in people with COPD, results are promising. McDonald et al. demonstrated that a supervised 12-week programme involving a low-energy high-protein diet and resistance training in people with a BMI ≥30kg/m^2 reduced BMI and improved dyspnoea, exercise tolerance, symptoms of depression and quality of life while preserving muscle mass [105]. This study, while small and lacking a control group, does highlight the benefits that targeting of one treatable trait has on multiple outcomes.

Beyond weight loss, diet quality itself affects respiratory outcomes. Diets high in ultra-processed foods have been associated with increased asthma severity, while diets rich in fibre, antioxidants and healthy fats have been linked to reduced inflammation and better asthma control [106–109]. It is important that lifestyle interventions do not become weight-centric in messaging [98]. Focusing on improvements in diet, breathing, sleep and confidence, rather than appearance can reduce stigma and improve long-term engagement [98].

3.3.2. *Pharmacotherapy*

Pharmacotherapy can be appropriate for individuals with a BMI ≥30 kg/m^2, or ≥27 kg/m^2 in the presence of a comorbidity. GLP-1 receptor agonists, such a liraglutide and semaglutide, are effective for weight loss and it is possible that they may improve asthma outcomes. Foer et al. conducted a retrospective health record review of patients with asthma and type 2 diabetes and found use of GLP-1R agonist medication was associated with a significant reduction in asthma exacerbation risk compared with patients using basal insulin, sulfonylureas, SGL-2 (sodium-glucose cotransporter-2) inhibitors, or DPP-4 (dipeptidyl peptidase-4) inhibitors [110,111]. It is important to note that in non-asthma populations, lean body mass loss has been observed during GLP-1 therapy [112] highlighting the need for personalised adjunct nutritional, physical activity and strength training support during treatment.

Metformin has been shown to reduce the risk of experiencing an asthma exacerbation by approximately 30 % in a large cohort study, while addition of a GLP-1 receptor agonist further reduced this risk [113]. However, interestingly, this beneficial effect was independent of glucose control, BMI, and asthma phenotype. While this leaves the mechanism driving these improvements uncertain, it does highlight the need to conduct clinical trials that test metformin and/or GLP-1 receptor agonists as potential therapeutic options for people living with obesity and airway disease. Currently long-term asthma specific outcomes are missing, however there are several registered trials looking to further explore GLP-1 therapy in asthma (NCT05254314, ACTRN12624001278527p).

In COPD, evidence is limited, however, emerging data suggests GLP-1 receptor agonists may reduce exacerbations [114] and, in addition to weight loss, increase pulmonary function [115]. As with all intervention options, pharmacotherapy should be offered through a shared decision-making process and framed as a valid option – and not a last resort due to failure.

3.3.3. *Bariatric surgery*

For some individuals with a BMI ≥40 kg/m^2, or 35 kg/m^2 in the presence of comorbidities and uncontrolled disease, bariatric surgery may offer substantial benefits. In asthma, surgery has been associated with reduced symptoms, fewer exacerbations and improved quality of life [116,117]. Studies by Dixon and Boulet have shown improved lung mechanics, reduced medication use and decreased airway inflammation post-surgery [118,119]. Despite the efficacy of bariatric surgery on weight loss and asthma outcomes it is largely unavailable to most patients due to cost.

In COPD, bariatric surgery may reduce exacerbation and hospitalisation risk, and morbidity from acute exacerbations [120,121]. Due to the higher perioperative risks associated with advanced disease, surgical referrals need to be individualised and consider health status, risk profile and the individual's preferences. For all surgical interventions, the post-operative care delivered by a multidisciplinary team is just as critical as pre-operative preparation to ensure sustained success [122].

3.4. Integrating personalised care

Whilst clinicians have the best intentions for the patients they treat, there is still an unacceptable stigma associated with obesity and this can impact how the individual will engage in care, and how effective the therapeutic relationship will be between the patient and the clinicians. Stigma reduces care-seeking, worsens adherence and affects mental health [98].

In a 2023 qualitative study of 20 health care professionals (HCP) involved in the management of inpatients living with clinically severe obesity, a key theme identified was 'barriers'. Within this theme was a sub theme of 'provider issues' providing key insights that related to a lack of teamwork in caring for this population, a prevailing culture of blaming the patient, and a gap in HCP education, knowledge and training. Furthermore, while some HCPs discussed improvements in staff attitudes and a reduction in stigmatization, there were also many recent examples provided that identified inappropriate hospital staff behaviour [123], indicating that stigma around obesity continues. Therefore, weight-inclusive care that prioritises behaviour change, and symptom relief above weight loss alone is needed in practice.

As health professionals our language matters, particularly in pursuit of person-centred care. Conversations about obesity need to be respectful and empathetic; by inviting the conversation and ensuing we use language that patients are comfortable with. The behavioural change model of the 5 As (Ask, Assess, Advise, Agree, Assist) used for many years in smoking cessation strategies has now been proposed as an approach to treating people living with obesity [124]. An example of how this can be used in the context of obesity, incorporating appropriate language, effective behavioural, lifestyle, pharmacological and surgical treatments, and referral and follow up as part of a holistic approach is outlined in Fig. 2 [124]. Patient engagement and shared decision making must be a priority in identifying and treating this super extra pulmonary trait.

When managing obesity as a treatable trait in people with airway disease, it is important to understand the clinical implications of obesity on lung and holistic health, personalise the approach to treatment using nutritional and physical activity support in addition to standard asthma and COPD treatment, calling on multidisciplinary colleagues to support interventions, and deliver care in way that is supportive and empathetic. This is the treatable traits approach (Fig. 3).

4. Conclusion

Obesity in obstructive airways diseases is common and has deleterious effects on patient outcomes. Given its impact, and the major improvements in multiple outcomes with successful treatment, we propose obesity as a super trait. Management of obesity requires shared decision making and supportive therapeutic relationships. Treatment interventions include lifestyle changes, pharmacotherapy, and surgery, but treatment must be personalised according to the needs of the patient. A treatable traits approach is an ideal model for the management of obesity in people with obstructive airway diseases.

CRediT authorship contribution statement

Vanessa M. McDonald: Writing – review & editing, Writing – original draft, Visualization, Supervision, Resources, Methodology, Conceptualization. **Tamara Blickisdorf:** Writing – review & editing, Writing – original draft, Project administration, Conceptualization. **Joice M. de Oliveira:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Hayley A. Scott:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization.

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Declaration of competing interest

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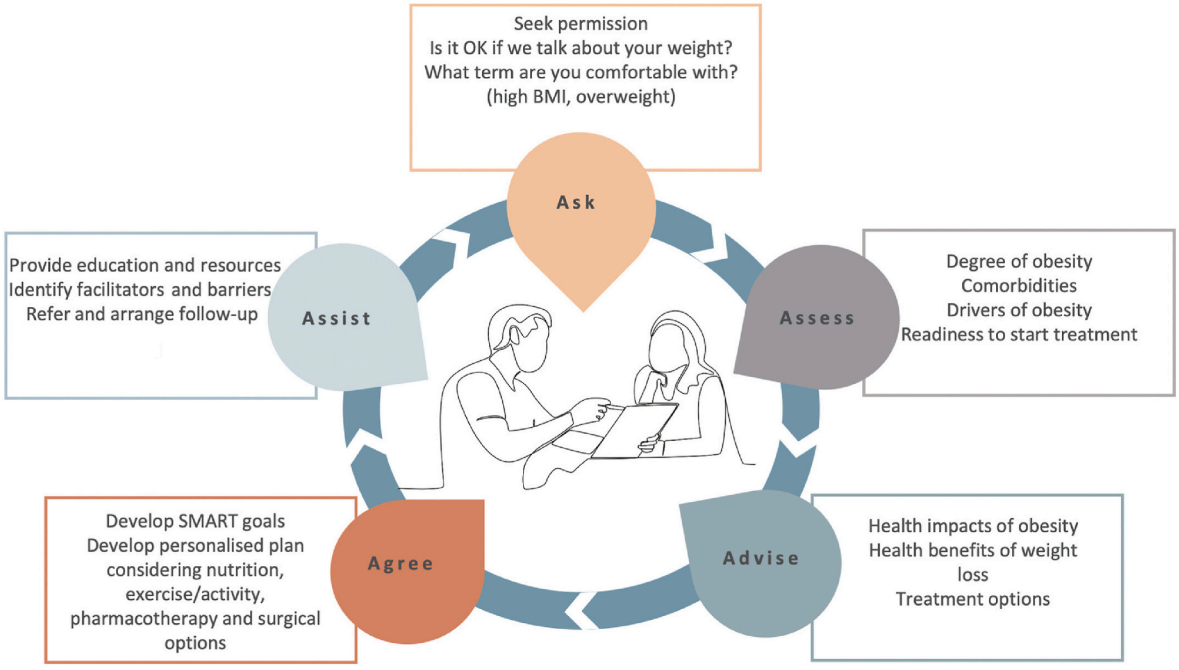

Fig. 2. A model using the 5 As of behaviour change to guide interactions with people living with obesity for person centred management. Content has been reproduced with permission from the Centre of Excellence in Treatable Traits, originally developed as part of the Centre of Excellence in Treatable Traits (https://treatabletraits.org.au).

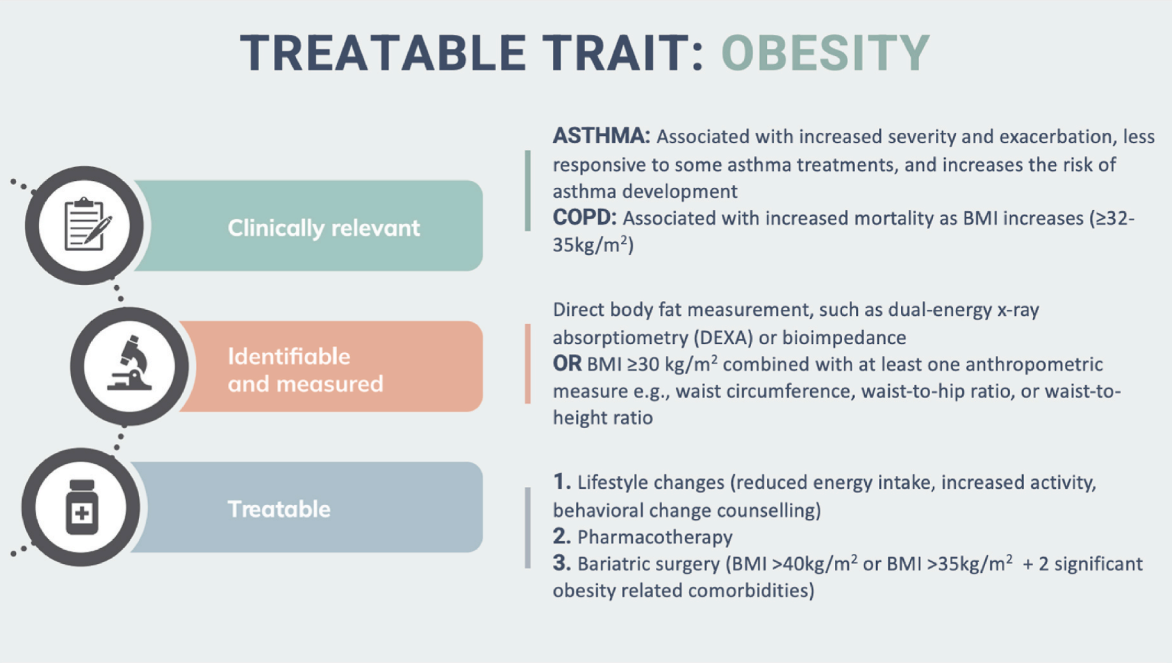

Fig. 3. Obesity as a treatable trait. Content has been reproduced with permission from the Centre of Excellence in Treatable Traits, originally developed as part of the Centre of Excellence in Treatable Traits (https://treatabletraits.org.au).

TB has no conflicts of interest to declare.
JMO has no conflicts of interest to declare.
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