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Dynamics between psychological distress and body mass index throughout adult life; evidence from 3 large cohort studies

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ABSTRACT

Background: Associations between body mass index (BMI) and psychological distress (PD) have been reported; however, few longitudinal studies have accounted for likely life-course differences in BMI and PD stability, consistency, and their interplay across time.

Methods: Via random intercepts cross-lagged panel models, we assessed the predictive effects (from BMI to PD or vice-versa) across the last two centuries in the Coronary Artery Risk Development in Young Adults [CARDIA, beginning in 1985-6] study using the Center for Epidemiological Studies-Depression Scale [CES-D], and in the National Child Development Study [NCDS, beginning in 1958] and British Cohort Study [BCS, beginning in 1970] using the Malaise Inventory [MI]), assessed at least 4 times in adult life.

Findings: In CARDIA (n = 4724), NCDS58 (n = 7149) and BCS70 (n = 5967), autoregressive effects were stronger for BMI than for PD, meaning that carry-over effects from one occasion to the next were larger for BMI than for PD. Small interindividual correlations between traits of higher BMI and higher PD were identified among females ($r_{female}<|0{\cdot}2|$) but not males ($r_{male}<|0{\cdot}03|$) in CARDIA and NCDS. Cross-lagged effects were very weak or close to zero (standardized effects $\eta<|0{\cdot}1|$).

Interpretation: In the United States, depressive symptoms and BMI were positively correlated at the trait level among females. In the United Kingdom, relationships between PD and BMI were inconsistent between generations, with effect sizes of unlikely clinical importance, indicating negligible dominance of an intraindividual effect of BMI on PD or vice versa.

1. Introduction

Morbidity and mortality associated with obesity is an increasing public health concern (Abdelaal et al., 2017). The co-occurrence of obesity and psychological distress has been well described (Abdelaal et al., 2017), especially for depressive and anxiety disorders (Husky et al., 2018). However, the current literature reports controversial bi-directional findings, inconsistent evidence regarding the magnitudes of these correlations, and heterogeneous interrelated trajectories that differ according to sociodemographic moderators, such as race and sex, and across age groups (Brandheim et al., 2013; de Wit et al., 2010; Luppino et al., 2010; Patalay and Hardman, 2019).

Methodological inconsistencies regarding study design and choice of the statistical model to analyze the data may also, in part, explain mixed findings. Most studies have relied on cross-sectional data (Benson et al., 2013; de Wit et al., 2010; Jeffers et al., 2013; Tyrrell et al., 2019; Wiltink et al., 2013) so the direction of the association between psychological distress and BMI cannot be determined. In longitudinal studies, bidirectional associations have been found, with a pooled odds ratio (OR) of 1·55 for the association between high body mass index (BMI) at baseline

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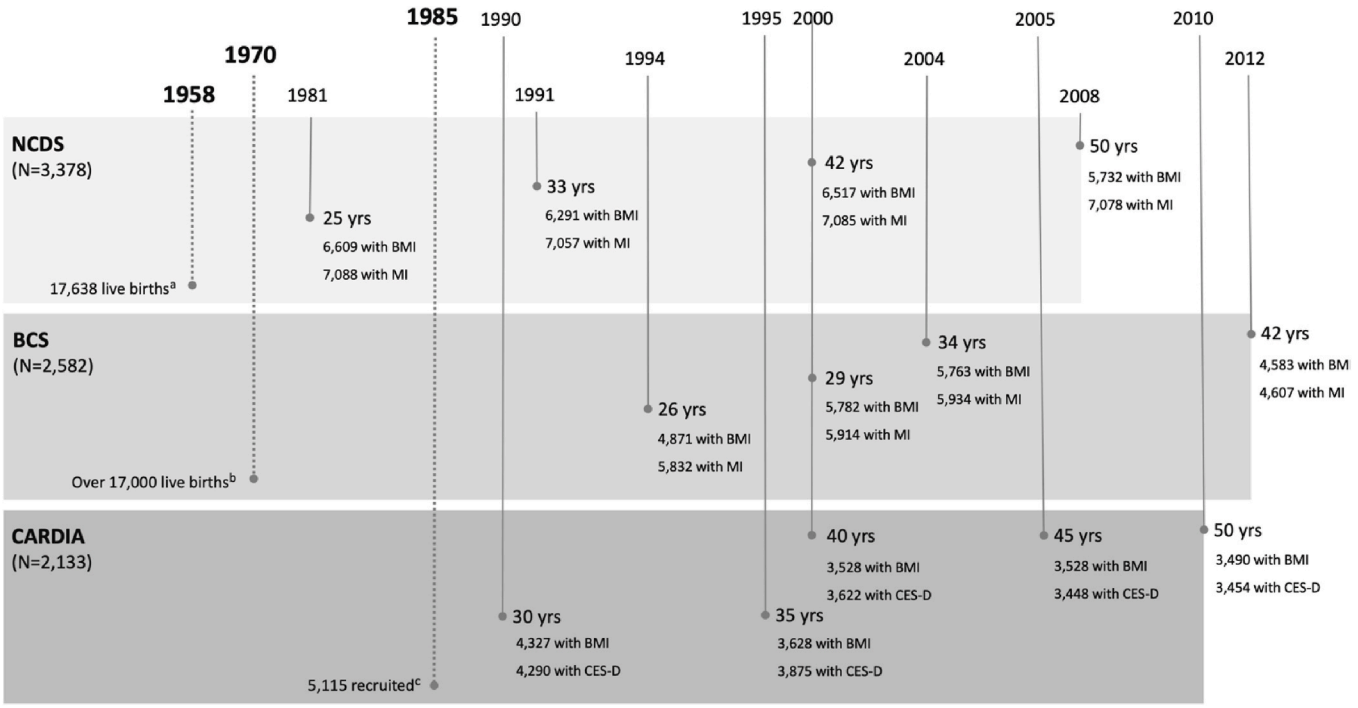

Fig. 1. Timeline of the assessments and sample size for the three cohorts. Cohorts with year of assessment and age per wave, and number of cases with valid data for BMI and psychological symptoms. a. Live births in England, Scotland, and Wales during a single week at March 3–9 (51); b. Live births in Great Britain between April 5–11; c. Recruited individuals aging 18–30 years during 1985–1986 in Birmingham (AL), Chicago (IL), Minneapolis (MN), and Oakland (CA); BCS, British Cohort Study; BMI, Body Mass Index; CARDIA, Coronary Artery Risk Development in Young Adults; CES-D, Center for Epidemiologic Studies scale; MI, Malaise Inventory; NCDS, National Child Development Study.

and depression/depressive symptoms at follow-up, and a pooled OR of 1·20 for a depression exposure predicting an obesity outcome (Luppino et al., 2010). However, neither of these ORs can account for the question of whether obesity might lead to depressive symptoms or vice versa, and how much of these characteristics are related to traits of obesity or depression vs. predictions from one occasion to another. Some longitudinal studies have tried to capture the dynamics of change over time, but these models have assumed that the effect of a predictor is constant over the period over which the outcome was assessed (Anderson et al., 2007; Bjerkeset et al., 2008), limiting the accuracy of the estimation to that period (Abrahamowicz et al., 1996).

Another methodological issue is the categorization of continuous variables as categorical (Streiner, 2002) (e.g. BMI separated into "overweight" or "obese" (Luppino et al., 2010), or exceeding a cut-off on a depression screening instrument (Anderson et al., 2007; Pine et al., 2001; Richardson et al., 2003), rather than the severity of these measures being considered as continuous measurements. The former can result in lost statistical power (Sauerbrei and Royston, 2010; Streiner, 2002).

When repeated measures are available for both outcomes, the cross-lagged panel model (CLPM) can be used to identify the most likely direction of predictive relationships. The CLPM examines the effects of two or more variables on each other over time (Rogosa, 1980) (cross--lagged effect) while considering the effects of each variable at a given occasion on the same variable at the subsequent occasion of measurement (autoregressive effect). Recently, the CLPM has been applied to investigate temporal associations between BMI and psychological symptoms in childhood (Brandheim et al., 2013) and at older ages (Kim et al., 2014). For instance, the UK Millennium birth cohort followed 19 517 individuals, assessing BMI and internalizing symptoms across six waves from participant ages 9 months to 14 years old. Cross-lagged models in that study showed weak prediction of cross-domain internalizing symptoms on BMI at age 7 years, but BMI predicted internalizing symptoms at age 14 years (Brandheim et al., 2013), and in a Korean longitudinal study (Kim et al., 2014), depressive symptoms predicted weight loss in older adults; however, these relationships have yet to be fully investigated in adults, a period in which people often gain excessive weight (de Wit et al., 2010).

One criticism of the CLPM is that its parameters do not represent actual within-person relationships over time, which can lead to erroneous conclusions regarding the presence, predominance, and direction of the influences of one variable on the other (Hamaker et al., 2015). Advances in structural equation modeling have allowed the incorporation of random intercepts into the CLPM (i.e., the RI-CLPM). This model disentangles between- and within-person effects by accounting for time-invariant stability (traits) (Hamaker et al., 2015). In the context of psychological–BMI relationships, the model offers a new opportunity to reveal the occasion-specific (state) and consistent and long lasting (trait) features, using repeated measures of BMI and psychological symptoms, and to test correlations between the traits vs. reciprocal predictions between the occasion-specific measures over time.

In the RI-CLPM, reciprocal "cross-lagged" predictions are within person effects, and they can be interpreted to test whether periods in which a person has higher depressive symptoms place a person at increased risk for subsequently higher BMI, vs. whether following periods in which a person has increased BMI, that person is likely to present changes in psychological status. Within-subject fluctuations in BMI or psychological symptoms and enduring between-subjects behaviors (commonly referred to as "traits") are dissociable under repeated measures designs. Without controlling for trait-like carry-over and dispositional effects, we are prone to erroneous interpretations in the reciprocal prediction; for example, if two behaviors are heavily traits (i.e., a large between subjects effect), it is less likely to observe a strong reciprocal prediction (i.e., within-subjects effect). A between-persons hypothesis would be that on average, adults who have higher psychological symptoms compared to adults with lower psychological symptoms are more likely to have higher BMI. These between-subjects aspects are ascertained by random intercepts in the model, which estimate stable

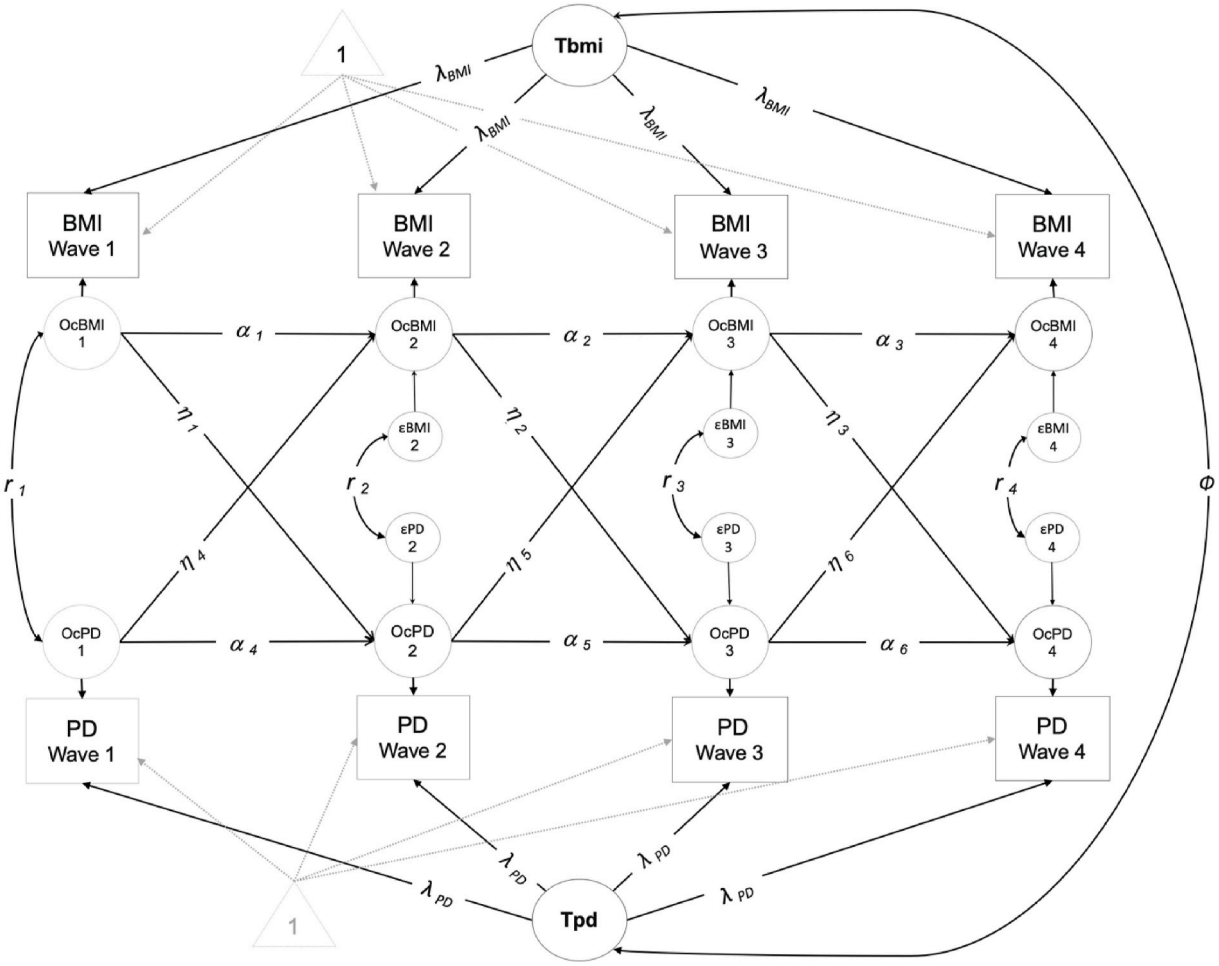

Fig. 2. Schematic representation of Cross-lagged Panel Model with Random Intercept. Triangles represent constants (for the mean structure); squares denote observed variables; circles represent latent variables; squares = observed variables; α = regression coefficient; autoregressive), η = regression coefficient (cross-lagged, no constrains across time); λ = factor loadings (unstandardized factor loadings are constrained to be 1 across the time); ε = dynamic errors; r = correlation coefficient; Φ = trait; OcBMI/OcPD = occasion-specific latent variables; Tbmi/Tped = trait-like variables; BMI= Body Mass Index; PD=Psychological Distress; p = p-value.

time-invariant trait-like features. By modelling trait variance, the RI-CLPM is also adjusting for stable unmeasured covariates that affect both BMI and depressive symptoms; for instance, biases reported based on sex and ethnicity (Masselink et al., 2018). It is important to disentangle these effects because between-person associations are most indicative of which groups might benefit from an intervention (e.g. age, gender or ethnic group), while within-person associations may be most useful to detect potential modifiable targets (e.g. weight loss intervention vs. interventions for depressive symptoms) (Masselink et al., 2018).

Most studies have included two waves of assessment (Fitzsimons and Benedetta, 2017; Hajek and König, 2018; Herva et al., 2006; Koponen et al., 2008; Norris et al., 2020; Sachs-Ericsson et al., 2007) which are sufficient to specify a CLPM and address stability by controlling for the previous measure. However, this is insufficient for an RI-CLPM, which can further account for trait-like time-invariant stability, arguably more important when the goal is to clarify predictive effects (Hamaker et al., 2015). Thus, we can address the question of whether relationships between psychological symptoms and BMI are in fact predictive in a specific direction, or simply correlated with one another.

In this study, we aimed to determine the reciprocal effects of BMI and psychological symptoms as each evolve from early adulthood to middle age, and the stability of these constructs through life considering trait and occasion specific characteristics. To explore the interplay between different facets of psychological distress, we used data from three large cohorts from different historical time points and two countries, accounting for changes in the shape of the distribution of BMI over the last 30 years. Owing to established sex and ethnic differences in the trajectories of BMI (Sacker and Wiggins, 2002) and the prevalence of mental disorders (Stanikova et al., 2018), RI-CLPM models were considered in males and females separately, and in blacks and whites separately in the United States cohort.

2. Methods

2.1. Cohorts

We used data from one American cohort and two British birth cohorts, described in Fig. 1.

The National Child Development Study (NCDS58) birth cohort sample size for this is 6888 participants (53% female) who completed the Malaise Inventory (MI) scale and had BMI valid data. Since the identification of gender as a social group was not asked directly to participants, sex assigned at birth was adopted, considering participants to be “male” or “female”. The study sample included 5967 BCS cohort members (57% female) with valid data who responded to the four surveys, representing 51% of the sample in the last assessment (2012) and relatively homogenous in terms of ethnicity. For information regarding sampling and survey design of the NCDS see https://cls.ucl.ac.uk/cls-st

Table 1
Depressive symptoms and BMI.

Cohort	NCDS (N = 7149					BCS (N = 5967)					CARDIA (N = 4724)				
	Age[a]	Male (n = 3378)		Female (n = 3771)		Age	Male (n = 2582)		Female (n = 3385)		Age	Male (n = 2133)		Female (n = 2591	
Wave		N	Mean (SD)	N	Mean (SD)		N	Mean (SD)	N	Mean (SD)		N	Mean (SD)	N	Mean (SD)
BMI															
1	23	3107	23.047 (2.84)	3502	22.161 (3.21)	26	1611	24.423 (3.40)	3260	23.270 (3.93)	30	1944	25.980 (4.67)	2383	26.290 (6.74)
2	33	3035	25.470 (3.66)	3256	24.529 (4.78)	30	2514	25.578 (3.81)	3268	24.266 (4.62)	35	1738	27.167 (5.14)	2147	27.818 (7.47)
3	42	3091	26.496 (3.83)	3426	25.376 (4.97)	34	2528	26.457 (4.09)	3235	25.122 (5.00)	40	1615	28.288 (5.46)	2013	29.139 (7.75)
4	50	2763	28.112 (4.48)	2969	26.845 (5.49)	42	2015	27.418 (4.57)	2568	26.129 (5.47)	45	1527	28.927 (5.72)	2001	29.815 (7.83)
5											50	1513	29.498 (5.91)	1977	30.682 (8.01)
	Malaise Inventory −9 Items[b]										CES-D[c]				
1	23	3357	17.230 (1.20)	3731	16.512 (1.62)	26	2524	16.692 (1.56)	3308	15.895 (1.76)	30	1925	10.367 (7.21)	2365	11.946 (8.75)
2	33	3341	17.351 (1.19)	3716	16.816 (1.59)	30	2556	16.835 (1.56)	3358	16.337 (1.69)	35	1732	9.987 (7.39)	2143	11.270 (8.78)
3	42	3340	16.811 (1.61)	3745	16.308 (1.77)	34	2569	16.650 (1.74)	3365	16.201 (1.88)	40	1603	8.530 (7.04)	2019	9.664 (8.39)
4	50	3337	16.879 (1.66)	3741	16.295 (2.01)	42	1947	16.489 (1.82)	2660	16.077 (1.90)	45	1492	8.630 (6.98)	1956	9.859 (8.46)
5											50	1499	8.960 (7.00)	1955	9.850 (8.24)

BCS=British Cohort Study, BMI=Body Mass Index, CARDIA=Coronary Artery Risk Development in Young Adults, CES-D = Center for Epidemiologic Studies scale, NCDS= National Child Development Study, SD=Standard Deviation.
[a] Average age.
[b] Higher scores indicate less symptoms, range 9–18.
[c] The higher, the more depressive symptoms, range 0–60.

udies/1958-national-child-development-study-2/

The 1970 British Cohort Study (BCS70) sample for this analysis included 5967 participants (57% female) with valid data from ages 26 to 42 (2012) years. The study sample included 5967 BCS cohort members (57% female) with valid data who responded to the four surveys, representing 51% of the sample in the last assessment (2012). The sample was also relatively homogenous in terms of ethnicity (Elliott and Shepherd, 2006). British cohort assessment involved collection of sociodemographic and anthropometric measures and the MI (Elliott and Shepherd, 2006).

For information regarding sampling and survey design of the BCS see https://cls.ucl.ac.uk/cls-studies/1970-british-cohort-study/

The CARDIA is a 30-year cohort study investigating risk factors and the evolution of cardiovascular disease in young adults, with nine waves of data collection from 1985 to 2016. A total of 5115 black (non-Hispanic) and white (non-Hispanic) individuals age 18–30 years (Cutter et al., 1991). For information regarding sampling and survey design of the CARDIA see https://www.cardia.dopm.uab.edu.

In BCS and NCDS, the MI was used to assess psychological distress. In CARDIA, the Center for Epidemiologic Studies Depression Scale (CES-D) scale was used to assess depressive symptoms.

2.2. Measurements

2.2.1. Body mass index

BMI was calculated as weight in kilograms (kg) divided by height in square meter (m^2) for each individual. In CARDIA, body weight measurement was performed using a balance beam scale, with the participant wearing light clothing and no shoes. Height was measured using a vertically mounted metal centimeter ruler and a metal carpenter's square. In the NCDS, weight and height were self-reported at ages 23, 42 and 50, measured by a trained interviewer at age 33. In BCS, weight and height was self-reported in all waves (Cutter et al., 1991; Elliott and Shepherd, 2006).

2.2.2. Malaise Inventory (MI)

The MI is a self-report scale with yes/no responses to questions related to psychological distress (Rodgers et al., 1999) adopted by NCDS and BCS. The MI items address five clusters of symptoms, including three DSM-5 criteria (energy loss, sadness and agitation) and two non-DSM-5 criteria comprising irritability and pessimism. The items are scored such that higher scores reflect lower psychological distress.

The main analysis involved the nine-item version of the MI, which includes feelings of upset and irritability, with higher factor loadings (Grant et al., 1990), and excludes questions related to somatic symptoms and sleep problems, with finding acceptable internal consistency (Furnham and Cheng, 2015). Good evidence for longitudinal invariance of the MI has been reported (Ploubidis et al., 2019) wherein the nine items exhibited high stability throughout adulthood, especially among men, due mostly to interindividual trait differences (Scarpato et al., 2020).

2.2.3. Center for Epidemiological Studies Depression Scale (CES-D)

The CES-D is a 20-item self-reported scale designed to assess depressive symptoms in adolescents and adults (Radloff, 1977) adopted by CARDIA. Designed to assess somatic symptoms, depressed affect, and absence of positive affect or anhedonia, as well as interpersonal challenges. Higher scores reflect a greater number and frequency of depressive symptoms.

The approval of the Multicentre Research Ethics Committee (MREC) and London – Central Research Ethics Committee (CREC) and also registration at Integrated Research Application System (IRAS) were requested to monitor the NCDS, Biomedical Research and BCS activities. Approval from National Institutes of Health (NIH) and respective collaborating universities were requested for CARDIA studies. Additional information can be found in Elliott and Shepherd (2006) and Cutter et al. (1991). Written informed consent was obtained from all subjects, after the nature of the procedures had been fully explained. All procedures contributing to this work comply with the ethical standards

Table 2
Goodness-of-fit indices for depressive symptoms based on NCDS, BCS and CARDIA.

Cohort Model	Fit Indices					
	χ2 (df)	RMSEA	90 Percent C.I.	CFI	TLI	SRMR
NCDS						
Model 1	266.219 (26) *	0.051	0.045 to 0.056	0.987	0.972	0.082
Model 2	287.696 (36) *	0.044	0.040 to 0.049	0.986	0.979	0.094
S–B scaled Chi-square (Δdf), p-value	24.190 (10), 0.0071					
BCS						
Model 1	131.071 (26) *	0.037	0.031 to 0.043	0.995	0.989	0.051
Model 2	136.823 (36) *	0.031	0.025 to 0.036	0.995	0.992	0.053
S–B scaled Chi-square (Δdf), p-value	7.706 (10), 0.6575					
CARDIA						
Model 1	212.329 (50) *	0.037	0.032 to 0.042	0.989	0.981	0.027
Model 2	219.407 (64) *	0.032	0.027 to 0.037	0.990	0.986	0.029
S–B scaled Chi-square (Δdf), p-value	13.5586 (14), 0.4831					

Model 1 = less restrictive model where all cross-lagged and autoregressive effects were freely estimated within and between groups; Model 2 = more restrictive model where cross-lagged and autoregressive of the parameters in Model 1 equal across males and females, but variant across time; S–B scaled Chi-square = Satorra-Bentler Scaled Chi-square Difference test; C.I. = confidence interval; Δdf = difference of the degrees of freedom; Const. lagged-parameters = constrained lagged-parameter; * = p value < 0.0001; BCS = British Cohort Study, CARDIA = Coronary Artery Risk Development in Young Adult, CES-D = Center for Epidemiologic Studies scale; CFI = Comparative Fit Index; df = degree of freedom; NCDS = National Child Development Study; RMSEA = Root Mean Square Error of Approximation; SRMR = Standardized Root Mean Square Residual; TLI = Tucker-Lewis Index.

of the Helsinki Declaration of 1975, as revised in 2008.

For the study protocol see https://osf.io/2y58s/

2.3. Statistical analysis

To evaluate the dynamics BMI and psychological distress/depressive symptoms we used the random-intercept cross-lagged panel model (RI-CLPM) as depicted in Fig. 2. Mplus version 8.3 (Muthén and Muthén, n. d.) all the models and because the observed variables were skewed and missing values were present, the maximum likelihood robust estimation method was applied in all analyses, which uses the Huber–White sandwich estimator to estimate robust standard errors. Moreover, under ignorable missing data conditions (missing completely at random and missing at random), full information maximum likelihood is more efficient than other methods of dealing with missing data (Enders and Bandalos, 2001). Details about the specification of RI-CLPM and its advantages in relation to the traditional cross-lagged panel model might be found in Hamaker et al. (2015). Briefly stating, cross-lagged effects (represented by η) were estimated by regressing the occasion-specific latent variables in a crossed fashion, where an occasion-specific BMI variable was regressed on the occasion-specific psychological distress/depressive symptoms variable one occasion before, and vice versa. The α path are first order auto-regressive effects. In case of psychological symptoms as outcome, they express to what extent a deviation in the person-specific mean of psychological distress/depressive symptoms at an earlier time point is associated with a subsequent deviation from the person-specific mean psychological distress/depressive symptoms, controlling for previous (t −1) deviations from the person-specific mean in BMI (Hamaker et al., 2015).

Two trait-like individual difference variables (called TBMI and TPD/Tdep) that were constantly present over time were created from the observed variables (represented by ovals in Fig. 2). Because we expected that sex groups may differ in their dynamic processes, for each cohort we ran two RI-CLPM under a multigroup approach were sex was set as the grouping variable as described by Mudler & Hamaker (Mulder and Hamaker, 2020). Model 1 is the *basic multigroup RI-CLPM* where cross-lagged parameters and auto-regressive effects where freely estimated between male and females. The constrained cross-lagged multigroup version were cross-lagged and autoregressive parameters where fixed as equal between male and female, called as Model 2, assumed that the dynamics is similar. Chi-square difference test (Satorra and Bentler, 2010) was used to compare the less restricted model (Model 1) against the more restricted version of multigroup RI-CLPM.

Sensitivity analyses were conducted for black males and females, and for white males and females in CARDIA given its population heterogeneity; here black males and females were run separately via USEOBSERVATION command in Mplus.

Model fit indices were evaluated according to the following fit measures and their cut-offs, as proposed by Schermelleh-Engel (Schermelleh-Engel and Müller, 2003): comparative fit index (CFI), root mean square error approximation (RMSEA), standardized root mean square residual (SRMR), and χ^2 p-value. A RMSEA value equal to or less than 0.05 indicates a good approximate model fit. The p-value for the corresponding test of approximate fit should be equal to or less than 0.05. The CFI value should be greater than or equal to 0.97. Furthermore, an SRMR value greater than 0.05 and less than 0.1 indicates a good model fit. All Mplus syntaxes and unstandardized factor solutions can be found in Supplement 2.

Role of the funding source

The funder of the study had no role in study design, data collection, analysis or interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

3. Results

Table 1 shows the mean, SD, minimum and maximum for each wave across time for the three cohorts. In these analyses, the sample comprised a total 17 840 (54·58% female and 45·42% male) adults followed from 18 to 50 years old. In the CARDIA study, depressive symptoms decreased between age 30 and 40 years and slightly increased between age 45 and 50 years in both sexes, with higher scores at 30 years of age (Table 1). A similar pattern was found in the British cohorts; in males, MI scores were slightly increased (i.e., a decrease in psychological distress) during the first half of male adulthood (between 23 and 33 years old) and were then decreased (i.e., increased symptoms) between 34 and 50 years of age, with less psychological distress in males. Cohorts born more recently tended to have higher psychological distress scores. In the NCDS, participants age 50 years had more symptoms than those age 23 years.

There is a substantial increasing in BMI across time/aging. The increase in BMI in NCDS for men, for example, is as much as 5 points, and people in all cohorts were overweight on average at the last wave. Starting in a normal level in the first wave (BMI = 19·0–24 kg/m2), reaching overweight level by age 30 years (BMI = 25·0 to 29·0 kg/m2) (Table 1) American cohort shows the same BMI progressive growth pattern, but with higher rates in females.

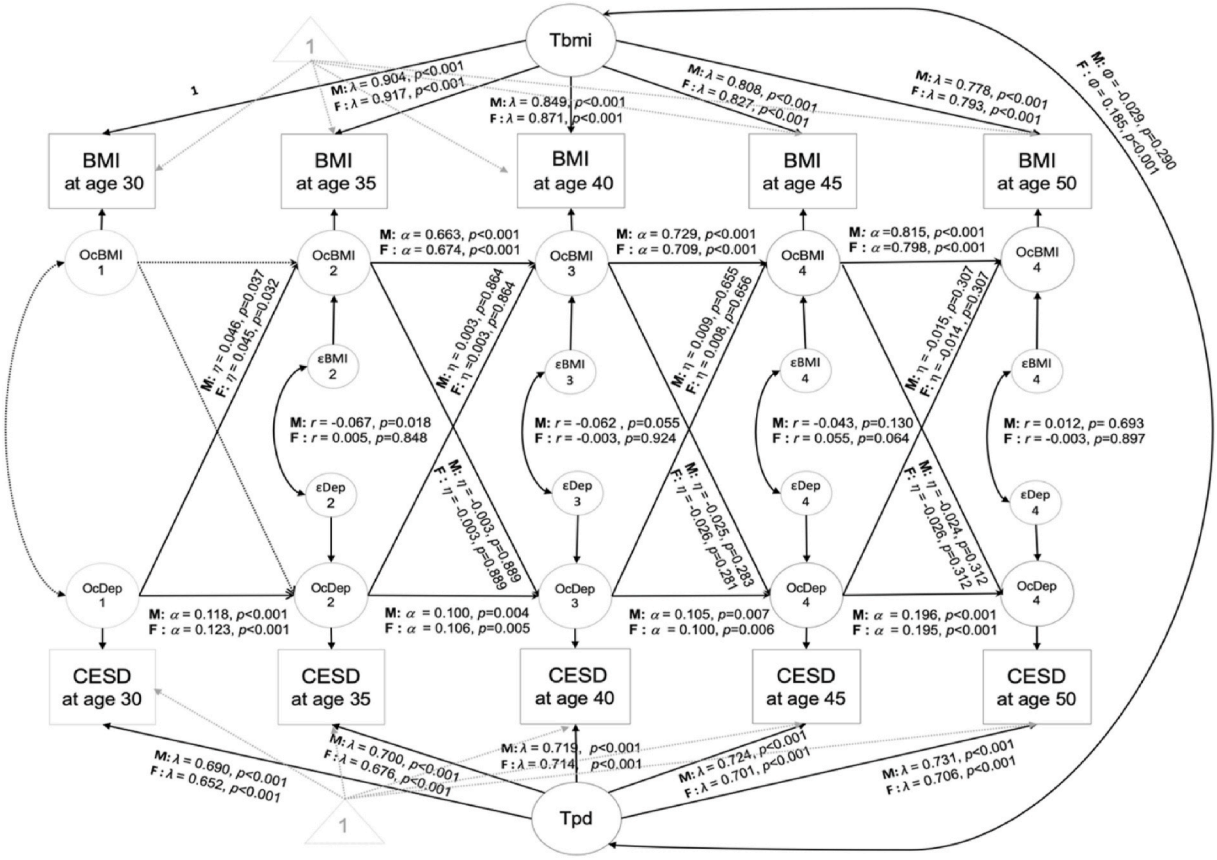

Fig. 3. Parameters for RI-CLPM models of BMI and Depressive Symptoms on CARDIA by sex. Omitted Autoregressive effects between OcBMI1-OcBMI2, Dynamic errors from first occasions and Cross-lagged for OcBMI1-OcDep2; λ = standardized factor loadings; ε = dynamic errors; Dep = Depressive symptoms; OcBMI/OcDep = occasion-specific latent variables; Tbmi/Tdep = trait-like variables; squares = observed variables; α = standardized regression coefficient (autoregressive), η = standardized regression coefficient (cross-lagged); p = p-value; r = correlation coefficient (error); Φ = trait. N = 4724; n_{male} = 2133; n_{female} = 2591. Ages 29.97(SD = 3.64), 35.02 (SD = 3.66), 40.18 (SD = 3.64), 45.21 (SD = 3.63), 50.16 (SD = 3.63).
Note: we fixed the variance of OcBMI1 to 0 to avoid estimation problems. Therefore, the standardized loading is one.

The RI-CLPM returned good fitting models according to all four goodness-of-fit indices (Table 2) and for BCS and CARDIA the Model 2 level of invariance was held, where cross-lagged and autoregressive effects were held equal in male and females, meaning that for BCS and CARDIA cross-lagged and autoregressive effects were invariant over males and females.

Considering the best fit models, Figs. 3–5 show the standardized solution for the auto-regressive and cross-lagged effects estimates, and correlations between traits for NCDS, BCS, and CARDIA, respectively. For BCS and CARDIA, the standardized estimates are derived from the Model 2. Each standardized parameter is reported for male and female. We reiterate that unstandardized paths for BCS and CARDIA are the same for males and females and are only the standardized paths different, as shown in Figs. 4 and 5. In other words, while the unstandardized cross-lagged and autoregressive effects are constrained to be the same across groups, the standardized cross-lagged and autoregressive effects can still be different (Hamaker et al., 2015). Moreover, important so these paths included in Figs. 3–5 are not the same over time (i.e., in BCS, autoregressive from age 26 to age 30, from 30 to age 34, and from 34 to 42 are not constrained to be equal).

In Figs. 3–5, the factor loadings differ over time points and between males and females. Note that the factor loadings in Figs. 3–5 are standardized loadings, and these can differ because the variances of the occasion-specific variables can differ over time points and across males and females.

In terms of between-subject effects Tbmi and TPD/Tdep had small correlations with each other among female participants in the NCDS ($\Phi_{PD} = -0{\cdot}06$, p = 0·01) and CARDIA ($\Phi_{Dep} = 0{\cdot}18$, p < 0·01) but not in the BCS ($\Phi_{PD} = -0{\cdot}03$, p = 0·17). For British cohorts, higher scores on Malaise Inventory indicates less symptoms. Thus, in NCDS, among females, the higher BMI, the more experience psychological distress. In CARDIA, among females, the higher BMI, the more depressive symptoms. However, note that the magnitude of the correlation is either close to zero or very weak.

The within-person cross-lagged paths (standardized regression coefficients [η]) for the three cohorts (Figs. 3–5) had very small magnitudes of effect size, ranging from $\eta = 0{\cdot}00$ (p = 0·99) to $\eta = -0{\cdot}06$ (p < 0·01).

In sensitivity analyses by ethnicity in the CARDIA study, very small cross-lagged effects were observed, ranging from $\eta = 0{\cdot}01$ to $\eta = -0{\cdot}15$ (Table 3). The largest effect sizes for cross-lagged parameters were found among black males, the largest of which was $\eta = -0{\cdot}15$ (p = 0·01).

In the sensitivity analysis, autoregressive effects of BMI increased with age, and these were stronger than the autoregressive effects seen in depressive symptoms, with higher effects in males, as seen in all cohorts for both sexes. In terms of between-subjects effects in the sensitivity analysis for the trait correlations, Tbmi and TPD/Tdep exhibited very small positive correlations among black females ($\Phi_{Dep} = 0{\cdot}09$, p = 0·01) and also white females ($\Phi_{Dep} = 0{\cdot}17$, p < 0·01). Thus, both black and white females with higher BMI are more likely to report depressive symptoms.

A comparison of Model 1 and 2 effects can be found in the Supplementary Material, Table 4.

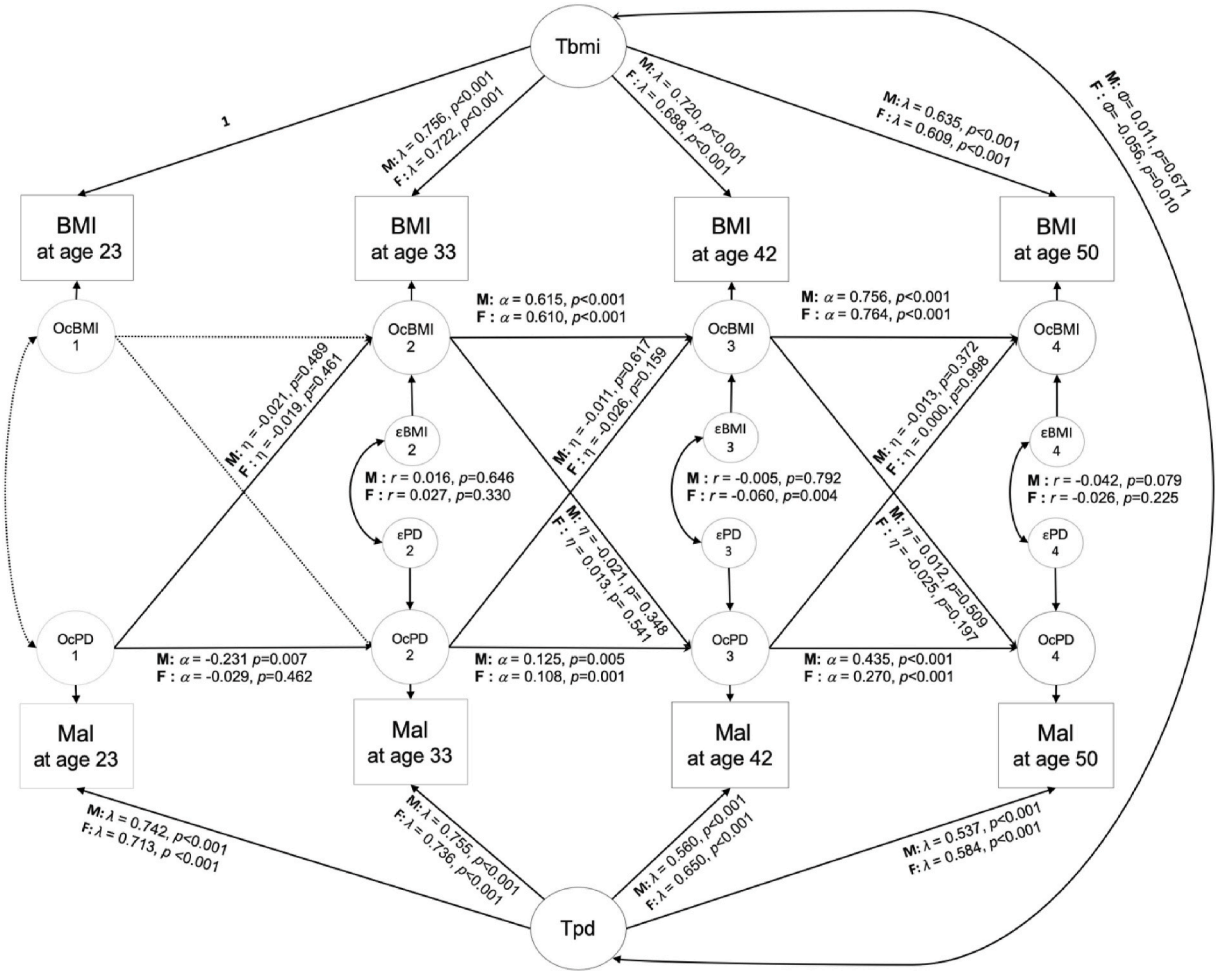

Fig. 4. Parameters for RI-CLPM models of BMI and Depressive Symptoms on NCDS by sex. Omitted Autoregressive effects between OcBMI1-OcBMI2, Dynamics errors from first occasions and Cross-lagged for OcBMI1-OcDep2; μ and λ = factor loadings; ε = dynamics errors; OcBMI/OcPD = occasion-specific latent variables; PD=Psychological Distress symptoms; Tbmi/Tpd = trait-like variables; squares = observed variables; α = standardized regression coefficient; autoregressive), η = standardized regression coefficient (cross-lagged); p = p-value; r = correlation coefficient; Φ = trait. N = 7149; n_{male} = 3378; n_{female} = 3771.
Note: we fixed the variance of OcBMI1 to 0 to avoid estimation problems. Therefore, the standardized loading is one

4. Discussion

We investigated the reciprocal effects of BMI and psychological distress, across three different large cohorts, in two countries spanning two generations. Both BMI and psychological distress were heavily dependent on interindividual traits that persisted throughout adulthood. BMI increased with age/time, where people in all cohorts could be considered almost overweight on average at the last wave in agreement with what has been described in different cohorts (Yang et al., 2021). Moreover, at later time points it was influenced more heavily by occasion-specific effects than by trait effects. BMI trait was weakly correlated (or showed close to zero correlations) with the trait of psychological distress. An increase in the influence of occasion-specific effects at later points was also observed in measures of psychological distress, although occasion-specific psychological symptoms had smaller carryover effects than occasion-specific BMI measures. These strong trait influences, differential autoregressive effects, taken together with good model fit indices, justify the use of the RI-CLPM to disentangle these important sources of information before considering longitudinal dynamics between these measures.

In the CARDIA cohort, we found small correlations between BMI and depressive symptoms at the level of their traits (i.e., between subjects) and generally very small, if any, predictive effects of their occasion-specific variances (i.e., cross-lagged paths). These findings confirm those of a previous study in the same cohort that used a parallel growth curve model (Needham et al., 2010), which also found no evidence to support an influence of initial BMI on depressive symptoms over time; however, the present results are inconsistent with the previous finding of a small but significant effect of depressive symptomatology on the rate of change in BMI among white adults (Patalay and Hardman, 2019). It is possible that estimating and accounting for trait components of BMI and CES-D scores, and their correlations with each other, in the RI-CLPM model, might have partly accounted for the previously observed predictive effects.

The generally weak evidence to support cross-lagged predictions in this study diverges from that of studies reporting longitudinal predictive relationships between psychological symptoms and BMI or vice versa (Luppino et al., 2010). However, among females, individual differences in depressive symptoms in the CARDIA study, and psychological distress in the BCS, were positively correlated with BMI at the level of their traits, consistent with many cross-sectional studies reporting relationships between BMI and depressive symptoms or disorders (Benson et al., 2013; de Wit et al., 2010; Jeffers et al., 2013; Wiltink et al., 2013). Here, these correlations were weak, even without adjusting for potential confounders such as race/ethnicity, socioeconomic status, etc. Therefore, it should be considered that this small relationship at the population level may stem from common underlying interindividual differences that are related to both BMI and psychological distress.

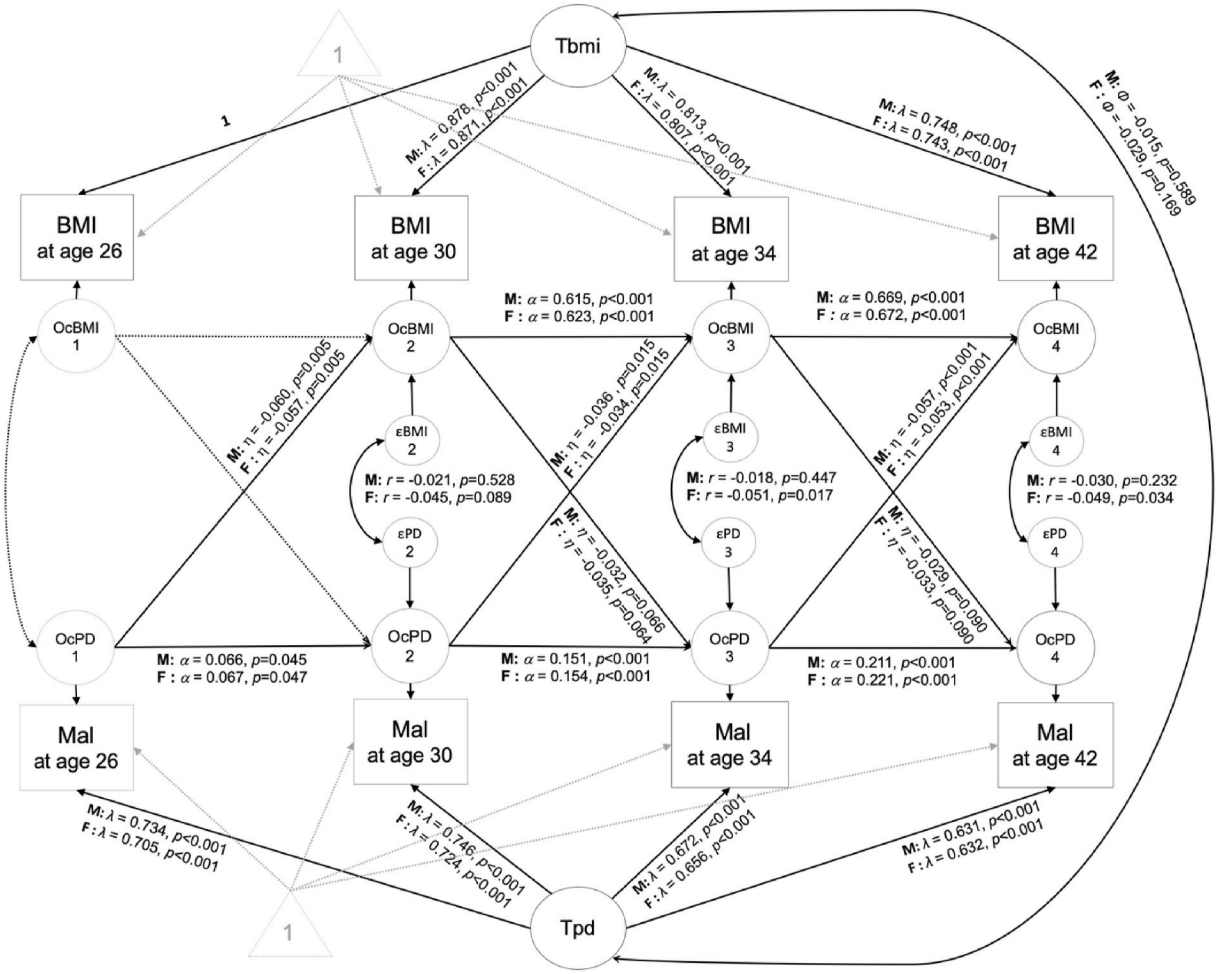

Fig. 5. Parameters for RI-CLPM models of BMI and Depressive Symptoms on BCS by sex. Omitted Autoregressive effects between OcBMI1-OcBMI2, Dynamic errors from first occasions and Cross-lagged for OcBMI1-OcDep2; μ and γ = factor loadings; ε = dynamic errors; BMI=Body Mass Index; Mal = Malaise Inventory; OcBMI/OcPD = occasion-specific latent variables; PD=Psychological Distress symptoms; Tbmi/Tdep = trait-like variables; squares = observed variables; α = standardized regression coefficient (autoregressive), η = standardized regression coefficient (cross-lagged); p = p-value; r = correlation coefficient; Φ = trait. N = 5967; n_{male} = 2582; n_{female} = 3385. Note: we fixed the variance of OcBMI1 to 0 to avoid estimation problems. Therefore, the standardized loading is one

Based on genetic risk, Milaneschi and colleagues (Milaneschi et al., 2017), point out that major depression and obesity-related traits may arise from shared pathophysiologic mechanisms, although their findings would be more consistent with an interaction. The present small correlation at the trait-level (i.e., a predisposition towards both psychological symptoms and BMI) would be consistent with the expected magnitude of a common underlying genetic association producing an effect that is stable over time; however, the results do not preclude non-genetic common elements such as environmental exposures.

By applying random intercepts in the CLPM, we found that autoregressive effects of BMI and psychological distress varied in their magnitudes across time, such that with increasing age BMI became more stable than psychological distress due to carryover effects from one occasion to the next. Given this observation, estimates using statistical approaches such as generalized estimating equations, which are commonly used to track associations between measurements (Hurlbert et al., 2019; Kmetz, 2019) and traditional CLPMs should be interpreted with caution because they do not account for the presence of the trait or for heterogeneity within the trait effects. Considering that the RI-CLPM captures an individual's temporal deviation from their expected scores – rather than from the group means – the RI-CLPM model can assess individual static and dynamic characteristics underlying psychological and physiological features that may go undetected by traditional CLPM. Future studies might evaluate heterogeneity in the interplay between BMI and psychological distress within the RI-CLPM over development and aging. In children and teens, a CLPM was used to examine the interplay between obesity and internalizing mental illness was in the Millennium Cohort Study, highlighting its cross-domain temporal pathways in middle childhood (Patalay and Hardman, 2019); however, that study did not apply a random intercept. In that study, small but significant associations between changes in these measures over time were found only in distinct phases of development, suggesting inconsistent relationships over the human life span.

The reasons for slight inconsistencies in the findings between the two British cohorts are unclear; however, a study using the same British cohorts described differences between them in manifestations of psychological distress. For instance, the cohort born more recently tended to have higher psychological distress scores, with more pronounced differences in males (Ploubidis et al., 2017). It is also notable that the significant predictions had small effect sizes, leading to the same conclusion of weak evidence to support clinically meaningful predictive relationships.

As a limitation of the study, assessment measures and many cultural elements were inconsistent between cohorts, precluding exact replication of findings; however, regardless of these cultural and methodological differences, the results were convergent. As a further limitation, we did not control for or examine potential mediators underlying the associations between weight gain/loss and depressive symptoms, such

Table 3
Parameters for RI-CLPM models of BMI and Depressive Symptoms on CARDIA by ethnicity.

Model/Parameters[b]		Black[a] (n = 2378)						White[a] (n = 2346)					
		Female (n = 1349)			Male (n = 1029)			Female (n = 1242)			Male (n = 1104)		
		Estimate	SE	p	Estimate	SE	p	Estimate	SE	p	Estimate	SE	p
Trait loadings	Tbmi-age 35	0.925	0.005	**<0.001**	0.903	0.018	**<0.001**	0.884	0.013	<0.001	0.903	0.009	<0.001
	Tbmi-age 40	0.879	0.008	**<0.001**	0.856	0.014	**<0.001**	0.827	0.014	<0.001	0.840	0.013	<0.001
	Tbmi-age 45	0.841	0.009	**<0.001**	0.788	0.017	**<0.001**	0.773	0.021	<0.001	0.820	0.013	<0.001
	Tbmi-age 50	0.804	0.010	**<0.001**	0.762	0.015	**<0.001**	0.747	0.016	<0.001	0.779	0.013	<0.001
	Tdep-age 30	0.673	0.017	**<0.001**	0.682	0.022	**<0.001**	0.618	0.023	<0.001	0.668	0.023	<0.001
	Tdep-age 35	0.676	0.023	**<0.001**	0.654	0.028	**<0.001**	0.684	0.026	<0.001	0.708	0.025	<0.001
	Tdep-age 40	0.723	0.023	**<0.001**	0.713	0.029	**<0.001**	0.685	0.030	<0.001	0.703	0.025	<0.001
	Tdep-age 45	0.714	0.025	**<0.001**	0.701	0.027	**<0.001**	0.671	0.026	<0.001	0.713	0.025	<0.001
	Tdep-age 50	0.728	0.024	**<0.001**	0.709	0.028	**<0.001**	0.664	0.028	<0.001	0.717	0.022	<0.001
	Tbmi-Tdep correlation	0.093	0.035	**0.008**	−0.082	0.041	**0.044**	0.167	0.043	<0.001	0.011	0.039	0.770
Autoregressive	OcBMI2-OcBMI3	0.627	0.030	**<0.001**	0.711	0.044	**<0.001**	0.735	0.030	<0.001	0.589	0.039	<0.001
	OcBMI3-OcBMI4	0.719	0.034	**<0.001**	0.805	0.026	**<0.001**	0.684	0.058	<0.001	0.643	0.045	<0.001
	OcBMI4-OcBMI5	0.779	0.026	**<0.001**	0.853	0.020	**<0.001**	0.799	0.040	<0.001	0.774	0.024	<0.001
	OcDep1-OcDep2	0.057	0.052	0.272	0.197	0.057	**0.001**	0.032	0.047	0.492	0.206	0.053	<0.001
	OcDep2-OcDep3	0.161	0.066	**0.015**	0.026	0.070	0.713	0.017	0.076	0.823	0.143	0.069	0.038
	OcDep3-OcDep4	0.061	0.071	0.392	0.106	0.081	0.188	0.105	0.076	0.166	0.133	0.076	0.081
	OcDep4-OcDep5	0.102	0.064	0.111	0.290	0.075	**<0.001**	0.231	0.055	<0.001	0.197	0.067	0.003
Cross-lagged	OcDep1-OcBMI2	0.032	0.037	0.396	0.046	0.047	0.329	0.008	0.042	0.843	0.054	0.045	0.222
	OcDep2-OcBMI3	−0.009	0.031	0.765	−0.018	0.037	0.638	0.017	0.030	0.569	0.006	0.042	0.883
	OcDep3-OcBMI4	0.056	0.032	0.078	−0.017	0.036	0.648	−0.025	0.044	0.574	0.017	0.039	0.666
	OcDep4-OcBMI5	−0.041	0.024	0.086	0.007	0.031	0.813	0.010	0.030	0.746	−0.019	0.032	0.555
	OcBMI2-OcDep3	−0.032	0.041	0.438	−0.139	0.061	**0.024**	0.017	0.052	0.751	0.079	0.041	0.052
	OcBMI3-OcDep4	−0.076	0.042	0.067	−0.081	0.056	0.145	0.019	0.049	0.700	0.013	0.048	0.792
	OcBMI4-OcDep5	−0.044	0.052	0.398	−0.147	0.058	**0.011**	0.020	0.039	0.608	0.054	0.045	0.233
Measurement errors	εBMI2-εDep2	−0.024	0.042	0.563	−0.128	0.041	**0.002**	0.010	0.041	0.797	−0.011	0.041	0.789
	εBMI3-εDep3	−0.009	0.043	0.828	−0.122	0.046	**0.009**	−0.010	0.043	0.822	−0.014	0.051	0.781
	εBMI4-εDep4	0.014	0.048	0.763	−0.096	0.046	**0.037**	0.110	0.036	0.003	−0.007	0.041	0.861
	εBMI5-εDep5	−0.067	0.037	0.067	0.008	0.047	0.869	0.066	0.042	0.112	0.036	0.042	0.392

CARDIA=Coronary Artery Risk Development in Young Adults; CES-D = Center for Epidemiologic Studies scale; SE = standard error.
[a] Not Hispanic.
[b] Trait factor loadings form first occasions constrained to 1; omitted Cross-lagged for OcBMI1-OcDep2 and Autoregressive OcBMI1-OcBMI2; standardized estimates statistically significant at $p < 0.05$ are in bold.

as use of antidepressants or other medications that could interfere with metabolism and consequent accumulation or loss of fat. Unmeasured confounding can be a problem in RI-CLPM (or any model) too, especially considering time varying confounding as for example, smoking, but since the results showed no or/very weak associations between BMI and depression/psychological distress, we believe that our lack of adjustment is unlikely to change the main conclusions even under negative confounding/suppression effects (MacKinnon et al., 2000). Furthermore, the associations between depression and BMI could be different between subgroups, for example, smokers, or patients with a diagnosis of major depression (Milaneschi et al., 2017), which is beyond the scope of this work and which might be addressed in future works.

The RI-CLPM did not constrain BMI and depression/psychological distress means over time and consequently the average trajectory was not assumed to take on any shape (see Usami et al., 2019). Inspecting the BMI means across time, there is a growth pattern, although it is not necessarily linear. For depression/psychological distress, there is no clear trend. We were not able to fit adequately cross-lagged models that explicitly estimate the development of BMI trajectories as, for example, the latent change score (see Hamagami and J. McArdle, 2001; McArdle and Hamagami, 2001) where linear trends in the BMI mean structure would be estimated (data not shown). Under RI-CLPM, we verified that the two measures are heavily influenced by traits; therefore, regardless of temporal mean-level trends, within-subjects changes in terms of cross-lagged parameters, which were shown to have very weak effect sizes, are able to explain very little of the total variance.

Although the RI-CLPM brings us closer to understanding the natural course of adult psychological symptoms and BMI, with less temporal bias compared with traditional CLPMs (Hamaker et al., 2015), clinical diagnostic tools were not used, precluding examination of specific depressive or anxiety disorders. Similarly, we examined BMI as the most robust and common epidemiological measure related to obesity, to connect the present findings with the most abundant literature; however, BMI may be less sensitive to some distress-related anthropometric changes, such as body fat percentage, or waist circumference.

Although associations between BMI and psychological distress have been reported, previous studies have faced difficulties disentangling state and trait features when considering the dynamics between these characteristics. In three cohorts collected on two continents across two centuries, both BMI and psychological distress measurements were heavily dependent on enduring interindividual traits, which correlated weakly with each other among females. Evidence to support meaningful predictive effects of psychological distress on subsequent changes in BMI, or vice versa, within individuals over time was weak and inconsistent at the population level.

CRediT authorship contribution statement

B.S. Scarpato: Project administration, Formal analysis, Visualization, Writing – original draft. **W. Swardfager:** Conceptualization, Methodology, Writing – review & editing. **M. Eid:** Formal analysis, Methodology. **G.B. Ploubidis:** Data curation, Writing – original draft. **B. J. MacIntosh:** Validation. **C.Y. Wu:** Writing – original draft, Writing – review & editing. **L.J. Launer:** Formal analysis, Conceptualization, Project administration, Supervision, Data curation, Writing – original draft. **H. Cogo-Moreira:** Writing – original draft, Writing – review & editing.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2021.10.030.

Declaration of interest

The authors declare no conflict of interest.

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