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Does decision-making at age 11 predict prodromal eating pathology at ages 14 and 17? A prospective, observational, UK population-based cohort study

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ABSTRACT

Objectives We examined whether decision-making at age 11 and 14 is associated with prodromal eating pathology at age 14 and whether it would persist across adolescence and also be present at age 17.

Design This prospective, observational, population-based cohort study used a longitudinal design.

Setting Data from the Millennium Cohort Study (MCS), a UK longitudinal cohort study involving 19 244 families from England, Scotland, Wales and Northern Ireland, were analysed.

Participants We modelled data from 8922 boys and girls aged 11, 14 and 17 (MCS sweeps 5, 6 and 7).

Primary and secondary outcomes We investigated decision-making using the risk-taking, quality of decision-making, deliberation time, delay aversion and risk adjustment subscales of the Cambridge Gambling Task and prodromal eating pathology through binary response items measuring: body dissatisfaction (whether the participant perceived their body as being too overweight); intention to lose weight (whether participants reported a strong desire to lose weight); dietary restriction (whether participants reported actively eating less to influence their shape/weight) and excessive exercise (whether participants reported exercising in a driven way in order to influence weight/shape). Data were analysed using latent class analysis and logistic regression.

Results Lower scores on quality of decision-making (OR=0.46) and deliberation time (OR=0.99) at age 14 were associated with prodromal eating pathology at both ages 14 and 17 (all p<0.05), indicating an association between less frequently opting to bet on the most likely outcome and taking less time to decide on which bet to choose and the persistence of prodromal eating pathology over adolescence. Lower deliberation time (OR=0.99) and delay aversion (OR=0.62) at 11 and lower risk-taking scores at 14 (OR=0.43) were associated with the absence of prodromal eating pathology at 14 and 17 (all p<0.05), indicating that a moderate approach under conditions of risk in childhood and mid-adolescence is associated with reduced eating pathology across adolescence.

Conclusions Training advantageous decision-making might protect from later prodromal eating pathology.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ Decision-making in those with eating pathology has been examined using cross-sectional designs and this study aimed to advance the evidence base through a longitudinal study.
⇒ The Millennium Cohort Study (MCS) is an ongoing cohort study of 19 244 families involving a multidisciplinary survey aimed at investigating the influence of early family context on child development throughout childhood and into adolescence. The sample design over-represents families living in areas of high child poverty, areas with high proportions of ethnic minority populations across England and the three smaller UK countries.
⇒ Due to the multidisciplinary nature of the MCS, the measurement of eating pathology is not based on a standardised clinical interview.
⇒ Some of the identified effects in this research are small and should be interpreted with caution.

INTRODUCTION

Eating disorders (EDs) are serious psychological and psychiatric illnesses which frequently have an adolescent onset.[1] Experimental studies provide evidence of poorer decision-making in adults with EDs relative to non-ED controls.[2–4] This evidence; however, relied largely on cross-sectional adult samples usually recruited from ED clinics. Using data from the Millennium Cohort Study (MCS), a UK Longitudinal cohort study which enrolled 19 244 families whose children were born across England, Scotland, Wales and Northern Ireland in 2000–2001, our previous work using longitudinal data has added to this literature. We have found decision-making, specifically the quality of decision-making and risk-taking subscales of the Cambridge Gambling Task (CGT)[5 6] at age 11, is associated with symptoms thought to be prodromes of EDs (early symptoms indicative of the onset of an illness[7] measured using questions from the MCS assessing body dissatisfaction, intention to lose weight, dietary restriction, significant under/overweight and excessive exercise) at age 14.[8] This suggests that being

able to make advantageous decisions under conditions of risk in childhood might offer some prevention from prodromal eating pathology occurring at the time when most people who are later identified to have developed a clinical illness appear to be most at risk of developing this disorder (ie, mid adolescence).[1] These findings were developed further using latent class analysis (LCA),[9] which identified two groups within the cohort at age 14, one endorsing prodromal eating pathology and the other without prodromal eating pathology. Logistic regression models showed higher risk-taking scores were associated with a 60% greater probability of being in the prodromal eating pathology group, and higher scores on quality of decision-making were associated with a 30% lower probability of being in the prodromal eating pathology group. This further supports the idea that disadvantageous decision-making in childhood is associated with the presence of clusters of concerning symptoms (high body dissatisfaction, a strong desire to lose weight, the use of dietary restriction and exercise to influence weight and the presence of 'overweight', according to the UK90,[10] indicating the likely presence of emerging eating pathology. Decision-making skills are thought to follow a J-shaped developmental trajectory, declining from late childhood into early adolescence and improving in mid adolescence and into early adulthood.[11] However, data also show that the risk-taking component of decision-making measured by the CGT in the MCS improves less between the ages of 11 and 14 in those who endorse prodromal eating pathology compared with individuals without prodromal eating pathology who were more likely to be of 'average' weight, according to the UK90[10] with minimal disordered attitudes and behaviours in relation to eating and weight/shape.[12] This contributes to suggestions that EDs have a neurodevelopmental aetiology.[13]

These findings corroborate longitudinal work involving clinical participants in which the decision-making skills of 14 adults with anorexia nervosa were assessed at two time points (before and after weight restoration)[14] with the Iowa Gambling Task.[15] This research found there was no significant improvement in advantageous decision-making over time. However, these findings are derived from a clinical context in which acute and chronic starvation may be confounders and sampling neglects the more diverse groups of individuals with ED symptoms in the wider community. As there exists this notable paucity of longitudinal investigations into decision-making and ED prodromes, our research team have sought to reduce this gap in the literature. This is of particular interest because it may be possible to teach advantageous decision-making using, for example, gamification to help young people make advantageous decisions around eating and exercise, and this has been piloted in elementary school students.[16] It could also be of use to enhance decision-making skills with interventions that orient young people to the consequences of their choices. Further, computational psychiatry suggests suboptimal decision-making may be a component of psychopathology more broadly.[17]

This study; therefore, aimed to develop the evidence base on the role of decision-making in childhood in the later development of prodromal eating pathology in adolescence by investigating new data from MCS participants at age 17. The objective was to include new age 17 data in our models and to understand whether our findings on the relationship between decision-making at age 11 and prodromal eating pathology at age 14 would persist across adolescence and also be present at age 17.

It was hypothesised that less advantageous decision-making on the CGT, in particular, differences on the risk-taking and quality of decision-making variables of the CGT, would be associated with the presence of prodromal eating pathology (body dissatisfaction, intention to lose weight, dietary restriction, excessive exercise and significant under/overweight) not only at age 14, but also at age 17, indicating the possible persistence of an ED prodrome.

METHODS

Design

This prospective, observational, population-based cohort study used a longitudinal design.

Participants

The MCS is an ongoing cohort study developed as a multidisciplinary survey aimed at investigating the influence of early family context on child development and outcomes throughout childhood and into adolescence. Further investigation is planned in adulthood. To date, the MCS has enrolled 19 244 families whose children were born across the UK countries of England, Scotland, Wales and Northern Ireland in 2000–2001. The sample design over-represents families living in areas of high child poverty, areas with high proportions of ethnic minority populations across England and the three smaller UK countries. There have been data from seven sweeps to date. MCS children were around 9 months old at sweep 1, and 3, 5, 7, 11, 14 and 17 years old at sweeps 2–7, respectively. Data from sweeps 5,–7 are used in this study. This is because in the MCS, ED-related attitudes and behaviours were first measured at age 14 and the CGT was administered at ages 11 and 14. The analytical sample included singletons and first-born twins or triplets with available information on ED-related attitudes and behaviours at age 14 and/or age 17 and with available CGT data at age 11 or 14 (n=8922).

Patient and public involvement

The findings of this study were shared at a workshop for carers of loved ones with EDs and with a patient group of individuals with EDs.

Measures

Decision-making under conditions of risk

The CGT assesses decision-making and risk-taking behaviour outside a learning context under conditions of uncertainty.[5 6] Participants view a computer screen

displaying 10 boxes (red and blue) which appear in varying ratios (6:4, 7:3, 8:2, 9:1) of red to blue. Participants track a yellow token hidden inside one of these boxes. They have to choose: (A) which colour of box they believe the token is hidden behind (red or blue) and (B) the number of points they want to gamble. The five CGT measures of decision-making used in this study include: (1) the average number of points placed on bet after the most likely outcome was chosen (risk-taking); (2) the mean proportion of trials where the most likely colour outcome was selected (quality of decision-making); (3) the mean reaction time for making a selection (deliberation time); (4) the tendency to stake higher bets on favourable compared with unfavourable trials (risk adjustment) and (5) the total difference between risk-taking scores (points gambled) in the ascending and descending conditions (delay aversion). A sixth CGT measure, overall proportion bet, was excluded from the analysis due to its significant correlation (>0.90) with the risk-taking variable.

Prodromal eating pathology

Symptoms indicating the emergence of ED thoughts and behaviours were measured at age 14 and 17 using the available eating, dieting and body image questions from the MCS. These binary response items questions measured: body dissatisfaction (whether the participant perceived their body as being too overweight); intention to lose weight (whether the participant report a strong desire to lose weight); dietary restriction (whether the participant reported having actively eaten less to influence their shape/weight) and excessive exercise (whether the participant reported exercising in a driven way in order to influence weight and shape). These items reflect symptoms considered to be prodromes of EDs, that is symptoms that are a feature of the disorder in question (in this case, EDs) and can indicate the future onset of a clinical disorder.[7] The items align with key diagnostic features of clinical EDs.[18]

Confounders

Covariates known to be associated with exposure and outcome were included in the models: sex, ethnicity (according to the UK census groups of white, black, Indian, Pakistani/Bangladeshi, mixed or other), family poverty (below the poverty line or not), IQ, (verbal and non-verbal) derived in the MCS at age 5 from three subscales of the British Ability Scales,[18] pubertal status at age 11 (breast growth or menstruation or hair on body for females, and voice change or facial hair or hair on body for males) and internalising and externalising symptoms at age 11, assessed using the mother-rated Strengths and Difficulties Questionnaire (SDQ).[19] The SDQ is a valid and reliable tool for measuring such problems symptoms in children. It consists of 20 'difficulties' items related to behaviour (in the past 6 months), with each item scored on a 3-point scale (0 = 'not true', 1 = 'somewhat true' and 2 = 'certainly true'). Items are summed to form four subscales (emotional, conduct, hyperactivity and peer problems) or two (internalising symptoms, the sum of the scores on the emotional and peer problems items, and externalising symptoms, the sum of the scores on the conduct and hyperactivity problems items), which was used for this analysis. An objective measure of underweight and overweight based on the most widely used reference panel, the UK90,[10] sensitive to sex and age, and developed for the British population was also included. Cut-offs were based on the age of the cohort member at the time of data collection. The underweight cut-off point was the second centile and the overweight cut-off point was the 85th centile, according to the UK90. It is important to acknowledge that people can have EDs at any weight, and so we have included this variable in our modelling.

Data analysis

All analyses were performed in STATA V.16.0.[20] In all models, the MCS sampling stratum was controlled to account for the disproportionate stratification of the MCS survey design. To identify potential clusters of ED symptoms at ages 14 and 17 in the cohort, we used LCA using the prodromal eating pathology items (body dissatisfaction, intention to lose weight, dietary restriction, excessive exercise). We ran 1–3 class models for each age (ie, 14 and 17) and used three goodness of fit indices to determine which of these models fits best: (1) The Bayesian information criterion (BIC); (2) the Akaike information criterion (AIC) and (3) the entropy of each model. Lower BIC and AIC values indicate better fit to the data. Entropy ranges from 0 to 1, with higher values indicating that the latent classes are clearly distinguishable. Based on these indices we considered the two-class solution as optimal at both ages, with class 1 representing those who were more likely to report prodromal eating pathology (ie, endorsement of the body dissatisfaction, intention to lose weight, dietary restriction and excessive exercise items) and class 2 representing those who were less likely to report prodromal eating pathology (ie, no endorsement of these items). We then divided our sample into four groups: group 1 included those who were more likely to report prodromal eating pathology at age 14 only; group 2 included those who were more likely to report prodromal eating pathology at age 17 only; group 3 included those who were more likely to report prodromal eating pathology at ages 14 and 17 and group 3 included those who were less likely to report prodromal eating pathology at ages 14 and 17. Finally, we fitted four different sets of logistic regression models in order to explore the association between CGT measures and symptom clusters. Missingness in our sample ranged between 1.60% for body dissatisfaction measured at age 14 and 29.71% for dietary behaviour measured at age 17. To handle missingness, we used multiple imputation by chained equations (20 imputed datasets).

Table 1 Frequencies of prodromal eating pathology group membership (total sample n=8922)

Group	Yes (%)	No (%)
Group 1- Prodromal eating pathology at age 14 only	720 (8.1)	8202 (91.9)
Group 2- Prodromal eating pathology at age 17 only	2397 (6.9)	6525 (73.1)
Group 3- Prodromal eating pathology at ages 14 and 17	3712 (41.6)	5210 (58.4)
Group 4- Prodromal eating pathology not detected at ages 14 and17	2093 (23.5)	6829 (76.5)

The groups that are listed in this table are mutually exclusive.
Bold text indicates significant findings.

RESULTS

Table 1 shows that that around 40% of the sample provided responses indicating that they endorsed the presence of prodromal eating pathology group at both age 14 and 17.

Table 2 provides data on the relationship between CGT variables measured at ages 11 and 14 and prodromal eating pathology at age 14, or age 17, or both at ages 14 and 17. The data show that lower quality of decision-making and lower deliberation time at age 14 were associated with prodromal eating pathology at age 14 and 17. Lower delay aversion and lower deliberation time at age 11 and lower risk-taking at age 14 were associated with being in the group where prodromal eating pathology was not detected at both ages 14 and 17.

DISCUSSION

The aim of this study was to use new data from the MCS collected from participants at age 17 to explore whether decision-making measured by the CGT at age 11 and 14 was associated with prodromal eating pathology at ages 14 and 17, showing evidence of persistence across adolescence. The hypothesis, which was that less advantageous decision-making on the CGT, would be associated with the presence of prodromal eating pathology not only at age 14, but also at age 17, was partially supported by the data, because poorer quality of decision-making and lower deliberation time at age 14 were associated with prodromal eating pathology at both ages 14 and 17. This indicates that the persistence of prodromal eating pathology in mid to late adolescence can be understood in part as a function of less frequently placing bets on the most likely outcome (quality of decision-making) and taking less time to decide on which bet to choose (deliberation time) at age 14. This suggests that more hasty and less advantageous decision-making might be associated with the later presence of prodromal eating pathology. Further, lower scores on the deliberation time and delay aversion variables at age 11 and lower scores on the risk-taking variable at age 14 were associated with prodromal eating pathology not being reported at both ages 14 and 17. This suggests that the absence of prodromal eating pathology in both mid and late adolescence can be understood in part as a function of taking less time to decide on which bet to choose (deliberation time) at age 11, a reduced tendency to bet larger amounts due to an unwillingness or inability, to wait for bets to decrease on trials where bet amounts are presented in descending order compared with when bets are presented in ascending order (delay aversion) at age 11 and gambling less of the current points total (risk-taking) at age 14. This indicates that perhaps a moderate approach under conditions of risk could be protective of the later development and continuation of prodromal eating pathology. It might be that individuals continue to engage in dietary restriction and driven exercise, negative attitudes towards their shape/weight and continue to hold a desire to lose weight, betting on these as ways of feeling good or better about their body/weight/shape. However, the most likely outcome of this approach is not weight loss or well-being, but instead ongoing difficulties with eating and body image.[21] A clear implication is that children and adolescents could be supported to learn more about the attitudes and behaviours conducive to good outcomes, that is, a good enough body image and relationship with food throughout adolescence[22] and this may be one way of enhancing ED prevention programmes.

While it is not possible to know whether those who were observed to have prodromal eating pathology at age 14 then experienced a remission in these symptoms before recurring again around age 17, the finding that a significant group of individuals who were in the prodromal eating pathology group at age 14 were also in the prodromal eating pathology group at age 17 suggests that for many, these prodromal symptoms do not spontaneously resolve. It is therefore important that where an intention to lose weight, active dietary restriction and the use of driven exercise to influence shape/weight are identified by concerned family members, friends or teachers, this young person is supported early on prevent their symptoms from becoming more salient and habitual during this crucial stage in their development.

The study has a number of limitations. Due to the multidisciplinary nature of the MCS and the fact that we were not involved in decisions around which items were included in the survey, the measurement of ED symptoms is not based on a standardised clinical interview. The MCS did not include items on bingeing which would have been useful in assessing a broader range of prodromal eating pathology. Further, participants may not have accurately

Table 2 Adjusted models in imputed cases (n=8922)

Class 1- Prodromal eating pathology at age 14 only			
	b	**SE**	**OR**
Risk-taking, age 11	0.39	0.48	1.48
Quality of decision-making, age 11	−0.18	0.32	0.83
Deliberation time, age 11	0.00	0.00	1.00
Risk adjustment, age 11	0.01	0.08	1.01
Delay-aversion, age 11	0.20	0.30	1.22
Risk-taking, age 14	0.04	0.43	1.04
Quality of decision-making, age 14	0.27	0.44	1.31
Deliberation time, age 14	0.00	0.00	1.00
Risk adjustment, age 14	−0.06	0.06	0.94
Delay-aversion, age 14	0.16	0.32	1.17
Class 2- Prodromal eating pathology at age 17 only			
	b	**SE**	**OR**
Risk-taking, age 11	0.05	0.23	1.05
Quality of decision-making, age 11	0.00	0.22	1.00
Deliberation time, age 11	0.00	0.00	1.00
Risk adjustment, age 11	−0.03	0.05	0.96
Delay aversion, age 11	0.22	0.17	1.25
Risk-taking, age 14	0.12	0.27	1.13
Quality of decision-making, age 14	0.19	0.30	1.20
Deliberation time, age 14	−0.00	0.00	1.00
Risk adjustment, age 14	−0.02	0.04	0.97
Delay-aversion, age 14	0.14	0.22	1.15
Class 3- Prodromal eating pathology at ages 14 AND 17			
	b	SE	OR
Risk-taking, age 11	0.19	0.23	1.21
Quality of decision-making, age 11	0.01	0.22	1.01
Deliberation time, age 11	0.00	0.00	1.00
Risk adjustment, age 11	0.03	0.04	1.03
Delay-aversion, age 11	0.06	0.16	1.06
Risk-taking, age 14	0.50	0.26	1.65
Quality of decision-making, age 14	**−0.75***	0.29	0.46
Deliberation time, age 14	**−0.00†**	0.00	0.99
Risk adjustment, age 14	0.05	0.04	1.05
Delay-aversion, age 14	0.01	0.19	1.01
Class 4- Prodromal eating pathology not detected at ages 14 &17			
	b	SE	OR
Risk-taking, age 11	−0.51	0.26	0.59
Quality of decision-making, age 11	0.00	0.23	1.00
Deliberation time, age 11	**−0.00***	0.00	0.99
Risk adjustment, age 11	−0.02	0.05	0.97
Delay-aversion, age 11	**−0.46***	0.20	0.62
Risk-taking, age 14	**−0.84†**	0.29	0.43
Quality of decision-making, age 14	0.58	0.35	1.79
Deliberation time, age 14	0.00	0.00	1.00
Risk adjustment, age 14	−0.01	0.05	0.98
Delay-aversion, age 14	−0.29	0.22	0.74

Adjusted for: sex, ethnicity, sociio-economic status (SES), IQ at age 5, pubertal status, age, internalising and externalising symptoms at age 11, UK90 cut-offs for overweight and underweight at ages 14 and 17.
Bold text indicates significant findings.
*p<0.05.
†p<0.001.
SES, Socio-economic status.

reported or been aware of any symptoms that they might have. We also do not know whether those with prodromal eating pathology actually developed clinical EDs or whether their symptoms remitted, and these data would be interesting. It might be possible to learn more as the cohort are followed up again in future. Further, there may be other covariates not measured in the MCS that are relevant to prodromal eating pathology. However, fortunately the questions asked do embody key ED symptoms listed in the Diagnostic and Statistical Manual of Mental Disorders[23] and it should be noted that this work was more interested in the emergence of prodromal eating pathology in a large community cohort sample to challenge the present bias in the data towards clinical samples and improve the inclusion of a broader range of individuals in ED research. The effect of deliberation time at age 14 on group membership is extremely small and should be interpreted with caution. Finally, the study uses observational data and therefore, while we adjusted for relevant covariates in our modelling, the design is not experimental and therefore causation cannot be inferred.

In conclusion, this work builds on our previous studies,[8 9 12] which examined decision-making at ages 11 and 14 and ED prodromes at age 14 and is novel because it demonstrates that the association between decision-making at age 11 and 14 and the later presence of ED prodromes is persistent and not transitory. Training effective decision-making in childhood and early adolescence may offer a means of preventing ED prodromes, reducing the likelihood of a clinical disorder developing.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting, or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by Yorkshire and The Humber – Leeds East Research Ethics Committee (ref. 11/YH/0203) and London Central (ref. 13/LO/1786) National Health Service Research Ethics Committee. Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available in a public, open access repository. Accessible here: https://cls.ucl.ac.uk/cls-studies/millennium-cohort-study/

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REFERENCES

1 Schmidt U, Adan R, Böhm I, *et al*. Eating disorders: the big issue. *Lancet Psychiatry* 2016;3:313–5.
2 Ciberti A, Cavalletti M, Palagini L, *et al*. Decision-Making, impulsiveness and temperamental traits in eating disorders. *Clin Neuropsychiatry* 2020;17:199–208.
3 Guillaume S, Gorwood P, Jollant F, *et al*. Impaired decision-making in symptomatic anorexia and Bulimia nervosa patients: a meta-analysis. *Psychol Med* 2015;45:3377–91.
4 Giannunzio V, Degortes D, Tenconi E, *et al*. Decision-Making impairment in anorexia nervosa: new insights into the role of age and decision-making style. *Eur Eat Disord Rev* 2018;26:302–14.
5 Deakin J, Aitken M, Robbins T, *et al*. Risk taking during decision-making in normal volunteers changes with age. *J Int Neuropsychol Soc* 2004;10:590–8.
6 Rogers RD, Everitt BJ, Baldacchino A, *et al*. Dissociable deficits in the decision-making cognition of chronic amphetamine abusers, opiate abusers, patients with focal damage to prefrontal cortex, and tryptophan-depleted normal volunteers: evidence for monoaminergic mechanisms. *Neuropsychopharmacology* 1999;20:322–39.
7 Stice E, Ng J, Shaw H. Risk factors and prodromal eating pathology. *J Child Psychol Psychiatry* 2010;51:518–25.
8 Harrison A, Francesconi M, Flouri E. Decision-Making in childhood predicts prodromal eating pathology in adolescence. *J Dev Behav Pediatr* 2022;43:e407–13.
9 Harrison A, Francesconi M, Flouri E. Types of eating disorder prodrome in adolescence: the role of decision making in childhood. *Front Psychol* 2022;13:743947.
10 Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. *Arch Dis Child* 1995;73:25–9.
11 Smith DG, Xiao L, Bechara A. Decision making in children and adolescents: impaired Iowa gambling task performance in early adolescence. *Dev Psychol* 2012;48:1180–7.
12 Francesconi M, Flouri E, Harrison A. Change in decision-making skills and risk for eating disorders in adolescence: a population-based study. *Eur Psychiatry* 2020;63:e93.
13 Connan F, Campbell IC, Katzman M, *et al*. A neurodevelopmental model for anorexia nervosa. *Physiol Behav* 2003;79:13–24.
14 Bodell LP, Keel PK, Brumm MC, *et al*. Longitudinal examination of decision-making performance in anorexia nervosa: before and after weight restoration. *J Psychiatr Res* 2014;56:150–7.
15 Bechara A, Damasio H, Tranel D, *et al*. The Iowa gambling task and the somatic marker hypothesis: some questions and answers. *Trends Cogn Sci* 2005;9:159–62.
16 Jones BA, Madden GJ, Wengreen HJ, *et al*. Gamification of dietary decision-making in an elementary-school cafeteria. *PLoS One* 2014;9:e93872.
17 Montague PR, Dolan RJ, Friston KJ, *et al*. Computational psychiatry. *Trends Cogn Sci* 2012;16:72–80.
18 Elliott CD, Smith P, McCulloch K. *British ability scales II*. Windsor, UK: NFER-Nelson, 1996.
19 Goodman R. The strengths and difficulties questionnaire: a research note. *J Child Psychol Psychiatry* 1997;38:581–6.
20 STATACorp L. New in Stata 16 2019.
21 Pietiläinen KH, Saarni SE, Kaprio J, *et al*. Does dieting make you fat? A twin study. *Int J Obes* 2012;36:456–64.
22 Wilson RE, Marshall RD, Murakami JM, *et al*. Brief non-dieting intervention increases intuitive eating and reduces dieting intention, body image dissatisfaction, and anti-fat attitudes: a randomized controlled trial. *Appetite* 2020;148:104 556.
23 American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. Washington DC: American Psychiatric Association, 2013[dataset].