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Psychological Pathways Explaining the Prospective Association Between Obesity and Physiological Dysregulation

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Objective: Obesity is associated with a range of negative psychological conditions that may also affect physiological health. Across two studies, we tested whether a range of psychological measures explain why obesity is prospectively associated with physiological dysregulation, measured via clinical indicators of cardiovascular, immune system, and metabolic function. ***Method:*** We used comparable 4-year follow-up representative longitudinal data of U.K. and U.S. older adults (≥50 years) from the English Longitudinal Study of Ageing (2008/2009–2012/2013; Study 1; $n = 6{,}250$) and the Health and Retirement Study (2008/2010–2012/2014; Study 2; $n = 9{,}664$). A diverse range of psychological measures (e.g., depressive symptoms, life satisfaction, weight stigma, positive affect) were tested as candidate mediators in Studies 1 ($n = 14$) and 2 ($n = 21$). ***Results:*** Obesity predicted physiological dysregulation at follow-up across both studies. In Study 1, only weight stigma (measured between baseline and follow-up) explained 37% of the association between obesity and physiological dysregulation. In Study 2, only changes in weight stigma from baseline to follow-up (not baseline weight stigma) explained 13% of the effect of obesity on future physiological dysregulation. Mediation by weight stigma in both studies was partially attenuated when changes in body mass index from baseline to follow-up were controlled for. No other psychological measures explained the association between obesity and physiological dysregulation in either study. ***Conclusions:*** The prospective association between obesity and physiological dysregulation was largely not explained by psychological factors. However, experiencing weight stigma is associated with increased weight gain and this process may explain obesity-related declines in physiological health.

Public Significance Statement
These findings indicated that reducing chronic sources of psychological distress in people living with obesity (e.g., weight stigma) may be important to reducing obesity-related physiological health impairments.

Keywords: obesity, psychological well-being, weight discrimination, physiological health, biomarkers

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Obesity is associated with an increased risk of adverse health outcomes, including diabetes mellitus, musculoskeletal disorders, cardiovascular diseases, chronic kidney disease, cancer, and premature mortality (Abdelaal et al., 2017; Dai et al., 2020; GBD Obesity Collaborators, 2017). Obesity is thought to harm health by affecting a range of physiological processes that result in an increased risk of hypertension (Kotsis et al., 2010; Vaneˇcˇková et al., 2014), raised heart rates (Rossi et al., 2015), elevated C-reactive protein (CRP) levels indicative of low-grade systemic inflammation (Choi et al., 2013), dyslipidemia (Klop et al., 2013; Vekic et al., 2019), glucose intolerance, and insulin resistance (Kahn et al., 2006; Martyn et al., 2008). The pathways by which obesity is associated with worsening physiological functioning and biological ill health have traditionally been explained by the strain that adiposity (i.e., excess fat mass) places on the body (Després, 2012; Powell-Wiley et al., 2021; Tchernof & Després, 2013). More recently, it has been proposed that the damaging physiological effects associated with obesity may be in part caused by the psychological experience of obesity (Puhl et al., 2020; Tomiyama, 2014). Specifically, the pervasive weight stigma and negative emotional states that frequently accompany obesity have been proposed to lead to the stress-related activation and dysregulation of multiple physiological systems (Tomiyama, 2019). This possibility is underpinned by extensive evidence but has not been tested comprehensively to date.

Many people living with obesity experience weight stigma (Jackson, Steptoe, et al., 2015; Puhl et al., 2008). Although current literature on obesity medicine does not typically consider weight stigma as a chronic source of stress (Kumar et al., 2022; Siddiqui et al., 2022), it is plausible that stress from weight stigma drives negative health outcomes (Tomiyama, 2014). Weight stigma is thought to have damaging physiological effects because it elicits a chronic social-evaluative threat and feelings of stress and negative emotion (Emmer et al., 2020; Jackson, Beeken, & Wardle, 2015; Robinson et al., 2017). Experiencing weight stigma can activate the hypothalamic–pituitary–adrenal (HPA) axis, autonomic system, and cortisol secretion (Himmelstein et al., 2015; Schvey et al., 2014; Tomiyama et al., 2018), as well as systemic inflammation, as gauged by CRP levels (Sutin et al., 2014). In a cross-sectional study, the association between central adiposity (i.e., waist-to-hip ratio) and glycated hemoglobin (HbA1c) levels was stronger among those who experienced weight stigma (Tsenkova et al., 2011). Similarly, individuals with obesity who report weight bias internalization have greater odds of metabolic syndrome and high triglycerides (Pearl et al., 2017). Those with obesity are also at increased risk of depression (Frank et al., 2022; Luppino et al., 2010; Xu et al., 2011), anxiety disorders (Burke & Storch, 2015; Gariepy et al., 2010), negative affect (Pasco et al., 2013), loneliness (Hajek et al., 2021), hopelessness (Murphy et al., 2009), cynical hostility (Tindle et al., 2018), low positive well-being, such as low quality of life (Jackson, Beeken, & Wardle, 2015; Stephenson et al., 2021), life satisfaction (Ball et al., 2004; Katsaiti, 2012), happiness (Ul-Haq et al., 2014), and positive affect (Jorm et al., 2003). An integrative umbrella review of meta-analyses also concluded heavier body weight is associated with poor mental health (Robinson et al., 2020).

The stress-related negative emotional states associated with obesity have been found to have detrimental biological effects on the body (O'Connor et al., 2021). Negative emotions can increase the risk of inflammation which is thought to accelerate biological aging and disease risk (Renna, 2021; Segerstrom & Miller, 2004; Stellar et al., 2015). Likewise, higher depressive symptoms and/or perceived stress are associated with worse physiological health, including cortisol secretion (Chida & Steptoe, 2009), elevated blood pressure (Gasperin et al., 2009), CRP levels (Gowey et al., 2019), triglycerides (Bove et al., 2010), lower high-density lipoprotein (HDL; Gowey et al., 2019), and dysregulated glucose metabolism (Boyle et al., 2007). Increased pessimism was found to independently predict fasting insulin and insulin resistance in a cross-sectional study of postmenopausal women in the United States (Tindle et al., 2018). Moreover, a longitudinal study has shown that perceived stress mediated the negative effect of chronic stressors on a measure of "physiological dysregulation," an index derived from a range of clinical indicators reflecting cardiovascular, immune system, neuroendocrine function, and metabolic functioning (Glei et al., 2007).

The allostatic-load framework posits that such dysregulation measures can capture the physiological toll of the body's adaptation to chronic stressors, which over time can result in cumulative "wear and tear" across multiple physiological systems and ultimately lead to deterioration in health (Guidi et al., 2021; Wiley et al., 2016). Experiencing weight stigma has been shown to prospectively predict high levels of allostatic load 10 years later in a sample of U.S. adults (Vadiveloo & Mattei, 2017). Building on this work, a study of older U.K. adults showed that self-reported weight stigma (discrimination) could partly explain the prospective 4-year association between obesity and physiological dysregulation, measured as a combination of biomarkers related to cardiovascular, inflammatory, and metabolic dysregulation, indicative of allostatic load (Daly et al., 2019). However, while weight stigma predicted worsening physiological health in this study, the broader role of other psychological measured associated with obesity in affecting physiological function over time was not examined.

In the present research, we therefore aimed to examine the potential wider role of a diverse range of psychological factors in explaining obesity-driven declines in physiological function. To achieve these aims, we made use of two large-scale representative longitudinal cohorts of older U.K. (English Longitudinal Study of Ageing—ELSA) and U.S. (Health and Retirement Study—HRS) adults. To assess physiological function, we developed an index of physiological dysregulation as used in the previous research (Daly et al., 2019; Glei et al., 2007; Hampson et al., 2009; Juster et al., 2010). We used ELSA and HRS datasets because both cohorts collected physical, psychological, and biomarker data that allowed us to evaluate the role that a range of different psychological measures may have in explaining the longitudinal association between obesity and physiological dysregulation in older adults. Furthermore, older adults are at risk of worsening health, and composite measures of physiological dysregulation among the age populations of ELSA and HRS have been shown to be an indicator of preclinical disease (Arbeev et al., 2019) and predictive of subsequent mortality risk (Arbeev et al., 2020). Analyzing two different cohorts also enabled us to examine the cultural generalizability of the findings (United Kingdom vs. United States). We hypothesized that psychological factors associated with heavier body weight separately or together would in part explain why obesity is prospectively associated with physiological dysregulation.

Method

Participants

Study 1: English Longitudinal Study of Ageing

ELSA is a representative longitudinal study of adults ≥50 years living in private households in England. ELSA participants were recruited

in 2002 (Wave 1) from three waves of the Health Survey for England (HSE; 1998, 1999, 2001) and their health outcomes have been tracked biennially (Waves 2–10; 2004–2005 to 2021–2023). Further information on ELSA contents and methodology is available elsewhere (Rogers et al., 2017). For this study, we selected 4 years of ELSA records from Waves 4 (2008–2009) to 6 (2012–2013) where data on weight and height to calculate body mass index (BMI) were collected as part of the health and biomarker assessments (i.e., nurse data) and because a large range of psychological factors was measured at Wave 4 (Banks et al., 2021). Participants were retained for the analytical sample if their unique identity matched the health and biomarker assessments between the baseline and follow-up ($n = 6{,}250$). Ethics approval for each ELSA wave was obtained from different institutional review boards (IRBs; https://www.elsa-project.ac.uk/ethical-approval).

Study 2: Health and Retirement Study

In Study 2, we examined the generalizability of the findings from Study 1 among U.S. older adults. The methods and analysis approach for Study 2 were preregistered at https://doi.org/10.17605/OSF.IO/A6VWS. HRS is a nationally representative longitudinal survey of adults ≥50 years in the United States, recruited in 1992 and then biennially followed up. The content of HRS was expanded to include biomarker and psychological measures in 2006 (Wave 8) onwards through a mixed-mode design for follow-up. Half of the participants were assigned to the core interview only, and the other half participated in the enhanced face-to-face interview (EFTF), involving physical, biological, and psychological measures; this data collection strategy was then swapped between two half-sample groups in the next follow-up. Longitudinal data on the expanded content are available every wave (i.e., 2 years) on a representative half-sample and every 4 years at the individual level (Sonnega et al., 2014). Even though psychological measures were comprehensively assessed for the first time in 2006 (Wave 8), changes in the measurements of psychological factors have been consistently applied since 2008 (Wave 9). We combined samples from two waves where psychological measures were consistently collected using the same tools a baseline (Wave 9—2008 and Wave 10—2010) and measured changes in physiological dysregulation in a 4-year follow-up (Wave 11—2012 and Wave 12—2014). Similar to Study 1, participants were retained if their unique identity matched the biomarker assessments between baseline and follow-up ($n = 9{,}664$). Study 2 used harmonized datasets provided by the Research and Development (RAND) Corporation (https://hrsdata.isr.umich.edu/data-products/rand). University of Michigan's IRB provided ethics approval for all the HRS waves.

Independent Variable: Obesity

In Studies 1 and 2, participants' weight without shoes and in light clothing and standing height without shoes at baseline were objectively measured by research staff (see the online supplemental materials for full information). For both studies, BMI was calculated by dividing weight in kilogram (kg) by height in meters squared (m^2). Obesity was defined as BMI ≥ 30 kg/m^2.

Dependent Variable: Physiological Dysregulation

Following previous literature (Daly et al., 2017, 2019; Hampson et al., 2009), biomarkers related to cardiovascular, metabolic system functioning, and inflammation levels were used to generate a composite measure of physiological dysregulation indicative of allostatic load (Wiley et al., 2016). Cardiovascular measures were average between systolic and diastolic blood pressure, and resting pulse rate. Metabolic system functioning was assessed using the ratio of total blood cholesterol to HDL cholesterol, and glycated hemoglobin (HbA1c) levels. Inflammation was measured using CRP. The residualized change approach was used to separately develop an index of physiological dysregulation between the baseline and follow-up. The *z*-score method was used to assess physiological dysregulation levels (Daly et al., 2019; Hampson et al., 2009; Juster et al., 2010). All biomarkers were *z*-score standardized. Continuous summary scores of physiological dysregulations were calculated by averaging the computed *z*-scores if at least three-quarters of biomarkers were available (≥4 out of 5; e.g., as in Daly et al., 2019). The average *z*-score of physiological dysregulations at baseline and follow-up waves were restandardized to produce an index with a mean of 0 and a *SD* of 1.

Candidate Mediators: Psychological Measures

We selected all psychological measures collected in each cohort that we identified as being potentially associated with obesity and/or worsening physiological health (see the online supplemental materials for full information and Table A1 in the online supplemental materials for published evidence for associations with obesity and physiological/physical health). Table 1 presents all mediators examined in Studies 1 and 2. Fourteen psychological measures were tested as candidate mediators in Study 1. Most of them were from the same baseline wave as obesity status (i.e., Wave 4; 2008–2009), except for weight stigma, positive affect, and the big five personality traits (neuroticism, extraversion, conscientiousness, agreeableness, openness to experience) assessed in Wave 5 (2010–2011). In Study 2, we examined 21 psychological measures that were fitted from the baseline as obesity status (i.e., Waves 9 and 10; 2008 and 2010). Twelve psychological measures in HRS—depressive symptoms, life satisfaction, loneliness, social support, social strain, weight stigma, positive affect, big five personality traits—were also collected in ELSA and used in Study 1. Tables A2 and A3 in the online supplemental materials present the correlation between psychological measures in Studies 1 and 2, respectively.

Covariates

Covariates adjusted from the baseline wave in Study 1 included age (in years) and age squared (to account for a potential nonlinear association), sex (female; male), ethnicity (White; non-White), marital status (single; married or cohabitating; separated, divorced, or widowed), the completion of degree-level qualification (yes; no), participation in paid employment in the last week (yes; no), and quintiles of total household wealth (net nonpension wealth). Likewise, Study 2 also controlled the same number of covariates with most of the covariates being defined as the same as in Study 1, except for education defined as the number of years in education due to data completeness. In Study 2, total household wealth (net nonpension wealth) was calculated by RAND investigators (Bugliari et al., 2021) and was transformed into quintiles.

Mediation Analysis

We used the same mediation analyses for Studies 1 and 2. The mediation analysis examined whether having obesity (BMI ≥

Table 1
Psychological Measures Assessed as Candidate Mediators in Studies 1 and 2

Psychological measures	Measurement scales	Study 1 (ELSA)	Study 2 (HRS)
Depressive symptoms	Eight-item version of the Centre for Epidemiology Depression Scale (CED-S; *total score: 0–8*)	Wave 4	Waves 9 and 10
Enjoyment of life	Four items from the Control, Autonomy, Self-Realization, and Pleasure (CASP-19) quality of life questionnaire (*total score: 0–12*)	Wave 4	—
Eudemonic well-being	Fifteen items from the Control, Autonomy, Self-Realization, and Pleasure (CASP-19) quality of life questionnaire (*total score: 0–45*)	Wave 4	—
Life satisfaction	Five items from the Satisfaction with Life Scale (SWLS; *total score: 1–35*)	Wave 4	Waves 9 and 10
Loneliness	Three-item version of the UCLA Loneliness scale (*total score: 1–9*)	Wave 4	Waves 9 and 10
Social support	Three items on positive experience of social support (e.g., as in Khondoker et al., 2017; *average score: 1–4*)	Wave 4	Waves 9 and 10
Social strain	Three items on negative experience of social support (e.g., as in Khondoker et al., 2017; *average score: 1–4*)	Wave 4	Waves 9 and 10
Weight stigma	Perceived Everyday Experiences with Discrimination Scale (*weight stigma was treated as a dichotomous variable: yes vs. no*)	Wave 5	Waves 9 and 10
Positive affect	Thirteen items from the Positive and Negative Affect scale (PANAS-X; *total score: 1–65*)	Wave 5	Waves 9 and 10
Negative affect	Twelve items from the Positive and Negative Affect scale (PANAS-X; *total score: 1–60*)	—	Waves 9 and 10
Purpose in life	Purpose in life domain (seven items) from the Ryff Measures of Psychological Well-being (*total score: 1–42*)	—	Waves 9 and 10
Anxiety	Five items from the Beck Anxiety Inventory (BAI; *total score: 1–20*)	—	Waves 9 and 10
Hopelessness	Two items adapted from Beck et al. (1974) and two from Everson et al. (1997; *total score: 1–24*)	—	Waves 9 and 10
Optimism	Three items from the Life Orientation Test-Revised (LOT-R; *total score: 1–18*)	—	Waves 9 and 10
Pessimism	Three items from the Life Orientation Test-Revised (LOT-R; *total score: 1–18*)	—	Waves 9 and 10
Cynical hostility	Five items from the Cook-Medley Hostility Inventory (*total score: 1–30*)	—	Waves 9 and 10
Perceived constraint	Five items on perceived constraints (e.g., as in Infurna & Mayer, 2015; *total score: 1–30*)	—	Waves 9 and 10
Perceived mastery	Five items on perceived mastery (e.g., as in Infurna & Mayer, 2015; *total score: 1–30*)	—	Waves 9 and 10
Neuroticism	Four items from The Midlife Development Inventory (MIDI) Personality Scales (*total score: 1–16*)	Wave 5	Waves 9 and 10
Extraversion	Five items from The Midlife Development Inventory (MIDI) Personality Scales (*total score: 1–20*)	Wave 5	Waves 9 and 10
Conscientiousness	Five items from The Midlife Development Inventory (MIDI) Personality Scales (*total score: 1–20*)	Wave 5	Waves 9 and 10
Agreeableness	Five items from The Midlife Development Inventory (MIDI) Personality Scales (*total score: 1–20*)	Wave 5	Waves 9 and 10
Openness	Seven items from The Midlife Development Inventory (MIDI) Personality Scales (*total score: 1–28*)	Wave 5	Waves 9 and 10

Note. Study 1 (ELSA): baseline (Wave 4; 2008–2009); follow-up (Wave 6; 2012–2013); a wave between baseline and follow-up (Wave 5; 2010–2011). Study 2 (HRS): baseline (Waves 9 and 10; 2008 and 2010); follow-up (Waves 11 and 12; 2012 and 2014). ELSA = English Longitudinal Study of Ageing; HRS = Health and Retirement Study; UCLA = The University of California Los Angeles.

30 kg/m^2), relative to no obesity (BMI $<$ 30 kg/m^2) at baseline wave, had an indirect association with physiological dysregulation through any of the psychological measures. We first conducted single mediation models and if we found any evidence of mediation by more than one psychological measure ($p < .05$), measures were examined together in a multiple mediation model. To conduct single and multiple mediation models, we used "khb" command available in STATA (Kohler et al., 2011). All mediation models were adjusted for baseline physiological dysregulation and covariates. Findings from mediation model were presented as regression coefficients of direct, indirect, and total effects. The direct effect refers to the association between obesity status and physiological dysregulation while controlling for psychological measure(s), while the indirect effect represents this association through the hypothesized psychological measure(s). Direct and indirect effects together constitute the total effect. Based on "khb" command, for each hypothesized psychological measure, regression coefficients (point estimates) and 95% confidence intervals of direct, indirect, and total effects were calculated using the same sample size. We used inverse probability weighting (IPW) to address missing values and differences in characteristics between participants with vs. without longitudinal data (see the online supplemental materials for full information) .

Robustness Analyses

We tested whether the mediation by psychological variables was consistent for different classes of obesity (i.e., Class I [BMI $\geq$ 30–34.9] and Class II and III obesity [BMI $\geq$ 35]), relative to normal weight (BMI $<$ 25). We tested if the results were consistent using a different approach to handling missing data, multiple imputations by chained equations (MICE). In addition, mediation by psychological variables was also evaluated by comparing obesity and different obesity classes to overweight status (BMI $\geq$ 25–29.9). We also developed another physiological dysregulation measure by adding the number of biomarkers exceeding a given cutoff point (see the online supplemental materials).

In instances in which mediators were assessed more than one time from the baseline and up to follow-up, we explored whether changes in the mediator explained obesity-related changes in physiological dysregulation. In Study 1, psychological measures were repeatedly measured every 2 years, and we fitted changes (using difference scores) in psychological measures between Wave 4 (baseline) and Wave 5, and between Wave 4 (baseline) and Wave 6 (follow-up) in mediation models, except for weight stigma, positive affect, and personality traits available in Wave 5 only. In Study 2, psychological measures were available every 4 years, and difference scores of psychological variables between the baseline (Waves 9 and 10) and follow-up (Waves 11 and 12) were also examined in mediation models, except for anxiety and cynical hostility (only measured at baseline). For weight stigma in Study 2, four possible changes between waves were presented into two categories: (a) no stigma in both waves and stigma only at baseline wave (no increase in weight stigma); and (b) stigma only at follow-up wave and stigma in both waves (weight stigma increased or maintained).

Given that it is plausible that obesity and psychological measures may independently contribute to changes in physiological dysregulation (as opposed to psychological factors explaining the effects of obesity), we also ran linear regression models to identify whether any psychological measures separately or together predicted changes in physiological dysregulation (see the online supplemental materials). The level of significance was set at $p < .05$ for main mediation analyses and to correct for multiple comparisons, $p < .01$ was set for additional robustness analyses.

For Study 2 (HRS), some robustness analyses were requested by peer reviewers and therefore not preregistered, including mediation analyses of (a) the big five personality traits, (b) overweight status as the reference category, and (c) physiological dysregulation assessed as the numbers of biomarkers indicating unhealthy physiological conditions.

Results

Table 2 presents the baseline characteristics of the participants from ELSA (Study 1) and HRS (Study 2). Participants from both studies were comparable in terms of age and sex. Participants in HRS had a higher number of participants with obesity and that were non-White.

Study 1: English Longitudinal Study of Ageing

Table 3 shows findings from single mediation models of psychological measures in U.K. samples. Enjoyment of life, eudemonic well-being, and weight stigma were statistically significant mediators of the relationship between obesity and physiological dysregulation in single mediation models (Table 3). However, only weight stigma remained a statistically significant mediator when these three psychological measures were included in a multiple mediation model (Table 4), whereby obesity was associated with increased odds of reporting weight stigma which in turn predicted physiological dysregulation (mediating 31% of the association). Results were broadly consistent across all robustness analyses (i.e., only weight stigma as a significant mediator) when different classes of obesity (e.g., Classes II and III) relative to normal weight were tested (Table B1 in the online supplemental materials), missing values were addressed using MICE approach (Tables B2 and B3 in the online supplemental materials), and overweight status was used as the reference group (Table B4 in the online supplemental materials). However, no mediation by any psychological measures was observed when physiological dysregulation was assessed as the number of biomarkers (Table B5 in the online supplemental materials). There were also no significant mediators identified when changes in psychological measures between Waves 4 and 6 (Table B6 in the online supplemental materials) and Waves 4 and 5 (*findings are not presented*) were examined. Table B7 in the online supplemental materials shows the direct associations between each baseline psychological measure and physiological dysregulation at the follow-up wave, adjusting for obesity status, baseline physiological dysregulation, and covariates. Although depressive symptoms, enjoyment of life, eudemonic well-being, positive affect, and weight stigma were statistically significantly associated with physiological dysregulation when examined individually ($p < .01$; Table B7 in the online supplemental materials), when psychological measures were entered together into a model to predict physiological dysregulation, none were independently associated with changes in physiological dysregulation at $p < .01$, although positive affect and weight stigma remained significant at $p < .05$ (Table B8 in the online supplemental materials).

Table 2
Baseline Demographic Characteristics of the Participants in Studies 1 and 2

Variables	Study 1 (ELSA; $n = 6{,}250$)[a]		Study 2 (HRS; $n = 9{,}664$)[a]	
	M (*SD*)[b]	%[b]	*M* (*SD*)[b]	%[b]
Age (years)	64.61 (9.41)		64.26 (9.31)	
Female		53.41		54.52
White		96.45		84.85
BMI baseline (in kg/m^2)	28.31 (5.21)		29.91 (6.03)	
BMI < 25[c]		26.23		20.45
Overweight		42.83		36.47
Class I obese		20.63		25.40
Class II and III obese		10.30		17.68

Note. M = Mean; *SD* = standard deviation; ELSA = English Longitudinal Study of Ageing; HRS = Health and Retirement Study; BMI = body mass index.
[a] Maximum number of analytical samples (i.e., participants that had their unique identity matched biomarker assessments between baseline and follow-up waves). [b] Values were weighted using weights from biomarker assessments. [c] Including those with underweight (BMI < 18.5 kg/m^2); only 40 (0.83%) and 49 (0.50%) participants with underweight in ELSA and HRS, respectively.

Study 2: Health and Retirement Study

Analyses for Study 2 suggested no evidence of mediation by baseline psychological factors on the prospective association between obesity and physiological dysregulation (Table 5) and across all robustness analyses (Tables C1–C5 in the online supplemental materials). However, examining changes in candidate mediators between baseline and follow-up waves showed that weight stigma statistically significantly mediated the association between obesity and physiological dysregulation by 13.0% ($p < .01$; Table C6 in the online supplemental materials), whereby obesity was associated with an increased likelihood of experiencing weight stigma which in turn predicted physiological dysregulation. Mediation by changes in weight stigma was consistent when comparing Class II and III versus normal weight (by 16%) and overweight was used as the reference group (*findings are not presented*). Findings from regression analyses demonstrated that cynical hostility was associated with physiological dysregulation, but not independently in the presence of other psychological measures (Tables C7 and C8 in the online supplemental materials).

Exploratory Analyses on Weight Stigma and Weight Gain

Study 1 showed that weight stigma (measured between baseline and follow-up) was a strong mediator of the longitudinal association between obesity and physiological dysregulation in U.K. participants. In Study 2, changes in weight stigma between waves, but not weight stigma at baseline, partly mediated this association. To examine whether statistically significant mediation by weight stigma in both

Table 3
Single Mediation Models of Psychological Measures in Study 1

Mediation models (reference group: non-obesity [BMI < 30 kg/m^2])	Point estimates	95% CI	Effect ratio
Mediator: depressive symptoms ($n = 3,801$)			
Total effect	0.073*	[0.006, 0.139]	
Direct effect	0.069*	[0.003, 0.136]	
Indirect effect	0.003	[−0.001, 0.008]	
Mediator: enjoyment of life ($n = 3,532$)			
Total effect	0.097**	[0.026, 0.168]	
Direct effect	0.090*	[0.019, 0.160]	
Indirect effect[a]	0.008*	[0.001, 0.014]	7.8%
Mediator: eudemonic well-being ($n = 3,531$)			
Total effect	0.097**	[0.026, 0.167]	
Direct effect	0.089*	[0.019, 0.160]	
Indirect effect[a]	0.008*	[0.001, 0.014]	8.0%
Mediator: life satisfaction ($n = 3,535$)			
Total effect	0.096**	[0.026, 0.166]	
Direct effect	0.094**	[0.024, 0.164]	
Indirect effect	0.002	[−0.002, 0.005]	
Mediator: loneliness ($n = 3,530$)			
Total effect	0.096**	[0.026, 0.167]	
Direct effect	0.095**	[0.025, 0.166]	
Indirect effect	0.001	[−0.001, 0.003]	
Mediator: social support ($n = 3,570$)			
Total effect	0.089*	[0.019, 0.160]	
Direct effect	0.086*	[0.015, 0.156]	
Indirect effect	0.003	[−0.001, 0.008]	
Mediator: social strain ($n = 3,566$)			
Total effect	0.090*	[0.021, 0.161]	
Direct effect	0.088*	[0.018, 0.158]	
Indirect effect	0.003	[−0.001, 0.007]	
Mediator: weight stigma ($n = 3,543$)			
Total effect	0.090*	[0.020, 0.160]	
Direct effect	0.056	[−0.015, 0.128]	
Indirect effect[a]	0.034**	[0.011, 0.056]	37.4%
Mediator: positive affect ($n = 3,526$)			
Total effect	0.084*	[0.013, 0.155]	
Direct effect	0.081*	[0.010, 0.152]	
Indirect effect	0.003	[−0.003, 0.009]	
Mediator: neuroticism ($n = 3,527$)			
Total effect	0.089*	[0.018, 0.161]	
Direct effect	0.089*	[0.018, 0.160]	
Indirect effect	0.000	[−0.001, 0.001]	
Mediator: extraversion ($n = 3,532$)			
Total effect	0.092*	[0.020, 0.163]	
Direct effect	0.092*	[0.020, 0.163]	
Indirect effect	0.000	[−0.003, 0.003]	
Mediator: conscientiousness ($n = 3,530$)			
Total effect	0.091*	[0.020, 0.162]	
Direct effect	0.089*	[0.018, 0.160]	
Indirect effect	0.002	[−0.001, 0.006]	
Mediator: agreeableness ($n = 3,534$)			
Total effect	0.090*	[0.019, 0.161]	
Direct effect	0.092*	[0.020, 0.163]	
Indirect effect	−0.002	[−0.005, 0.001]	
Mediator: openness ($n = 3,527$)			
Total effect	0.090*	[0.019, 0.162]	
Direct effect	0.090*	[0.019, 0.162]	
Indirect effect	−0.000	[−0.002, 0.001]	

Note. BMI = body mass index; CI = confidence interval.
[a] The positive direction of indirect effects of psychological measures resulted from both pathways a (i.e., association between obesity and psychological measure) and b (i.e., association between psychological measure and physiological dysregulation) in the same direction. Both pathways a and b were in the negative direction for enjoyment of life and eudemonic well-being, but in the positive direction for weight stigma. All mediation models were adjusted for baseline physiological dysregulation, age, age^2, sex, paid employment status, ethnicity, marital status, educational level, and household wealth. Statistical significance levels and 95% CIs are based on models using robust standard errors.
* $p < .05$. ** $p < .01$.

Table 4
Multiple Mediation Models of Psychological Measures in Study 1

Mediation model (reference group: non-obesity [BMI < 30 kg/m^2])	Point estimates	95% CI	Effect ratio
Mediators: enjoyment of life, eudemonic well-being, weight stigma ($n = 3,338$)			
Total effect	0.100**	[0.027, 0.174]	
Direct effect	0.063	[−0.011, 0.138]	
Indirect effect	0.037**	[0.012, 0.062]	36.9%
Decomposition of indirect effects from multiple mediation model			
Enjoyment of life	0.004	[−0.002, 0.010]	4.2%
Eudemonic well-being	0.002	[−0.004, 0.008]	2.0%
Weight stigma	0.031*	[0.006, 0.056]	30.7%

Note. All mediation models were adjusted for baseline physiological dysregulation, age, age^2, sex, paid employment status, ethnicity, marital status, educational level, and household wealth. Statistical significance levels and 95% CIs are based on models using robust standard errors. BMI = body mass index; CI = confidence interval.
* $p < .05$. ** $p < .01$.

studies may have been related to weight gain from baseline to follow-up, we conducted additional mediation analyses of weight stigma by controlling for changes in BMI between baseline and follow-up (Tables B9 and C9 in the online supplemental materials for Studies 1 and 2, respectively). In Study 1, weight stigma remained a statistically significant mediator but accounted for a smaller amount of the relationship (22% as opposed to 37%). Similarly, in Study 2 the significant indirect effect of changes in weight stigma on physiological dysregulation remained significant but a smaller proportion of variance was explained (8% as opposed to 13%).

Discussion

Contemporary psychological models of obesity propose that vulnerability to weight stigma, reduced well-being, and negative feelings are central to understanding the health consequences of obesity (Puhl et al., 2020; Tomiyama, 2019). In this study we found partial support for this idea: across two cohorts of U.K. and U.S. older adults, the experience of weight stigma independently explained the prospective effect of obesity on physiological dysregulation. In Study 1, weight stigma was measured 2 years after baseline and could independently explain 31% of the link between obesity and physiological dysregulation 4 years later. In Study 2, changes in weight stigma from baseline to follow-up (and not baseline weight stigma) explained 13% of the effect of obesity on physiological dysregulation. We also tested whether the mediating role of weight stigma overlapped with weight gain over the course of the study. In Studies 1 and 2, the proportion of variance mediated by weight stigma reduced by approximately half when changes in BMI were controlled but remained significant at $p < .05$ in both studies. Taken together, these findings indicate that weight stigma may act on physiological health via weight gain.

Weight stigma is a common source of psychological distress among people living with obesity as society perceives that factors within personal control (e.g., physical inactivity, overeating) are responsible for adiposity (Puhl & Heuer, 2010). The prevalence of weight stigma increases with higher obesity classes, from 19% for people in Class I obesity to 42% for those in Class II and III obesity according to a meta-analysis of most U.S. studies (Spahlholz et al., 2016). Weight stigma may act as a chronic stressor that contributes to worsening physiological functioning and stress-induced biological and behavioral mechanisms (Tomiyama, 2014; Tomiyama et al., 2018). Biological responses to exposure to chronic stressors (e.g., weight stigma) can activate the sympathetic nervous system (SNS) and HPA axis, including cortisol secretion that have harmful effects on immune, metabolic, neuroendocrine, and cardiovascular systems, which over time can build up allostatic load (Hamer & Steptoe, 2012; Merabet et al., 2022; Whitworth et al., 2005; Yao et al., 2019). These direct biological mechanisms (not through weight gain) may therefore be responsible for part of the association between weight stigma and physiological dysregulation observed. In addition, mediation by weight stigma through weight gain may occur. For example, increased cortisol secretion can accelerate weight gain (Vicennati et al., 2009) and increase fat accumulation (Anagnostis et al., 2009). Weight stigma may also lead to weight gain through behavioral mechanisms, such as impairing weight management. Those who experience weight stigma are more likely to report overeating and unhealthy eating and reduced motivation to engage in physical activity (Tomiyama, 2014; Vartanian & Porter, 2016; Vartanian & Shaprow, 2008; Zhu et al., 2022). In line with this literature, previous longitudinal analyses using ELSA and HRS data showed that reporting weight stigma at baseline increased the odds of becoming and/or remaining obese over the follow-up period (Jackson et al., 2014; Sutin & Terracciano, 2013). However, an alternative hypothesis is that weight gain may increase likelihood of reporting weight stigma and therefore both weight stigma and physiological dysregulation are "symptoms" of historic weight gain.

Contrary to our predictions, we found little evidence that a broad array of psychological factors could explain the prospective association between obesity and physiological dysregulation. In Study 1 (U.K. sample), a set of psychological factors (depressive symptoms, enjoyment of life, eudemonic well-being, life satisfaction, loneliness, social support, social strain, positive affect, and big five personality traits), did not account for a significant proportion of the link between obesity and subsequent dysregulation. Similarly, in Study 2 (U.S. sample), we examined a number of the same psychological factors and a range of other factors (negative affect, purpose in life, anxiety, hopelessness, optimism, pessimism, cynical hostility, perceived constraint, and perceived mastery). However, none of the psychological factors examined mediated the association between obesity and physiological dysregulation. However, it may be the case that some psychological factors contribute to the initial development of obesity and therefore indirectly contribute to obesity-related worsening of physiological health.

Table 5
Single Mediation Models of Psychological Measures in Study 2

Mediation models (reference group: non-obesity [BMI < 30 kg/m^2])	Point estimates	95% CI	Effect ratio
Mediator: depressive symptoms ($n = 7{,}332$)			
Total effect	0.169***	[0.120, 0.219]	
Direct effect	0.170***	[0.120, 0.219]	
Indirect effect	−0.001	[−0.001, 0.001]	
Mediator: life satisfaction ($n = 6{,}395$)			
Total effect	0.187***	[0.132, 0.241]	
Direct effect	0.187***	[0.132, 0.241]	
Indirect effect	−0.000	[−0.001, 0.000]	
Mediator: loneliness ($n = 6{,}380$)			
Total effect	0.185***	[0.131, 0.240]	
Direct effect	0.185***	[0.131, 0.240]	
Indirect effect	−0.000	[−0.001, 0.001]	
Mediator: social support ($n = 6{,}430$)			
Total effect	0.185***	[0.130, 0.239]	
Direct effect	0.185***	[0.130, 0.239]	
Indirect effect	−0.000	[−0.001, 0.001]	
Mediator: social strain ($n = 6{,}427$)			
Total effect	0.184***	[0.130, 0.238]	
Direct effect	0.183***	[0.128, 0.238]	
Indirect effect	0.001	[−0.001, 0.003]	
Mediator: weight stigma ($n = 6{,}402$)			
Total effect	0.182***	[0.127, 0.236]	
Direct effect	0.178***	[0.123, 0.234]	
Indirect effect	0.003	[−0.011, 0.017]	
Mediator: positive affect ($n = 6{,}370$)			
Total effect	0.181***	[0.126, 0.235]	
Direct effect	0.180***	[0.125, 0.235]	
Indirect effect	0.001	[−0.001, 0.002]	
Mediator: negative affect ($n = 6{,}378$)			
Total effect	0.183***	[0.128, 0.237]	
Direct effect	0.183***	[0.128, 0.237]	
Indirect effect	0.000	[−0.001, 0.001]	
Mediator: purpose in life ($n = 6{,}349$)			
Total effect	0.184***	[0.129, 0.239]	
Direct effect	0.184***	[0.129, 0.238]	
Indirect effect	0.000	[−0.002, 0.002]	
Mediator: anxiety ($n = 6{,}385$)			
Total effect	0.182***	[0.127, 0.237]	
Direct effect	0.182***	[0.128, 0.238]	
Indirect effect	−0.000	[−0.002, 0.001]	
Mediator: hopelessness ($n = 6{,}407$)			
Total effect	0.185***	[0.130, 0.239]	
Direct effect	0.184***	[0.129, 0.238]	
Indirect effect	0.001	[−0.001, 0.003]	
Mediator: optimism ($n = 6{,}372$)			
Total effect	0.187***	[0.132, 0.241]	
Direct effect	0.186***	[0.131, 0.241]	
Indirect effect	0.001	[−0.001, 0.002]	
Mediator: pessimism ($n = 6{,}371$)			
Total effect	0.186***	[0.131, 0.240]	
Direct effect	0.185***	[0.131, 0.240]	
Indirect effect	0.001	[−0.001, 0.002]	
Mediator: cynical hostile ($n = 6{,}260$)			
Total effect	0.193***	[0.141, 0.244]	
Direct effect	0.191***	[0.139, 0.243]	
Indirect effect	0.002	[−0.001, 0.005]	

(*table continues*)

Table 5 (*continued*)

Mediation models (reference group: non-obesity [BMI < 30 kg/m^2])	Point estimates	95% CI	Effect ratio
Mediator: perceived constrain ($n = 6{,}392$)			
Total effect	0.184***	[0.129, 0.238]	
Direct effect	0.184***	[0.129, 0.238]	
Indirect effect	0.000	[−0.000, 0.001]	
Mediator: perceived mastery ($n = 6{,}393$)			
Total effect	0.184***	[0.129, 0.238]	
Direct effect	0.184***	[0.130, 0.239]	
Indirect effect	−0.001	[−0.002, 0.001]	
Mediator: neuroticism ($n = 6{,}372$)			
Total effect	0.183***	[0.129, 0.238]	
Direct effect	0.183***	[0.128, 0.237]	
Indirect effect	0.000	[−0.001, 0.001]	
Mediator: extraversion ($n = 6{,}380$)			
Total effect	0.182***	[0.127, 0.237]	
Direct effect	0.182***	[0.127, 0.236]	
Indirect effect	0.000	[−0.001, 0.001]	
Mediator: conscientiousness ($n = 6{,}372$)			
Total effect	0.184***	[0.130, 0.239]	
Direct effect	0.184***	[0.129, 0.238]	
Indirect effect	0.001	[−0.001, 0.002]	
Mediator: agreeableness ($n = 6{,}377$)			
Total effect	0.183***	[0.129, 0.238]	
Direct effect	0.183***	[0.129, 0.238]	
Indirect effect	0.000	[−0.001, 0.001]	
Mediator: openness ($n = 6{,}356$)			
Total effect	0.181***	[0.126, 0.236]	
Direct effect	0.181***	[0.126, 0.235]	
Indirect effect	0.001	[−0.001, 0.002]	

Note. All mediation models were adjusted for baseline physiological dysregulation, age, age^2, sex, paid employment status, ethnicity, marital status, years in education, and household wealth. Statistical significance levels and 95% CIs are based on models using robust standard errors. BMI = body mass index; CI = confidence interval.
*** $p < .001$.

As we found no convincing evidence that other psychological factors explained the prospective association between obesity and physiological dysregulation (aside from weight stigma), the association between obesity and physiological dysregulation may be attributable to biological processes particularly related to visceral or central obesity (Després, 2012; Powell-Wiley et al., 2021; Tchernof & Després, 2013). Adipose tissue plays a major role in systemic metabolic regulation, including the process of storing energy in the form of lipids, controlling lipid distribution and acting as an endocrine organ to yield bioactive factors (Luo & Liu, 2016). The expansion of adipose tissue can occur during the development of obesity and its distribution is important in determining metabolic complications. Adipose tissue mainly localized intra-abdominally in visceral depots or known as *central obesity* is metabolically active and associated with greater free fatty acid (FFA) delivery that can promote metabolic dysregulations, such as inflammation, dyslipidemia, and insulin resistance (Chait & den Hartigh, 2020; Jensen, 2006; Yki-Järvinen, 2014). Individuals with obesity and metabolic dysregulations tend to have high levels of abdominal visceral adipose tissue than those with obesity but normal metabolic risk profile, known as *metabolically healthy obesity* (Blüher, 2020; Després, 2012). High, relative to normal waist (a marker of excess visceral fat) is associated with high triglycerides among participants with normal BMI across three nationally representative populations (United States, United Kingdom, and Taiwan; Vasunilashorn et al., 2013). In addition to prominent biological mechanisms, having excess visceral fat is also associated with weight stigma, particularly in women across the BMI spectrum (Keirns et al., 2021). Therefore, the presence of excess visceral fat in the body, independently of BMI measure, may also contribute to deterioration of physiological health, which may occur as a result of experiencing chronic stress (i.e., weight stigma).

Strengths, Limitations, and Future Study Directions

This present study is the first to investigate whether a broad range of psychological factors associated with obesity contribute to the prospective association between obesity and physiological dysregulation. Although there were some minor differences in study methodologies between ELSA and HRS (e.g., ELSA collected venous blood samples, while HRS used dried blood spots), strengths include the use of two large representative longitudinal data from the United Kingdom and the United States to examine the generalizability of findings. In addition, we used both IPW and MICE approaches to address missing observations and potential selection bias. MICE approach also provided the estimations based on the bigger

analytical sample size and this approach has been shown to increase the statistical power (Arnold & Kronmal, 2003). Therefore, the null findings in this study are less likely due to lack of statistical power.

The present study also has limitations. In Study 1, weight stigma was only available in a wave between the baseline and follow-up (and therefore not concurrent with measurement of obesity). Furthermore, some psychological measures in ELSA were not available in HRS and vice versa. We were also not able to assess every psychological factor that may play a role in explaining the obesity-physiological dysregulation association. For instance, obesity has been shown to be associated with reduced happiness (Ul-Haq et al., 2014), which in turn, can predict lower levels of inflammation (Panagi et al., 2019) and hypertension (Blanchflower & Oswald, 2008). Likewise, the mediating role of quality of life (Trevisol et al., 2011; Ul-Haq et al., 2014) was not examined in this study. Additional factors of importance may include detailed measurement of chronic stress, given that obesity stigma is hypothesized to impact health through stress-based pathways (Kumar et al., 2022). Future research should consider examining other psychological pathways from obesity to physiological dysregulation. Another limitation of our study was that only two time point separated assessments of physiological dysregulation were used, and hence, might not adequately capture reliable changes in physiological dysregulation over time. Therefore, future research may benefit from examining repeated multiple assessments of physiological dysregulation. Furthermore, the present study only included older adult participants (≥50 years) and therefore, findings may not generalize to younger populations. Future research can benefit from exploring the extent to which psychological measures associated with heavier body weight are responsible for the deterioration of physiological health among adolescents, young, and middle-aged adults. White participants contributed to the majority of the samples in this study, and hence, more studies drawing on more diverse samples would be beneficial in future research.

Conclusions

The prospective association between obesity and physiological dysregulation was largely not explained by psychological factors. However, experiencing weight stigma is associated with increased weight gain and this process may explain obesity-related declines in physiological health.

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