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POPULATION STUDY ARTICLE

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Early life adversity and obesity risk in adolescence: a 9-year population-based prospective cohort study

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BACKGROUND: There are few prospective studies of factors that mediate the association between exposure to adverse childhood experiences (ACEs) and obesity in adolescence. Our aim was to address this limitation.
METHODS: We used prospective data from the Growing up in Ireland cohort study, with measurements at 9, 13, and 18 years old. The exposures were 14 adverse experiences before age 9. The main outcome was body mass index (BMI) at 18 years. Mediators were daily activity, diet quality, self-image and behavioural difficulties at 13 years.
RESULTS: Among the 4561 adolescents in the final cohort, 77.2% experienced any adversity, 50.5% were female and 26.7% were overweight/obese at 18 years. BMI Z was higher at ages 9 (0.54 vs 0.43, $p < 0.05$, 95% CI of difference: −0.22, −0.01) and 13 years (0.50 vs 0.35, $p < 0.05$, 95% CI of difference: −0.25, −0.06), in those exposed to an ACE, compared to those unexposed. Structural equation models revealed that behavioural difficulties ($\beta = 0.01$; 95% CI: 0.007–0.018, $p < 0.001$) and self-concept ($\beta = 0.0027$; 95% CI: 0.0004–0.0050, $p = 0.026$) indirectly mediate the association between exposure to ACEs and BMI at 18 years.
CONCLUSIONS: The association between ACEs and BMI in adolescence is mediated by behavioural difficulties and self-concept.

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IMPACT:

- In a previous study, we found modest associations between exposure to a range of adverse childhood experiences and weight gain at 13 years of age.
- The strength of the association between adverse childhood experiences and weight gain was lower at 18 years of age compared to the association observed at 13 years and was no longer significant after controlling for confounding and including possible mediators.
- The association between adverse childhood experiences and BMI in adolescence is indirectly mediated by behavioural difficulties and self-concept.

INTRODUCTION

The prevalence of obesity among adolescents in westernized countries is between 20 and 30% and has increased significantly over time.[1,2] Treatment and prevention of obesity in adolescence is challenging due to a range of biological, social and psychological factors.[2] Treatment and prevention efforts are important, as living with obesity in adolescence is associated with increased risk of type 2 diabetes, hypertension, coronary heart disease, different types of cancers, and premature mortality.[3,4] The determinants of obesity are complex and involve the interplay between environmental exposures, biologically inherited risk and psychological factors that influence health behaviours.[5,6] Exposure to adverse experiences in childhood are involved in this complexity as they are consistently associated with weight gain and obesity in adolescents.[7–10] The factors mediating this association have yet to be determined empirically.[11]

Adverse childhood experiences (ACEs) are harms that can affect children directly or indirectly by disrupting their sense of safety.[12] The original ACEs study focused on 10 experiences including physical, sexual and emotional abuse, physical and emotional neglect, caregiver mental illness, substance abuse and incarceration, mother treated violently and divorce.[12] Several paediatric observational studies demonstrated that exposure to a range of adverse experiences, including ACEs as well as others not included in the ACEs study (poverty, child apprehension, loss of a parent) in early childhood is associated with obesity in adolescence[13–17] and greater weight gain throughout adolescence.[13,18] The mechanisms or behaviours mediating the association between adverse experiences in childhood and obesity in adolescence are unclear. Given the robust association between exposure to adverse experiences in childhood and negative mental health outcomes[19] and high-risk behaviours,[20,21] and growing evidence that mental

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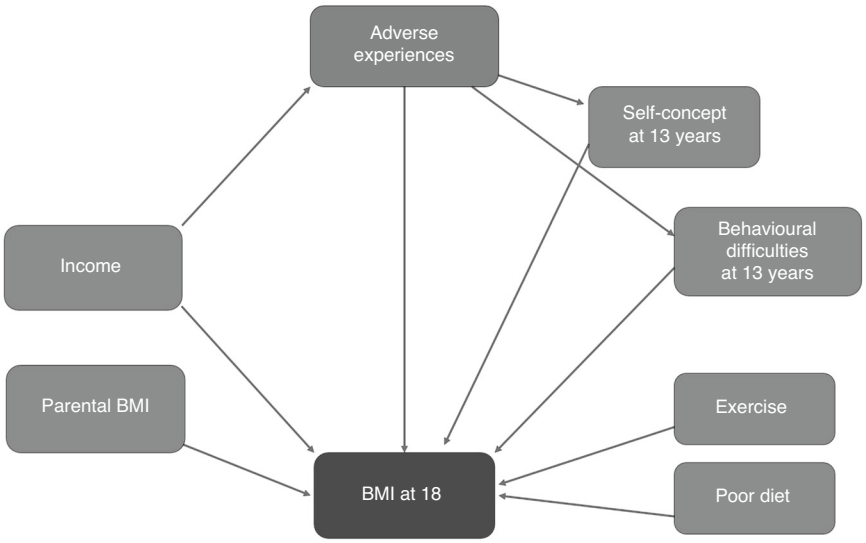

Fig. 1 Directed acyclic graph (DAG) describing possible pathways through which exposure to an adverse childhood experience may lead to a higher BMI at 18 years of age.

Table 1. Exposure to various types of adversity before 9 years old stratified by weight status at 18 years of age.

	Entire sample ($n = 4561$)	Healthy weight ($n = 3344$)	Overweight/obese ($n = 1217$)	*p* value
Death of a parent	1.4% (1.0–1.7)	1.4% (1.0–1.7)	1.5% (0.8–2.2)	0.733
Death of close family member	42.5% (41.0–43.9)	41.6% (40.0–43.3)	44.8% (42.0–47.6)	0.057
Death of close friend	5.6% (5.0–6.3)	5.4% (4.6–6.1)	6.4% (5.0–7.8)	0.171
Moving house	41.0% (39.6–42.4)	41.8% (40.1–43.5)	38.8% (36.0–41.5)	0.066
Moving country	9.8% (9.0–10.7)	10.2% (9.2–11.2)	8.9% (7.3–10.5)	0.185
Stay in foster home	0.7% (0.4–0.9)	0.7% (0.4–0.9)	0.7% (0.2–1.1)	0.998
Serious illness/injury	4.6% (4.0–5.2)	4.4% (3.7–5.1)	5.1% (3.9–6.3)	0.341
Serious illness/injury of a family member	14.4% (13.4–15.5)	14.1% (12.9–15.2)	15.5% (13.5–17.6)	0.210
Conflict between parents	9.4% (8.6–10.3)	9.2% (8.2–10.1)	10.2% (8.5–11.9)	0.289
Other disturbing event	1.9% (1.5–2.3)	1.8% (1.3–2.2)	2.1% (1.3–2.9)	0.566
ACE-specific events				
Parent in prison	0.4% (0.3–0.6)	0.4% (0.2–0.6)	0.5% (0.1–0.9)	0.737
Mental disorder in immediate family	2.8% (2.3–3.3)	2.8% (2.2–3.3)	2.9% (1.9–3.8)	0.821
Divorce/separation of parents	9.4% (8.5–10.2)	8.8% (7.8–9.7)	11.0% (9.3–12.8)	0.021
Drug taking/alcoholism in immediate family	2.2% (1.8–2.6)	2.3% (1.8–2.8)	1.9% (1.1–2.7)	0.433
Any adversity	77.2% (76–78)	76.6% (75–78)	78.6% (76–81)	0.156

health challenges are common among adolescents living with obesity,[22–24] it is possible that psychological or emotional factors could mediate the associations between adverse experiences in childhood and obesity risk in adolescence. Currently, there are no prospective studies examining the potential role of psychological or emotional factors mediating the association between exposure to adverse experiences in childhood and obesity in adolescence. Understanding the biopsychosocial factors that mediate the association between exposure to adverse experiences in childhood and obesity risk in adolescence is important for the scientific understanding of the causal determinants of adolescent obesity and clinically, this information could inform future intervention strategies.

To overcome the lack of evidence for possible mediators of the association between exposure to adverse experiences in childhood and obesity risk in adolescence, we analysed data from a large population-based cohort of children followed prospectively for 9 years to test the hypothesis that exposure to adverse experiences in childhood before 9 years of age is associated with higher BMI at 18 years of age and that this association would be partially mediated by psychological and emotional factors (Fig. 1).

DESIGN AND METHODS

Study population, design and sampling

The Growing up in Ireland Child Cohort enrolled 8568 9-year-old children born between 1 November 1997 and 31 October 1998 and their families into a prospective longitudinal cohort study originally designed to follow children at 13 and 18 years of age.[25,26] These children were sampled from the 56,497 9-year-old children residing in the Republic of Ireland according to the 2006 National Census (wave 1). Recruitment and data collection for the cohort occurred when the children were 13 years of age (wave 2) between 2012 and 2013 and again when they were 18 years of

age (wave 3), between 2015 and 2016. 6216 families completed the survey with 6039 having data in all three waves. There were no exclusion criteria for entry into the cohort study. Parents consented and the children assented to be included in the study during home visits. All stages of the Growing Up in Ireland project were approved by the Health Research Board's standing Research Ethics Committee based in Dublin, Ireland. Data were accessed via the Irish Social Science Data Archive (www.ucd.ie/issda).

Primary exposure variable

The primary exposures of interest were adverse experiences in childhood that occurred before 9 years of age. We studied two types of exposures, each treated as a binary variable. First, we defined exposure to any adversity as exposure to at least one of 14 adverse experiences reported by the primary caregiver during wave 1 of the study, that the child was exposed to before 9 years of age (Table 1).[13] Second, we restricted the exposure to at least one of the four of adverse experiences, included in the original ACEs study; divorce, parent in prison, parent with drug/alcohol abuse, and parent with a mental health disorder.[12,27] Children exposed to at least one of these four adverse experiences are classified as being exposed to an ACE. The other ten experiences, death of a parent, death of a close family member, death of a close friend, moving house, moving country, a stay in a foster home, serious illness for the child, serious illness for a family member, conflict between parents, and "other disturbing events", were adapted from the National Longitudinal Survey of Children and Youth.[25,28] Children whose parent reported "none" were treated as unexposed to any adversity.

Outcome variable of interest

The outcome measure was BMI, treated as a continuous variable (kg/m^2). Each young person's height and weight were measured objectively by a trained interviewer in the adolescent's home at 9, 13 and 18 years of age. BMI classifications of non-overweight, overweight, or obese, at age 9 and 13 was determined using age and sex specific *Z* scores developed from international data by the World Obesity Federation.[29,30]

Mediating variables of interest

Behavioural difficulties were measured using the strengths and difficulties questionnaire (SDQ)[31,32] and was completed by the primary caregiver when the child was 9, 13 and 18 years old. The SDQ has 5 subscales including 4 areas of difficulties: emotional, conduct, hyperactivity, peer problems, as well as a prosocial subscale.[31,32] Subscales were used to calculate a total SDQ score in which a higher total score indicated more behavioural difficulties. For the main structural equation model, we used SDQ at age 13 years as a mediator. SDQ at age 9 and 18 years were used to describe differences in behavioural difficulties throughout the 9-year follow-up period between children differentially exposed to any adverse experience.

Self-concept was measured using the Piers–Harris questionnaire (PH)[33] that was completed by the child at 9 and 13 years old. The PH questionnaire is a 60-item self-reported questionnaire that assesses self-concept through six different subscales: behavioural adjustment, intellectual and school status, physical appearance and attributes, freedom from anxiety, popularity, and happiness and satisfaction.[33] A higher score on the PH indicated a higher sense of self-concept. For our analyses we used PH at 13 years as a mediator.

Covariates and confounding variables

Potential confounders and covariates collected when the child was 18 years old were used in structural equation models and were chosen based on previously documented associations with weight status in childhood or adolescence.[34–36] Potential confounders of the relationship between adverse childhood event and BMI included household income and household social class. Household income was self-reported and based off the income of all household members and was equivalized. Other covariates included primary caregiver BMI. Primary caregiver BMI was measured objectively during the home visit that occurred when the adolescent was 18 years of age.

Self-reported dietary habits and physical activity were measured at 9, 13 and 18 years of age. As we have done previously,[13] the consumption of non-diet soda in the past 24 h at 13 and 18 years was used as a proxy for diet. A diet quality score was calculated based on a previous study.[37] Questions about how often the adolescent had consumed a type of food in the past 24 h were used. For foods classified as healthy, a score of 2 was given if consumed more than once, 1 if consumed once, and 0 if not consumed at all. Foods classified as unhealthy, were given a score of −2 if consumed more than once, −1 if consumed once, and 0 if not consumed at all. The scores of each of the different food categories were then summed together in order to calculate the total diet quality score.

Additionally, at ages 13 and 18 years, adolescents reported the number of times in the last 14 days that they accumulated at least 20 min of exercise, which was used as a proxy for habitual physical activity.

Statistical methods

A series of linear mixed effects models were used to test for differences in household income, parental BMI, self-concept, behavioural difficulties, and BMI at age 9, 13 and 18 years, between children exposed to any adverse experience or ACE and those who had not.

Our mediation hypotheses were tested with a structural equation model (SEM). Prior to conducting the structural equation model, we first investigated the measurement (latent) sub-model by performing a series exploratory and confirmatory factor analyses. The goal was to evaluate the psychometric properties of the "self-concept" and "behavioural difficulty" instruments in our sample, as determined from the Piers Harris and Strengths and Difficulties Questionnaire respectively. We used parallel analysis and scree plots to determine the dimensionality of each scale. Upon finding evidence for unidimensionality in each case, i.e. a single latent construct, we then obtained fit statistics (RMSEA, CFI, and SRMR) and modification indices from a confirmatory factor analysis model. We added residual covariance between items as necessary to obtain good model fit (RMSEA ≤ 0.08, CFI ≥ 0.95, SRMR ≤ 0.06),[38] where theoretically plausible. Thus, the confirmatory model was partially exploratory in nature. Results from these factor analyses are provided in the appendix.

The variables included in the full SEM model were determined from our conceptual model for the causal pathways between adverse childhood experiences and obesity in adolescence (Fig. 1). We assumed that income was a common cause of adverse childhood experiences and BMI at all three time points. Finally, mediation effects were calculated for the indirect and total association between exposure to any adverse experience, self-concept, behavioural difficulties, and BMI at 18 years of age. The magnitude of the mediation is calculated as the product of the estimated standardized paths between an exposure (any adversity/ACE) and a mediator, and a mediator and an outcome (BMI). The total effect of any adversity or ACE on BMI is calculated as the sum of the direct effect and two mediated effects. These estimates yield valid causal estimates in the absence of unmeasured confounding and interaction, and if the assumption of linear relationships is correct.[39] Separate structural equation models were conducted for exposure to any adverse experiences before 9 years of age and ACE-specific exposures. Diagonally-weighted least squares was used to estimate the SEM since adverse experience is endogenous in our model and was treated as a binary variable. Statistical significance was set at a two-tailed

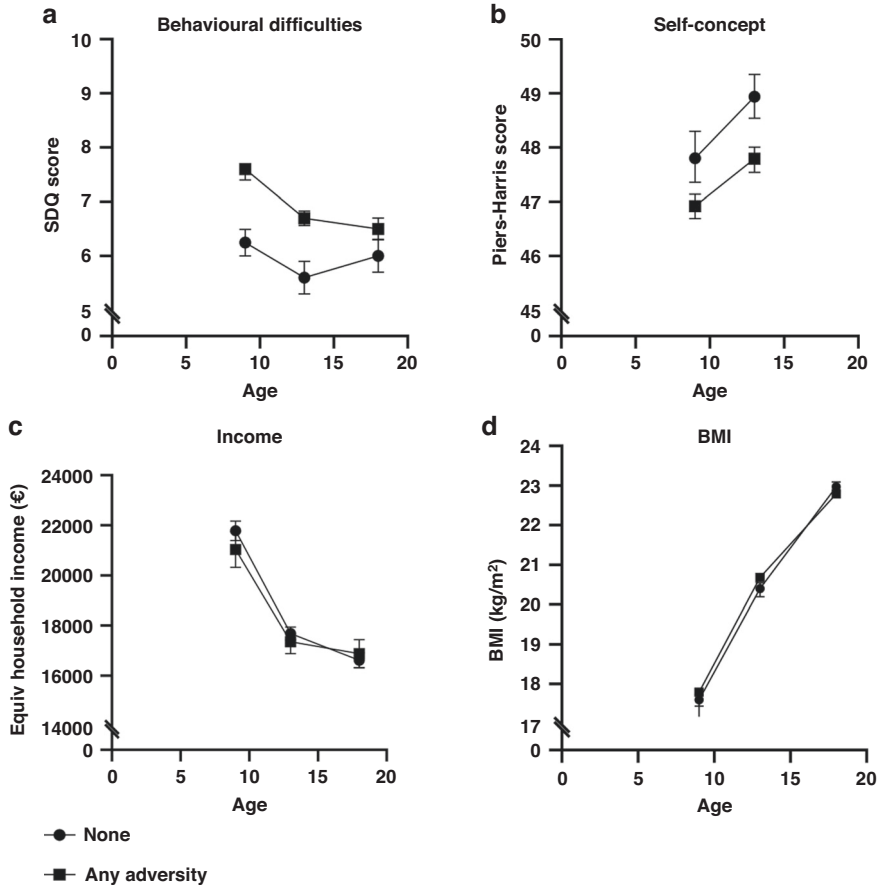

Fig. 2 Biopsychosocial variables from age 9 to 18 years among youth stratified according to exposure to adversity in childhood. Behavioural difficulties were measured using the Strengths and Difficulties Questionnaire (SDQ) and self-concept was measured using the Piers–Harris questionnaire. Differences in the changes in **a** behavioural difficulties; **b** self-concept; **c** household income (per person) and **d** (body mass index (BMI) for children exposed and unexposed to any adverse experience in childhood.

$\alpha = 0.05$. The R package lavaan[40] was used for all analyses on R version 3.0 (www.R-project.org). The R code used for all analyses is provided in the appendix.

RESULTS

A flow chart describing how we arrived at the final sample size is provided in sFigure 1 of the appendix. Of the original cohort of 8568 9-year-old children and their primary caregivers, 7423 pairs returned for follow-up (87% retention) in 2011 when children were aged 13 years. Of those who returned for follow-up at age 13, 6942 had complete data. In 2017, 6216 children returned for follow-up when they were the age of 18, and 1655 of these children did not have complete data. Compared to those excluded, adolescents included in these analyses had a higher mean household equivalized income at 18 years than those who did not (16,700€/person vs 15,600€/person), had similar BMIs at 18 years (22.91 kg/m^2 vs 23.08 kg/m^2), and had similar primary caregiver BMI at 18 years (26.76 kg/m^2 vs 26.68 kg/m^2) (sTable 1).

Compared to non-overweight adolescents, those living with obesity at 18 years, were more likely to have experienced the death of a family member, moving house and separation or divorce of parents (Table 1). Characteristics of adolescents who were exposed to any adversity and those who were not are provided in sTable 2. Trajectories for BMI, self-concept, behavioural difficulties, and household income between 9, 13 and 18 years of age for those exposed to any adverse experience and those unexposed to are provided in Fig. 2a–d. Compared to children that were not exposed to any adverse experience, those exposed to any adverse experience displayed a lower self-concept and higher behavioural difficulty scores throughout adolescence (Fig. 2a, b). Children exposed to any adverse experience also lived in households with lower income throughout adolescence compared to those not exposed to any adverse experience (Fig. 2c) and higher BMI at age 9 and 13 years (Fig. 2d). Differences in behavioural difficulties, BMI and household income between exposed and unexposed children were greatest at 13 years of age, with effect sizes diminishing by 18 years of age. The differences in behavioural difficulties, self-concept, income and BMI were similar when analyses were repeated for children exposed to only to the four ACE specific exposures, compared children exposed to no adverse experiences (sFigure 2).

Standardized path coefficients from the structural equation modelling of BMI at 18 years for any adverse experience in childhood and ACES are provided in Table 2 and sTable 3 respectively. In the full model, we found that exposure to any adverse experience was associated with lower self-concept ($\beta = -0.097$; 95% CI: -0.12 to -0.073) and greater behavioural difficulties ($\beta = +0.17$; 95% CI: $+0.14$ to $+0.21$) at age 13. These effect estimates and confidence intervals were nearly identical when models were repeated for exposure to an ACE-specific experience (sTable 3). Standard path coefficients also revealed that higher behavioural difficulties ($\beta = +0.066$; 95% CI: $+0.040$ to $+0.094$) and a lower self-concept ($\beta = -0.027$; 95% CI: -0.050 to -0.004) at 13 years old were both associated with an increase in BMI at 18 years. A higher parental BMI ($\beta = 0.27$; 95% CI: $+0.24$ to $+0.30$) and lower household income ($\beta = -0.05$; 95% CI: -0.079 to -0.014) when the adolescent was 18 years old were also both

Table 2. Standardized path coefficients and estimated correlations for specific associations within the structural equation modelling of BMI at 18 years for any adverse experience in childhood using a diet quality score as a measure of diet.

Outcome	Predictor	*β* (95% CI)
Self-concept	Any adversity	−0.095 (−0.120, −0.071)***
Behavioural difficulties	Any adversity	0.174 (0.141, 0.207)***
Any adversity	Income at 18	−0.095 (−0.136, −0.054)***
BMI at 18	Any adversity	0.013 (−0.030, 0.055)
	Self-concept	−0.029 (−0.052, −0.005)*
	Behavioural difficulties	0.070 (0.04, 0.100)***
	Income at 18	−0.047 (−0.079, −0.014)**
	Parent BMI at 18	0.267 (0.238, 0.296)***
	Exercise at 13	−0.065 (−0.096, −0.034)***
	Diet Quality Score at 13	0.006 (−0.024, 0.037)
Variable 1	**Variable 2**	***R*** **(95% CI)**
Self-Concept Popularity Subscale	Behavioural Difficulties Peer Subscale	−0.347 (−0.390, −0.305)***
Self-Concept Intellectual Subscale	Self-Concept Behavioural Adjustment Subscale	0.440 (0.386, 0.494)***
Behavioural Difficulties Emotional Subscale	Behavioural Difficulties Prosocial Subscale	0.295 (0.239, 0.351)***
Income at 18	Parent BMI at 18	−0.124 (−0.153, −0.095)***
Self-concept	Behavioural difficulties	−0.299 (−0.314, −0.284)***
Self-concept	Exercise at 13	0.259 (0.241, 0.278)***
Behavioural difficulties	Diet Quality Score at 13	−0.240 (−0.263, −0.217)***

SEM fit statistics: CFI = 0.904, RMSEA = 0.074, SRMR = 0.062.
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

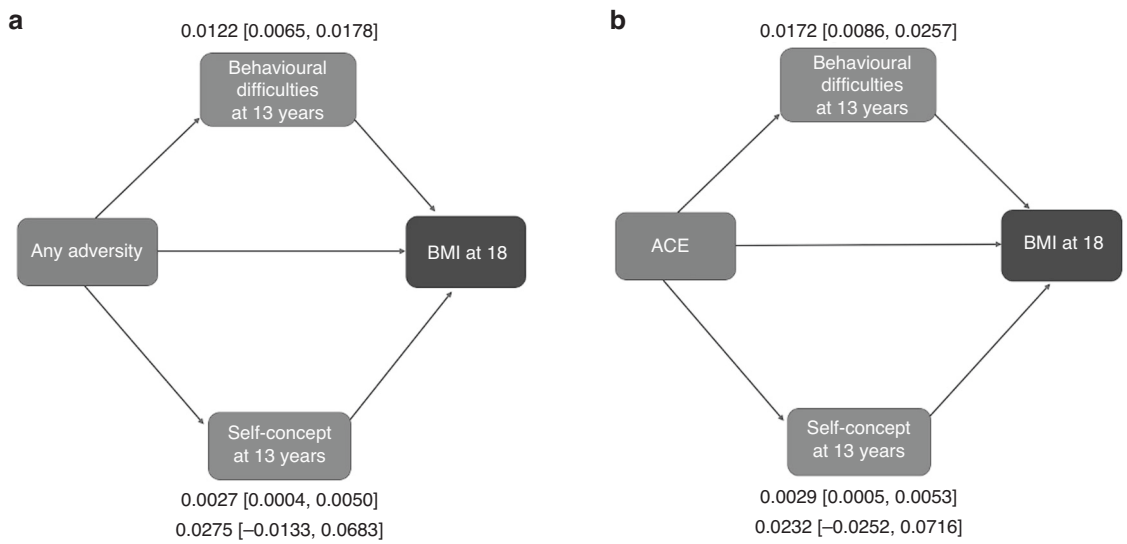

Fig. 3 Standardized estimates of mediation paths for indirect and total relationships between adverse experiences and BMI. **a** Standardized path coefficients for the expsoure to any adverse experiences, potential mediators and BMI at 18 years. **b** Standardized path coefficients for the expsoure to adverse childhood experiences (ACEs), potential mediators and BMI at 18 years.

associated with a higher child BMI at 18 years. Frequency of hard exercise was also associated with a lower BMI at age 18 years ($\beta = -0.066$; 95% CI: −0.097 to −0.035). Neither exposure to any adverse experience before 9 years of age, or diet quality score were directly associated with BMI at 18 years. Standard path coefficients between exposure to ACEs or any adverse experiences and behavioural difficulties were similar when daily soda intake was used in place of the diet quality score (sTables 4 and 5).

The standardized estimates of the mediation paths using self-concept and behavioural difficulties as mediators are presented in Fig. 3a, b. The mediation paths revealed that lower self-concept and increasing behavioural difficulties were both indirectly associated with BMI at 18 years of age, after exposure to either any adverse experience or an ACE. Neither exposure to an ACE or exposure to any adverse experience was directly associated with BMI at 18 years of age.

DISCUSSION

The original aim of this study was to determine if psychological and emotional factors mediate the association between exposures to adverse experiences in childhood and obesity risk in adolescence. To address this aim, e used a large, population-based cohort study with 9 years of follow-up data. We found that

exposure to adverse childhood experiences before 9 years of age was associated with increased behavioural difficulties, lower self-concept, and an elevated BMI at 13 years of age. In contrast to our conceptual causal model (Fig. 1), exposure to an ACE or any adverse experience was not directly associated with BMI at 18 years of age. Differences in weight status and behavioural difficulties observed at 13 years were no longer evident at 18 years of age. Using structural equation models, we found that lower self-concept and higher behavioural difficulties at 13 years of age, indirectly mediate the association between exposure to both ACEs and any adverse childhood experiences and BMI at 18 years of age. Future studies are needed with repeated measures of exposures to advance our understanding of the association between exposure to adverse experiences in childhood and obesity risk in adolescence.

Adolescent obesity is a complex chronic disease and the natural history includes several bio-psycho-social risk factors,[5] including exposure to adverse experiences in childhood.[9,13] In a recent systematic review, 11 of 12 prospective cohort studies published to date found consistent but modest associations between exposure to a variety of adverse experiences in childhood and obesity in adolescence.[9] Previous work by our group, using data from the same cohort, found that exposure to any adverse childhood experience was associated with increased weight gain and incident overweight/obesity at 13 years of age.[13] Most of these studies used multivariate logistic regressions and/or mixed modelling in their analyses[9] and only one of these studies examined possible mediators for this association.[14] In that study, the relationship between a child's long-term exposure to maternal depressive symptoms from the age of 1 month until sixth grade was associated with an elevated BMI at 15 years old, and this association was significantly mediated by adolescent's experience of depressive symptoms at 15 years old.[14] The data presented here do not support previous work by our group and others as we did not observe a direct association between exposure to any adverse experiences and BMI at 18 years of age. Rather, we found that behavioural difficulties and low self-concept indirectly mediate the association between exposure to adverse childhood experiences before 9 years of age and BMI at 18 years. These data provide insight into the potential role of psychological and emotional challenges in pathophysiology of obesity in late adolescence following exposure to adverse experiences in childhood.

Life course theory argues that exposures early in life influence health trajectories over an individual's life.[41] Previous studies of exposure to adverse experiences in childhood and obesity risk rely on retrospective cohort designs, cross sectional studies, or prospective cohort studies with limited measurements throughout adolescence.[7,9] To the best of our knowledge, none of the previous cohort studies in this area described trends in weight status and its determinants over time. The current study builds on our previous work[13] and provides novel insight into the life course effects of exposure to adverse experiences in childhood early in life, and long-term risk for obesity. Specifically, while we found modest associations between exposure to a range of adverse experiences in childhood and weight gain at 13 years of age, the strength of this association was lower at 18 years of age and was no longer significant after controlling for confounding and including possible mediators. Similarly, differences in behavioural difficulties and household income observed at 13 years of age were no longer different at 18 years of age. These data provide evidence that exposure to any adverse experiences or ACEs do not directly cause obesity in late adolescence but influence more proximal behaviours that influence weight gain. Additionally, these data question the deterministic viewpoint that exposure to any adverse experiences or ACEs have lifelong influences on obesity-related health outcomes.

The study is strengthened by the application of a robust analytical approach to a large, prospective cohort of children followed for nearly a decade. Despite these strengths, there are several limitations that need to be addressed. First, there was significant loss of follow-up with each wave, and similar to other cohorts,[7,9] loss to follow-up was not random, as more affluent families remained in the study. This loss to follow-up limits the generalizability of the study findings and may also introduce selection bias. Another limitation to our study is that not all of the ACEs from the original ACEs study[12] were included in the original wave of data collection (i.e. sexual abuse). Some studies looking at the impact of ACEs on obesity have found an effect with some of the ACEs not looked at in this study, such as sexual and physical abuse.[42–45] The lack of information on more severe forms of adverse experiences in childhood in our study may have reduced the strength of the associations observed here. Additionally, information on exposure to an ACE was binary in nature. Additional information, such as the length of exposure, repeated exposures or age at the time of exposure occurred could be associated with the psychological and emotional wellbeing of an adolescent. These factors could also mediate the lasting effects of exposure to adverse experiences in childhood on weight status throughout adolescence. Although our model adjusted for various sources of confounding, the observations made in our study could also have been influenced by unmeasured confounding.

CONCLUSIONS

Exposure to any adverse childhood experiences or ACEs before 9 years of age was directly associated with BMI at 18 years of age. Exposure to any adverse childhood experiences and ACEs were associated with lower self-concept and behavioural difficulties in early adolescence and these factors had modest mediating effects for the association between the exposure to adverse childhood experiences and BMI at 18 years old.

DATA AVAILABILITY

The datasets used for the current study are available from the Irish Social Science Data Archive: https://www.ucd.ie/issda/data/guichild/.

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AUTHOR CONTRIBUTIONS

J.M. devised the original proposal, secured funding for the analyses, and had overall responsibility for the study. H.S.D.V. helped develop the original proposal, was involved in the analysis of data, and drafted the manuscript. B.D. was responsible for data analysis and data interpretation. All authors contributed to data interpretation, reviewed and contributed to drafts of the manuscript, and approved the final report.

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COMPETING INTERESTS

The authors declare no competing interests.

CONSENT STATEMENT

All stages of the Growing Up in Ireland project were approved by the Health Research Board's standing Research Ethics Committee based in Dublin, Ireland and these analyses were approved by the Biomedical Research Ethics Board at the University of Manitoba.

ADDITIONAL INFORMATION

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