**RESEARCH**

# Eating disorders and physical multimorbidity in the English general population

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## Abstract

**Purpose** People with eating disorders may be at increased risk for physical health problems, but there are no data on the relationship between eating disorders and physical multimorbidity (i.e., ≥ 2 physical conditions) and its potential mediators. Thus, we investigated this association in a representative sample of adults from the UK, and quantified the extent to which this can be explained by various psychological and physical conditions, and lifestyle factors.

**Methods** Cross-sectional data of the 2007 Adult Psychiatric Morbidity Survey were analyzed. Questions from the five-item SCOFF screening instrument were used to identify possible eating disorder. Respondents were asked about 20 physical health conditions. Multivariable logistic regression and mediation analysis were conducted.

**Results** Data on 7403 individuals aged ≥ 16 years were analyzed [mean (SD) age 46.3 (18.6) years; 48.6% males]. After adjustment, possible eating disorder was associated with 2.11 (95%CI = 1.67–2.67) times higher odds for physical multimorbidity. Anxiety disorder explained the largest proportion this association (mediated percentage 26.3%), followed by insomnia (21.8%), perceived stress (13.4%), depression (13.1%), obesity (13.0%), and alcohol dependence (4.3%).

**Conclusion** Future longitudinal studies are warranted to understand potential causality and the underlying mechanisms in the association between eating disorder and multimorbidity, and whether addressing the identified potential mediators in people with eating disorders can reduce multimorbidity.

**Keywords** Eating disorder · Disordered eating · Chronic disease · Multimorbidity · UK · Adults · Epidemiology

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## Introduction

When two or more physical conditions exist simultaneously in an individual, this condition is called physical multimorbidity [1]. Multimorbidity is an important risk concept as it is associated with adverse outcomes such as lower quality of life [2], unmet need, sickness days, reduced perceived health status [3], and higher risk for premature mortality [4]. In addition, a recent systematic review found that multimorbidity is associated with increased total healthcare costs including hospital costs, care transition costs, and costs for primary care, dental care, emergency department use, and hospitalizations [5]. Multimorbidity has a global prevalence of 42.4% [6], and in the UK, the prevalence of multimorbidity has been reported to be high (approximately 54% for those aged ≥ 65 years) and this is predicted to rise [7] mainly due to rapid ageing occurring in this country. Considering the profound impact of multimorbidity and its high

prevalence, it is of utmost importance to identify groups at particularly high risk for this condition to inform targeted intervention and policy.

One group at potentially high risk for multimorbidity are those with eating disorders or wider abnormal eating behavior, as previous studies have shown that these conditions may increase risk for a variety of physical conditions such as osteoporosis [8], endocrine disorders, and kidney failure [9]. Eating disorders can be defined as a pathological relationship with food that leads to significant disruptions in a person's day to day life, and can lead to severe consequences, including premature mortality. These disorders have defined clinical criteria based on the Diagnostic and Statistical manual of mental disorders (DSM-5-TR) [10], or the International Classification of Diseases 11 (ICD-11) [11], and an estimated 70 million people worldwide suffer from eating disorders [12]. In particular, in the UK, it is thought that between 1.25 and 3.4 million people have an eating disorder [13]. A recent systematic review reported that the prevalence of eating disorders in adolescents and children was 22.4% [14], with some primary studies indicating higher prevalence rates for adults (~31%) [15], suggesting that this is a public health problem that needs to be addressed.

Eating disorders may lead to a higher risk for multimorbidity, for example, via low-grade inflammation [16]. For example, in the case of binge eating disorders, inflammation may be increased via overweight/obesity that often results in an increased production of free fatty acids, interleukin-6, tumor necrosis factor-α, hs-CRP, and rapid eating practices that result in elevated serum lipids [17, 18]. Indeed, inflammation can increase risk for multiple chronic conditions and multimorbidity, via alternations in immune function, for instance [19–23]. Moreover, eating disorders have been found to be associated with low bone mineral density [24], which in turn can increase risk for multiple chronic conditions (e.g., arthritis, osteopenia, osteoporosis) [25–27]. Finally, it is also possible that the association between eating disorders and multimorbidity is mediated by psychological factors (e.g., insomnia, stress, depression), lifestyle factors (e.g., smoking, alcohol consumption), and physical conditions (e.g., obesity, underweight) [28–41].

Despite the potential importance of eating disorders as a risk factor for multimorbidity, to the best of the authors' knowledge, no research currently exists on the association between eating disorders and physical multimorbidity. Given this background, the aim of the present study was to investigate the association between eating disorder symptoms and possible eating disorder (≥2 eating disorder symptoms) in a sample of 7403 individuals aged ≥16 years who participated in the UK Adult Psychiatric Morbidity Survey. A further aim was to identify to what extent a variety of psychological, lifestyle, and physical factors may explain this association.

## Methods

### The survey

Cross-sectional data of the 2007 Adult Psychiatric Morbidity Survey were analyzed. Survey details are available elsewhere [42, 43]. In brief, this was a representative survey of the English adult population (aged ≥16 years) residing in private households. Fieldwork was undertaken by the National Center for Social Research, in conjunction with the University of Leicester in October 2006 to December 2007. A multistage stratified probability sampling design was used. The small user postcode address file was used as the sampling frame with postcode sectors as the primary sampling units. Addresses were stratified by region and by socio-economic groupings. A Kish grid was used to randomly select one respondent when there was more than one person in a household. Computer-assisted personal interviewing (CAPI) was used to obtain information from respondents. The survey response rate was 57%. To account for non-response and the probability of selection, sampling weights were generated so that the sample was representative of the English population. The Royal Free Hospital and Medical School Research Ethics Committee provided ethical approval for the survey with informed consent being obtained from all participants.

### Physical conditions and physical multimorbidity

Respondents were asked about 20 physical health conditions (cancer, diabetes, epilepsy, migraine, cataracts/eyesight problems, ear/hearing problems, stroke, heart attack/angina, high blood pressure, bronchitis/emphysema, asthma, allergies, stomach ulcer or other digestive problems, liver problems, bowel/colon problems, bladder problems/incontinence, arthritis, bone/back/joint/muscle problems, infectious disease, and skin problems). To be counted, conditions had to have been diagnosed by a doctor or other health professional and have been present in the previous 12 months. The number of physical conditions was summed and categorized as 0, 1, 2, 3, 4, and ≥5. Multimorbidity was defined as two or more physical conditions [44].

### Eating disorder symptoms and possible eating disorder

Five items from the SCOFF screening tool [45] were used to assess eating disorder symptoms and possible eating disorder, and consisted of whether, in the past year, the participant: had lost more than one stone (6.35 kg) in 3 months (weight loss); had made him/herself vomit because he/she

felt uncomfortably full (self-sick for feeling full); worried that he/she had lost control over how much he/she eats (uncontrolled eating); believed to be fat when others said that he/she was too thin (self-perceived fatness); and thought that food dominated his/her life (food dominance). An affirmative reply to at least two items was considered a positive screen, and therefore classified as 'possible eating disorder' [45]. Although the SCOFF questionnaire is not a diagnostic instrument, it has been found to have high specificity (87–94%) and sensitivity (84–100%) in accordance with clinical diagnoses [45–48].

## Mediators

The potential mediators or influential factors in the association between possible eating disorder and physical multimorbidity considered in this study were insomnia, depression, anxiety disorder, alcohol dependence, lifetime smoking, perceived stress, obesity, and underweight.

Insomnia was defined as fulfilling all the following three criteria: (i) problems trying to get to sleep or getting back to sleep (if had woken up) in the past month; (ii) had problems with sleep on at least 4 nights in the past 7 nights in addition to (iii) taking at least 1 h trying to get to sleep on the night with least sleep [49]. The Clinical Interview Schedule Revised (CIS-R), which can be administered by lay interviewers, was used to generate ICD-10 diagnoses of depressive episode and anxiety disorder (generalized anxiety disorder, panic disorder, phobia, obsessive–compulsive disorder) in the prior week [50]. Alcohol consumption was assessed with the Alcohol Use Disorders Identification Test (AUDIT) [51]. Respondents whose AUDIT score was 10 or above were also assessed for alcohol dependence. This was done with the Severity of Alcohol Dependence Questionnaire (SADQ-C) [52], with a score of 4 or more (out of 60) being used to establish past 6-month alcohol dependence. Lifetime smoking referred to answering affirmatively to the question "Have you ever smoked a cigarette?" Participants were asked if their tasks at work and home very stressful with answer options "Most of the time", "Usually", "Occasionally", and "Not at all". Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared based on self-reported weight and height. Using the standard WHO definition, obesity referred to BMI $\geq 30$ kg/m$^2$, and underweight BMI $< 18.5$ kg/m$^2$.

## Control variables

The control variables included sociodemographic variables (i.e., age, sex, education, ethnicity). Education referred to presence or absence of qualification (degree, non-degree, A-level, GCSE, other), and ethnicity was dichotomized into White British or other.

## Statistical analysis

The analyses were done with Stata version 14.2 (Stata Corp LP, College Station, Texas). The difference in sample characteristics between those with and without possible eating disorder was tested by Chi-squared tests except for age which was a continuous variable (Student's $t$-test). Multivariable logistic regression analysis was done to assess the association between individual eating disorder symptoms or possible eating disorder (exposures) and individual physical conditions or physical multimorbidity (outcome), while adjusting for age, sex, education and ethnicity. Analyses with individual eating disorder symptoms as the exposure were also adjusted for the presence of $\geq 2$ individual eating disorder symptoms as different eating disorder symptoms tend to co-exist. We also tested for interaction by sex in the association between the individual eating disorder symptoms or possible eating disorder and physical multimorbidity by including the product terms of these conditions and sex in the model but since preliminary analysis showed that there is no significant interaction, the analyses were not stratified by sex. Finally, in order to gain an understanding on the extent to which various factors (i.e., insomnia, depression, anxiety disorder, alcohol dependence, lifetime smoking, perceived stress, obesity, underweight) may explain the relation between possible eating disorder and physical multimorbidity, mediation analysis using the stata *khb* (Karlson Holm Breen) command in Stata was conducted [53]. This method can be applied in logistic regression models and decomposes the total effect of a variable into direct and indirect effects. Using this method, the percentage of the main association explained by the mediator can also be calculated (mediated percentage). Each potential mediator was included in the model individually, with the exception of the model which included all the potential mediators simultaneously. The mediation analysis controlled for sex, age, education, and ethnicity. In order to generate nationally representative estimates, in all analyses, the sample weighting and the complex study design were taken into account. Odds ratios (OR) and their 95% confidence intervals (95%CI) are reported. The level of statistical significance was set at $p < 0.05$.

## Results

A total of 7403 individuals aged $\geq 16$ years were included in the analysis. The prevalence of possible eating disorder was 6.4%, while the prevalence of the individual eating disorder symptoms were: weight loss 10.8%; self-sick for feeling full 3.0%; uncontrolled eating 7.3%; self-perceived fatness 5.6%; food dominance 3.6%. The prevalence of physical multimorbidity was 35.1%. The sample characteristics are provided in Table 1. The mean (SD) age of the sample was 46.3

**Table 1** Sample characteristics (overall and by possible eating disorder)

| Characteristic | | Overall | Possible eating disorder | | p-value |
|---|---|---|---|---|---|
| | | | No | Yes | |
| Age (years) | Mean (SD) | 46.3 (18.6) | 47.0 (18.6) | 35.8 (14.2) | <0.001 |
| Sex | Male | 48.6 | 50.1 | 26.5 | <0.001 |
| | Female | 51.4 | 49.9 | 73.5 | |
| Qualification | No | 23.9 | 24.5 | 14.3 | <0.001 |
| | Yes | 76.1 | 75.5 | 85.7 | |
| Ethnicity | British White | 85.2 | 85.4 | 82.6 | 0.206 |
| | Other | 14.8 | 14.6 | 17.4 | |
| Insomnia | No | 86.3 | 87.4 | 70.0 | <0.001 |
| | Yes | 13.7 | 12.6 | 30.0 | |
| Depression | No | 97.0 | 97.7 | 88.1 | <0.001 |
| | Yes | 3.0 | 2.3 | 11.9 | |
| Anxiety | No | 93.3 | 94.6 | 74.7 | <0.001 |
| | Yes | 6.7 | 5.4 | 25.3 | |
| Alcohol dependence | No | 94.1 | 94.7 | 85.1 | <0.001 |
| | Yes | 5.9 | 5.3 | 14.9 | |
| Lifetime smoking | No | 34.7 | 34.8 | 33.8 | 0.707 |
| | Yes | 65.3 | 65.2 | 66.2 | |
| Perceived stress | Most of the time | 5.7 | 5.1 | 14.2 | <0.001 |
| | Usually | 7.0 | 6.5 | 14.1 | |
| | Occasionally | 47.9 | 47.5 | 54.0 | |
| | Not at all | 39.4 | 40.8 | 17.7 | |
| Obesity | No | 82.3 | 83.1 | 70.2 | <0.001 |
| | Yes | 17.7 | 16.9 | 29.8 | |
| Underweight | No | 97.5 | 97.6 | 97.1 | 0.585 |
| | Yes | 2.5 | 2.4 | 2.9 | |

*SD* standard deviation

Data are % unless otherwise stated

Possible eating disorder referred to SCOFF screen-positive

*p*-value was calculated by Chi-squared tests except for age (Student's *t*-test)

(18.6) years and 48.6% were males. People with possible eating disorder were significantly more likely to be younger, females, and have higher levels of education. Furthermore, they were also significantly more likely to have sleep problems, depression, anxiety, alcohol dependence, higher levels of perceived stress, and obesity.

The prevalence of possible eating disorder increased linearly with increasing number of physical conditions (Fig. 1). Specifically, the prevalence was 5.8% among those with no physical conditions but this increased to 8.5% among those with ≥4 physical conditions. In terms of the individual physical conditions, possible eating disorder was significantly associated with higher odds for allergy, arthritis, asthma, bladder problems/incontinence, problems with back/bone/joint/muscle, bowel/colon problems, cancer, cataracts/eyesight problems, infectious diseases, liver problems, migraines/frequent headaches, skin problems, and stomach ulcer or other digestive problems (Table 2). Next, in terms of the individual eating disorder symptoms, weight loss
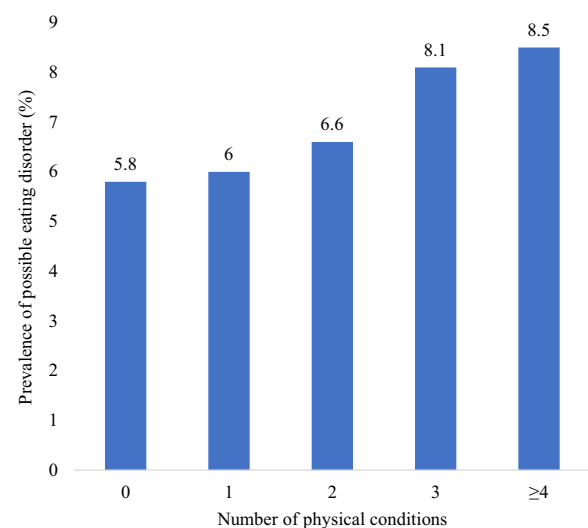


**Fig. 1** Prevalence of possible eating disorder by number of physical conditions

**Table 2** Association between possible eating disorder and individual physical condition (outcome) estimated by multivariable logistic regression

| Physical condition | OR | [95%CI] |
|---|---|---|
| Allergy | 1.47* | [1.08,2.02] |
| Arthritis | 1.60** | [1.19,2.14] |
| Asthma | 1.63** | [1.18,2.23] |
| Bladder problems/incontinence | 2.18** | [1.26,3.77] |
| Bone, back, joint or muscle problems | 1.38** | [1.09,1.74] |
| Bowel/colon problems | 2.19*** | [1.44,3.34] |
| Bronchitis/emphysema | 1.81 | [0.98,3.34] |
| Cancer | 3.70** | [1.56,8.76] |
| Cataracts/eyesight problems | 1.52** | [1.13,2.06] |
| Diabetes | 1.61 | [0.93,2.78] |
| Ear/hearing problems | 1.40 | [0.87,2.26] |
| Epilepsy/fits | 2.36 | [0.97,5.69] |
| Heart attack/angina | 1.60 | [0.73,3.52] |
| High blood pressure | 1.29 | [0.92,1.80] |
| Infectious disease | 6.77*** | [3.04,15.06] |
| Liver problems | 2.40* | [1.07,5.39] |
| Migraine or frequent headaches | 1.84*** | [1.33,2.54] |
| Skin problems | 1.52** | [1.13,2.04] |
| Stomach ulcer or other digestive problems | 2.14*** | [1.46,3.14] |
| Stroke | 2.81 | [0.52,15.08] |

*OR* odds ratio, *CI* confidence interval

Models are adjusted for age, sex, education, and ethnicity

Possible eating disorder referred to SCOFF screen-positive

*p < 0.05, **p < 0.01, ***p < 0.001

**Table 3** Association between individual eating disorder symptoms or possible eating disorder and physical multimorbidity (outcome) estimated by multivariable logistic regression

| | OR | 95%CI |
|---|---|---|
| Weight loss | 1.64*** | [1.37,1.97] |
| Self-sick for feeling full | 1.32 | [0.89,1.94] |
| Uncontrolled eating | 1.87*** | [1.34,2.60] |
| Self-perceived fatness | 1.69*** | [1.28,2.23] |
| Food dominance | 1.13 | [0.77,1.67] |
| Possible eating disorder | 2.11*** | [1.67,2.67] |

*OR* odds ratio, *CI* confidence interval

Models are adjusted for age, sex, education, and ethnicity. Analyses with the individual eating disorders as the exposure are also adjusted for presence of ≥ 2 eating disorder symptoms

The definitions of eating disorder symptoms and possible eating disorder were: weight loss (had lost more than 6.35 kg in 3 months); self-sick for feeling full (had made him/herself be sick because he/she felt uncomfortably full); uncontrolled eating (worried he/she had lost control over how much he/she eats); self-perceived fatness (believed to be fat when others said that he/she was too thin); food dominance (thought that food dominated his/her life); possible eating disorder (SCOFF screen-positive)

Physical multimorbidity referred to ≥ 2 physical conditions

***p < 0.001

was significantly associated with bowel/colon problems, cancer, cataracts/eyesight problems, heart attack/angina, high blood pressure, liver problems, migraine or frequent headaches, and stomach ulcer or other digestive problems (Appendix Table 5). Furthermore, uncontrolled eating was significantly associated with higher odds for asthma, bladder problems/incontinence, bowel/colon problems, diabetes, and high blood pressure, and self-perceived fatness with asthma, bowel/colon problems, skin problems, and stroke, while food dominance was significantly associated only with arthritis and liver problems.

Weight loss (OR = 1.64; 95%CI = 1.37–1.97), uncontrolled eating (OR = 1.87; 95%CI = 1.34–2.60), self-perceived fatness (OR = 1.69; 95%CI = 1.28–2.23), and possible eating disorder (OR = 2.11; 95%CI = 1.67–2.67) were associated with significantly higher odds for physical multimorbidity, but self-sick for feeling full and food dominance were not (Table 3). Mediation analysis showed that anxiety disorder explained the largest proportion of the association between possible eating disorder and physical multimorbidity (mediated percentage 26.3%), followed by insomnia (21.8%), perceived stress (13.4%), depression

(13.1%), obesity (13.0%), and alcohol dependence (4.3%) (Table 4). These factors collectively explained 57.1% of the association.

## Discussion

### Main findings

In the present study including a large representative sample of adults aged ≥ 16 years from the UK, possible eating disorder was significantly associated with a more than twofold increased odds for physical multimorbidity, while individual eating disorder symptoms of weight loss, uncontrolled eating, and self-perceived fatness were significantly associated with 1.64–1.87 times higher odds for physical multimorbidity. The factors which were most important in the association between possible eating disorder and physical multimorbidity were anxiety disorder and insomnia, which explained more than 20% of the association. Factors such as perceived stress, depression, obesity, and alcohol dependence also explained this association but to a lesser extent. To the best of the authors' knowledge, this is the first study to investigate the association between eating disorder symptoms or possible eating disorder and physical multimorbidity, and also the first to quantify the extent to which this association may be explained by a variety of factors.

**Table 4** Mediators in the association between possible eating disorder and physical multimorbidity (outcome)

| Mediator | Effect | OR [95%CI] | *p*-value | Mediated % |
|---|---|---|---|---|
| Insomnia | Total | 2.12 [1.66,2.70] | <0.001 | 21.8 |
| | Direct | 1.80 [1.41,2.30] | <0.001 | |
| | Indirect | 1.18 [1.12,1.24] | <0.001 | |
| Depression | Total | 2.11 [1.66,2.68] | <0.001 | 13.1 |
| | Direct | 1.92 [1.51,2.44] | <0.001 | |
| | Indirect | 1.10 [1.06,1.15] | <0.001 | |
| Anxiety disorder | Total | 2.11 [1.66,2.69] | <0.001 | 26.3 |
| | Direct | 1.74 [1.36,2.22] | <0.001 | |
| | Indirect | 1.22 [1.15,1.29] | <0.001 | |
| Alcohol dependence | Total | 2.11 [1.67,2.66] | <0.001 | 4.3 |
| | Direct | 2.04 [1.62,2.58] | <0.001 | |
| | Indirect | 1.03 [1.00,1.06] | 0.023 | |
| Lifetime smoking | Total | 2.11 [1.67,2.67] | <0.001 | NA |
| | Direct | 2.09 [1.66,2.64] | <0.001 | |
| | Indirect | 1.01 [1.00,1.02] | 0.122 | |
| Perceived stress | Total | 2.11 [1.66,2.68] | <0.001 | 13.4 |
| | Direct | 1.91 [1.50,2.43] | <0.001 | |
| | Indirect | 1.10 [1.06,1.15] | <0.001 | |
| Obesity | Total | 2.15 [1.68,2.76] | <0.001 | 13.0 |
| | Direct | 1.95 [1.52,2.50] | <0.001 | |
| | Indirect | 1.11 [1.06,1.15] | <0.001 | |
| Underweight | Total | 2.14 [1.68,2.72] | <0.001 | NA |
| | Direct | 2.14 [1.68,2.72] | <0.001 | |
| | Indirect | 1.00 [0.99,1.01] | 0.621 | |
| All mediators | Total | 2.19 [1.68,2.84] | <0.001 | 57.1 |
| | Direct | 1.40 [1.07,1.83] | 0.014 | |
| | Indirect | 1.56 [1.42,1.72] | <0.001 | |

*OR* odds ratio, *CI* confidence interval

Models are adjusted for age, sex, education, and ethnicity

Possible eating disorder referred to SCOFF screen-positive

Mediated percentage was only calculated in the presence of a significant indirect effect

The mediators were included individually in the model, except for the model which included all mediators simultaneously

## Interpretation of the findings

There are several plausible pathways that likely explain the relationship between possible eating disorder and physical multimorbidity. In our study, anxiety disorder, insomnia, perceived stress, depression, obesity, and alcohol dependence were identified as potential influential factors in this association, with anxiety disorder and insomnia being particularly important. Literature suggests that eating disorders and anxiety disorders frequently co-occur, and it is likely that the relationship is bi-directional [34]. Common neurobiological, genetic, and psychological elements may be implicated in this co-existence [34]. Furthermore, eating disorders may lead to anxiety, perceived stress, and depression by, for example, body dissatisfaction, low self-esteem and uncontrollability of the situation [54]. These psychological factors, in turn, may increase risk for physical multimorbidity via elevated inflammation and immune dysregulation [55]. In terms of insomnia, eating disorders may increase risk for insomnia due to malnutrition and orexin. Indeed, orexin, a neuropeptide released from the hypothalamus, is involved in the regulation of both sleep–wake and appetite; increased orexin signaling promotes greater wakefulness and feeding. It has been posited that levels of orexin are increased during the hunger state to promote wakefulness and incite the body to search for food [56]. In turn, insomnia may increase risk for multimorbidity through dysfunction of the inflammatory system [57].

Obesity was another important influential factor identified in the present study. Some eating disorders, such as binge eating disorder, likely cause obesity owing to large consumptions of high energy dense foods, and obesity is likely to be a key risk factor for multimorbidity by increasing risk for hypertension, unfavorable cholesterol profile, dysfunction in the regulation of blood sugar levels, and dysfunction of the inflammatory system [58]. Finally, alcohol dependence also explained a small proportion of the association between eating disorders and physical multimorbidity. Those with eating disorders may turn to alcohol as a coping mechanism, thereby making them susceptible to alcohol dependence [35]. In turn, alcohol dependence is known to increase risk for a variety of physical diseases including cardiovascular disease, liver disease, and malignant neoplasms [59].

It is also important to note that all the influential factors included in our study collectively explained less than 60% of the association between possible eating disorder and physical multimorbidity. It is possible that there are unmeasured mechanisms such as inflammation [16], change in bone structure [24], and malnutrition.

Interestingly, weight loss, uncontrolled eating, and self-perceived fatness were associated with higher odds for physical multimorbidity but self-sick for feeling full and food dominance were not. It is likely that extreme weight loss and uncontrolled eating are associated with physical multimorbidity as both likely promote inflammatory dysregulation, and perceived fatness likely promotes physical multimorbidity via stress, anxiety, and depression. In the case of self-sick and food dominance, it is possible that these two constructs alone have little impact on physiological and psychological mechanisms that drive chronic disease and physical multimorbidity.

## Strengths and limitations

The analysis of a large representative sample of English adults and validated measures are clear strengths of the

present study. However, findings must be interpreted in light of the study's limitations. First, the study was cross-sectional in nature, and thus, the direction of the association cannot be established. For example, weight loss can also be the consequence of the physical condition (e.g., cancer). Second, the variables included in the study were based on self-report, potentially introducing recall and social desirability bias into the findings. Third, there was no data on physical activity in the dataset despite this factor being associated with both eating disorder and multimorbidity. Thus, we were unable to examine the influence of this factor in the association between eating disorder and multimorbidity. Furthermore, although the SCOFF questionnaire does have favorable validity, it is not a diagnostic tool. Thus, future research should aim to use clinician diagnosed eating disorders to confirm these findings. Also, we used BMI to determine weight category. However, BMI may not be the optimal measure to employ to measure body mass since it does not distinguish between fat mass and fat-free mass, and thus, may categorize an individual with a high level of fat-free mass as overweight or obese. Different measures of adiposity (e.g., waist circumference, bioimpedance) may yield differing mediating effects. Next, while we did have data on underweight which could be considered a proxy of malnutrition, we did not have data on malnutrition per se, despite the fact that eating disorders are associated with a high risk for malnutrition, and malnutrition can increase risk for multiple physical diseases [60, 61]. For example, pathophysiologic changes of the esophagus, stomach, and intestines develop with malnutrition, which can lead to gastrointestinal conditions such as celiac disease and inflammatory bowel disease [62]. Finally, mediation and confounding are identical from a statistical point of view, and can be distinguished only on conceptual grounds [63]. Thus, although the influential variables that were assessed in this study could be conceptualized as mediators, the mediated percentage estimated for each potential influential factor in our study could also be due to confounding.

### Clinical implications and areas for future research

The results of our study suggest that people with possible eating disorder may be at high risk for physical multimorbidity. Clinicians should be aware of this so that people with eating disorders can be screened for physical conditions, and have them treated, or vice versa. Furthermore, our study results suggest that addressing the identified potential mediators in the present study among those with eating disorders may help in the prevention of physical multimorbidity. Indeed, a well-established and lasting treatment for depression, anxiety, alcohol dependence, perceived stress, and sleep problems is cognitive-behavioral therapy, which focuses on identifying, understanding, and changing thinking and behavior patterns [64–67]. Importantly, cognitive-behavioral therapy can also be utilized as a treatment approach for eating disorders per se [68]. Interestingly, in the present study, even individual eating disorder symptoms (i.e., weight loss, uncontrolled eating, self-perceived fatness) were associated with physical multimorbidity. Thus, these findings suggest that the detection of individual symptoms alone could identify people at high risk for multimorbidity. Finally, studies of longitudinal design on eating disorders and physical multimorbidity are necessary to provide a better understanding of temporal associations, causality, and the underlying mechanisms.

## Conclusion

In this large sample of UK adults, it was observed that possible eating disorder was significantly associated with physical multimorbidity and that this association was partly explained by anxiety disorder, insomnia, perceived stress, depression, obesity, and alcohol dependence. While temporal associations and causality could not be assessed in our cross-sectional study, the mere co-existence of possible eating disorder and physical multimorbidity could complicate the management of these conditions, while they can also mutually influence each other and lead to worse clinical outcomes. Future longitudinal studies are warranted to understand potential causality and the underlying mechanisms, and also whether addressing the identified potential mediators in people with eating disorders can lead to a lower risk for physical multimorbidity Table 5.

## Appendix

See Table 5

**Table 5** Association between individual eating disorder symptoms and individual physical conditions (outcome) estimated by multivariable logistic regression

| Physical condition | Weight loss | Self-sick for feeling full | Uncontrolled eating | Self-perceived fatness | Food dominance |
|---|---|---|---|---|---|
| Allergy | 1.05 | 0.57 | 1.45 | 0.80 | 0.99 |
| | [0.80,1.37] | [0.31,1.04] | [0.98,2.15] | [0.55,1.17] | [0.57,1.72] |
| Arthritis | 1.22 | 1.23 | 1.46 | 0.96 | 1.79* |
| | [0.96,1.57] | [0.57,2.66] | [0.95,2.26] | [0.62,1.49] | [1.04,3.08] |
| Asthma | 1.34 | 0.90 | 1.85* | 1.57* | 1.18 |
| | [1.00,1.79] | [0.54,1.49] | [1.14,3.01] | [1.07,2.31] | [0.70,1.97] |
| Bladder problems/incontinence | 1.42 | 1.54 | 3.06** | 0.78 | 1.30 |
| | [0.91,2.19] | [0.62,3.84] | [1.34,6.98] | [0.38,1.62] | [0.47,3.56] |
| Bone, back, joint or muscle problems | 1.14 | 0.92 | 1.19 | 1.13 | 0.85 |
| | [0.93,1.40] | [0.60,1.40] | [0.91,1.57] | [0.84,1.51] | [0.56,1.28] |
| Bowel/colon problems | 2.31*** | 1.18 | 1.79* | 1.65* | 1.11 |
| | [1.75,3.05] | [0.52,2.69] | [1.03,3.09] | [1.03,2.64] | [0.59,2.09] |
| Bronchitis/emphysema | 1.92 | 1.41 | 0.98 | 1.69 | 1.95 |
| | [0.87,4.27] | [0.43,4.61] | [0.25,3.84] | [0.38,7.49] | [0.73,5.22] |
| Cancer | 1.32* | 1.22 | 1.05 | 1.23 | 0.83 |
| | [1.05,1.66] | [0.74,2.01] | [0.69,1.61] | [0.88,1.72] | [0.44,1.54] |
| Cataracts/eyesight problems | 1.58* | 1.02 | 0.80 | 1.11 | 1.92 |
| | [1.10,2.27] | [0.38,2.75] | [0.41,1.56] | [0.55,2.23] | [0.95,3.90] |
| Diabetes | 1.10 | 1.38 | 1.87* | 1.05 | 1.87 |
| | [0.78,1.54] | [0.66,2.87] | [1.02,3.42] | [0.53,2.07] | [0.98,3.57] |
| Ear/hearing problems | 1.60 | 1.49 | 1.50 | 1.54 | 1.38 |
| | [0.94,2.70] | [0.50,4.44] | [0.71,3.15] | [0.68,3.49] | [0.45,4.24] |
| Epilepsy/fits | 1.19 | 1.89 | 1.17 | 1.24 | 0.95 |
| | [0.50,2.81] | [0.69,5.16] | [0.36,3.74] | [0.42,3.60] | [0.24,3.83] |
| Heart attack/angina | 2.37*** | 0.50 | 0.70 | 0.76 | 1.03 |
| | [1.44,3.88] | [0.11,2.19] | [0.33,1.48] | [0.28,2.03] | [0.34,3.12] |
| High blood pressure | 1.34* | 1.47 | 1.50* | 1.10 | 1.42 |
| | [1.05,1.73] | [0.82,2.64] | [1.06,2.11] | [0.78,1.55] | [0.84,2.39] |
| Infectious disease | 2.31 | 1.34 | 0.48 | 1.76 | 0.38 |
| | [0.97,5.50] | [0.36,4.92] | [0.09,2.49] | [0.58,5.30] | [0.09,1.52] |
| Liver problems | 2.95** | 1.55 | 0.98 | 0.61 | 4.88* |
| | [1.38,6.28] | [0.33,7.16] | [0.49,1.94] | [0.17,2.20] | [1.40,17.06] |
| Migraine or frequent headaches | 1.36* | 1.04 | 1.32 | 1.51* | 0.95 |
| | [1.03,1.79] | [0.57,1.90] | [0.85,2.05] | [1.00,2.28] | [0.59,1.51] |
| Skin problems | 1.01 | 0.69 | 1.39 | 1.13 | 0.85 |
| | [0.78,1.31] | [0.40,1.22] | [0.87,2.21] | [0.76,1.68] | [0.53,1.39] |
| Stomach ulcer or other digestive problems | 1.75** | 1.48 | 1.53 | 1.31 | 1.61 |
| | [1.26,2.45] | [0.74,2.98] | [0.81,2.86] | [0.80,2.15] | [0.86,3.01] |
| Stroke | 1.65 | 1.00 | 0.12 | 3.88* | 0.80 |
| | [0.52,5.22] | [1.00,1.00] | [0.01,1.53] | [1.08,13.86] | [0.02,30.48] |

Data are odds ratio [95% confidence interval]

Models are adjusted for age, sex, education, ethnicity, and presence of $\geq 2$ eating disorder symptoms

The definitions of eating disorder symptoms were: weight loss (had lost more than 6.35 kg in 3 months); self-sick for feeling full (had made him/herself be sick because he/she felt uncomfortably full); uncontrolled eating (worried he/she had lost control over how much he/she eats); self-perceived fatness (believed to be fat when others said that he/she was too thin); food dominance (thought that food dominated his/her life);

*$p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

## Declarations

**Competing interests** The authors declare no competing interests.

## References

1. NICE | The National Institute for Health and Care Excellence (2016) Multimorbidity: clinical assessment and management. https://www.nice.org.uk/guidance/ng56/chapter/recommendations#multimorbidity. Accessed 13 August 2023
2. Fortin M, Lapointe L, Hudon C et al (2004) Multimorbidity and quality of life in primary care: a systematic review. Health Qual Life Outcomes 2:1–12
3. Kim J, Keshavjee S, Atun R (2020) Trends, patterns and health consequences of multimorbidity among South Korea adults: analysis of nationally representative survey data 2007–2016. J Glob Health 10.
4. Gallo JJ, Hwang S, Joo JH et al (2016) Multimorbidity, depression, and mortality in primary care: randomized clinical trial of an evidence-based depression care management program on mortality risk. J Gen Intern Med 31:380–386
5. Soley-Bori M, Ashworth M, Bisquera A et al (2021) Impact of multimorbidity on healthcare costs and utilisation: a systematic review of the UK literature. Br J Gen Pract 71:e39–e46
6. Ho IS-S, Azcoaga-Lorenzo A, Akbari A et al (2022) Variation in the estimated prevalence of multimorbidity: systematic review and meta-analysis of 193 international studies. BMJ Open 12:e057017
7. National Institute for Health Research | NIHR (2018) Multi-morbidity predicted to increase in the UK over the next 20 years. https://evidence.nihr.ac.uk/alert/multi-morbidity-predicted-to-increase-in-the-uk-over-the-next-20-years/
8. Solmi M, Veronese N, Correll CU et al (2016) Bone mineral density, osteoporosis, and fractures among people with eating disorders: a systematic review and meta-analysis. Acta Psychiatr Scand 133:341–351
9. Cavoli GL, Mulè G, Rotolo U (2011) Renal involvement in psychological eating disorders. Nephron Clin Pract 119:c338–c341
10. American Psychiatric Association (2013) Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub
11. World health organization (2023) International Statistical Classification of Diseases and Related Health Problems (ICD). https://www.who.int/standards/classifications/classification-of-diseases. Accessed 13 August 2023
12. Sardar MR, Greway A, DeAngelis M et al (2015) Cardiovascular impact of eating disorders in adults: a single center experience and literature review. Hear Views Off J Gulf Hear Assoc 16:88
13. Priory (2023) Eating Disorder Statistics. https://www.priorygroup.com/eating-disorders/eating-disorder-statistics. Accessed 13 August 2023
14. López-Gil JF, García-Hermoso A, Smith L et al (2023) Global proportion of disordered eating in children and adolescents: a systematic review and meta-analysis. JAMA Pediatr 177(4):363–372
15. Trott M, Johnstone J, Pardhan S et al (2021) Changes in body dysmorphic disorder, eating disorder, and exercise addiction symptomology during the COVID-19 pandemic: a longitudinal study of 319 health club users. Psychiatry Res 298:113831
16. Dargél AA, Godin O, Kapczinski F et al (2015) C-reactive protein alterations in bipolar disorder: a meta-analysis. J Clin Psychiatry 76:3919
17. Succurro E, Segura-Garcia C, Ruffo M, et al (2015) Obese patients with a binge eating disorder have an unfavorable metabolic and inflammatory profile. Medicine (Baltimore) 94
18. Espinoza-Salinas A, Bobadilla-Olivares M, Millaqueo C et al (2022) Efecto agudo de ejercicio isométrico sobre parámetros autonómicos en adultos sedentarios obesos y con sobrepeso. Sport TK-Revista Euroam Ciencias del Deport 11:5
19. Bautista LE, López-Jaramillo P, Vera LM et al (2001) Is C-reactive protein an independent risk factor for essential hypertension? J Hypertens 19:857–861
20. Mendall MA, Patel P, Ballam L et al (1996) C reactive protein and its relation to cardiovascular risk factors: a population based cross sectional study. BMJ 312:1061–1065
21. Nielen MMJ, Van Schaardenburg D, Reesink HW et al (2004) Increased levels of C-reactive protein in serum from blood donors before the onset of rheumatoid arthritis. Arthritis Rheum Off J Am Coll Rheumatol 50:2423–2427
22. Lochhead P, Khalili H, Ananthakrishnan AN et al (2016) Association between circulating levels of C-reactive protein and interleukin-6 and risk of inflammatory bowel disease. Clin Gastroenterol Hepatol 14:818–824
23. Garrafa E, Casnici N, Squazzoni F et al (2017) C-reactive protein, lipoprotein (a) and cystatin C levels increase with multimorbidity in older persons. Eur J Intern Med 42:e25–e26
24. Meczekalski B, Podfigurna-Stopa A, Katulski K (2013) Long-term consequences of anorexia nervosa. Maturitas 75:215–220
25. Stamenkovic BN, Rancic NK, Bojanovic MR et al (2022) Is osteoarthritis always associated with low bone mineral density in elderly patients? Medicina (B Aires) 58:1207
26. Yale Medicine (2023) Low Bone Density. https://www.yalemedicine.org/conditions/bone-density-test. Accessed 13 August 2023
27. Esparza Montero MÁ (2021) Influence of the strength of the ankle plantar flexors on dynamic balance in 55–65-year-old women. Atena J Public Heal 3:3

28. Kenny TE, Van Wijk M, Singleton C, Carter JC (2018) An examination of the relationship between binge eating disorder and insomnia symptoms. Eur Eat Disord Rev 26:186–196

29. Araujo DMR, da Santos GF, Nardi AE (2010) Binge eating disorder and depression: a systematic review. World J Biol psychiatry 11:199–207

30. Keetile M, Navaneetham K, Letamo G (2020) Prevalence and correlates of multimorbidity among adults in Botswana: a cross-sectional study. PLoS ONE 15:e0239334

31. Wikström K, Lindström J, Harald K et al (2015) Clinical and lifestyle-related risk factors for incident multimorbidity: 10-year follow-up of Finnish population-based cohorts 1982–2012. Eur J Intern Med 26:211–216

32. Vancampfort D, Koyanagi A, Ward PB et al (2017) Perceived stress and its relationship with chronic medical conditions and multimorbidity among 229,293 community-dwelling adults in 44 low-and middle-income countries. Am J Epidemiol 186:979–989

33. López Sánchez GF, Vigueras Hernández MR, Lucas Casas P et al (2022) Impact of physical activity, BMI and sociodemographic and lifestyle factors on the risk of diabetes in 9511 Ghanaian adults. Sport TK Rev Euroam Ciencias del Deport 11:15

34. Swinbourne JM, Touyz SW (2007) The co-morbidity of eating disorders and anxiety disorders: a review. Eur Eat Disord Rev Prof J Eat Disord Assoc 15:253–274

35. Gadalla T, Piran N (2007) Co-occurrence of eating disorders and alcohol use disorders in women: a meta analysis. Arch Womens Ment Health 10:133–140

36. Anzengruber D, Klump KL, Thornton L et al (2006) Smoking in eating disorders. Eat Behav 7:291–299

37. Naish KR, Laliberte M, MacKillop J, Balodis IM (2019) Systematic review of the effects of acute stress in binge eating disorder. Eur J Neurosci 50:2415–2429

38. Yanovski SZ (2003) Binge eating disorder and obesity in 2003: could treating an eating disorder have a positive effect on the obesity epidemic? Int J Eat Disord 34:S117–S120

39. Sindi S, Pérez LM, Vetrano DL et al (2020) Sleep disturbances and the speed of multimorbidity development in old age: results from a longitudinal population-based study. BMC Med 18:1–10

40. Birk JL, Kronish IM, Moise N et al (2019) Depression and multimorbidity: considering temporal characteristics of the associations between depression and multiple chronic diseases. Heal Psychol 38:802

41. Felez-Nobrega M, Haro JM, Koyanagi A (2022) Multimorbidity, depression with anxiety symptoms, and decrements in health in 47 low-and middle-income countries. J Affect Disord 317:176–184

42. Jenkins R, Meltzer H, Bebbington P et al (2009) The British Mental Health Survey Programme: achievements and latest findings. Soc Psychiatry Psychiatr Epidemiol 44:899–904

43. Digital NHS (2009) Adult Psychiatric Morbidity in England - 2007, Results of a household survey. https://digital.nhs.uk/data-and-information/publications/statistical/adult-psychiatric-morbidity-survey/adult-psychiatric-morbidity-in-england-2007-results-of-a-household-survey

44. Garin N, Koyanagi A, Chatterji S et al (2016) Global multimorbidity patterns: a cross-sectional, population-based, multi-country study. Journals Gerontol Ser A Biomed Sci Med Sci 71:205–214

45. Morgan JF, Reid F, Lacey JH (1999) The SCOFF questionnaire: assessment of a new screening tool for eating disorders. BMJ 319:1467–1468

46. Garcia-Campayo J, Sanz-Carrillo C, Ibañez JA et al (2005) Validation of the Spanish version of the SCOFF questionnaire for the screening of eating disorders in primary care. J Psychosom Res 59:51–55

47. Luck AJ, MorganLuck JF, Reid F et al (2002) The SCOFF questionnaire and clinical interview for eating disorders in general practice: comparative study. BMJ 325:755–756

48. Botella J, Sepúlveda AR, Huang H, Gambara H (2013) A meta-analysis of the diagnostic accuracy of the SCOFF. Span J Psychol 16:E92

49. Freeman D, Brugha T, Meltzer H et al (2010) Persecutory ideation and insomnia: findings from the second British National Survey of Psychiatric Morbidity. J Psychiatr Res 44:1021–1026

50. Lewis G, Pelosi AJ, Araya R, Dunn G (1992) Measuring psychiatric disorder in the community: a standardized assessment for use by lay interviewers. Psychol Med 22:465–486

51. Saunders JB, Aasland OG, Babor TF et al (1993) Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption-II. Addiction 88:791–804

52. Stockwell TIM, Sitharthan T, McGRATH D, Lang E (1994) The measurement of alcohol dependence and impaired control in community samples. Addiction 89:167–184

53. Breen R, Karlson KB, Holm A (2013) Total, direct, and indirect effects in logit and probit models. Sociol Methods Res 42:164–191

54. Laporta-Herrero I, Jáuregui-Lobera I, Barajas-Iglesias B, Santed-Germán MÁ (2018) Body dissatisfaction in adolescents with eating disorders. Eat Weight Disord Anorexia, Bulim Obes 23:339–347

55. Vogelzangs N, Beekman ATF, De Jonge P, Penninx B (2013) Anxiety disorders and inflammation in a large adult cohort. Transl Psychiatry 3:e249

56. Allison KC, Spaeth A, Hopkins CM (2016) Sleep and eating disorders. Curr Psychiatry Rep 18:1–8

57. Irwin MR, Wang M, Ribeiro D et al (2008) Sleep loss activates cellular inflammatory signaling. Biol Psychiatry 64:538–540

58. Harvard T.H. Chan School of Public Health (2023) Obesity Prevention Source. https://www.hsph.harvard.edu/obesity-prevention-source/obesity-consequences/health-effects/. Accessed 13 August 2023

59. Cargiulo T (2007) Understanding the health impact of alcohol dependence. Am J Heal Pharm 64:S5–S11

60. Laraia BA (2013) Food insecurity and chronic disease. Adv Nutr 4:203–212

61. Fares K, Barada D, Hoteit M, Abou Haidar M (2020) Prevalence and correlates of food insecurity among Lebanese University students of Hadath Campus. Atena J Public Heal 2:5

62. Gibson D, Watters A, Mehler PS (2021) The intersect of gastrointestinal symptoms and malnutrition associated with anorexia nervosa and avoidant/restrictive food intake disorder: functional or pathophysiologic?—A systematic review. Int J Eat Disord 54:1019–1054

63. MacKinnon DP, Krull JL, Lockwood CM (2000) Equivalence of the mediation, confounding and suppression effect. Prev Sci 1:173–181

64. Anxiety and Depression Association of America (ADAA) (2020) Eating Disorders. https://adaa.org/understanding-anxiety/related-illnesses/eating-disorders

65. Gautam M, Tripathi A, Deshmukh D, Gaur M (2020) Cognitive behavioral therapy for depression. Indian J Psychiatry 62:S223

66. Epstein EE, McCrady BS, Hallgren KA et al (2018) Individual versus group female-specific cognitive behavior therapy for alcohol use disorder. J Subst Abuse Treat 88:27–43

67. Sarid O, Berger R, Segal-Engelchin D (2010) The impact of cognitive behavioral interventions on SOC, perceived stress and mood states of nurses. Procedia-Social Behav Sci 2:928–932

68. Fairburn CG (2008) Cognitive behavior therapy and eating disorders. Guilford Press