**Quality control checks of genotyping data**

Individuals with disproportionate levels of individual missingness (i.e., >3%), insufficient sample replication (identity by descent < 0.1), biological sex mismatch, and non-European ancestry (as defined by multi-dimensional scaling using the HapMap Phase II, release 22, reference populations) were excluded. SNPs with a minor allele frequency (MAF) of < 1%, excessive missingness (i.e., call rate < 95%), or a departure from the Hardy–Weinberg equilibrium ($P$ value < 5 x $10^{-7}$) were removed. Imputation was conducted with Impute3 using the HRC 1.0 as the reference panel [1] and phasing was carried out using ShapeIT (v2.r644). Finally, post-imputation quality control checks were performed; any SNPs with MAF less than 1%, Impute3 information quality metric of < 0.8, and not confirming to Hardy-Weinberg equilibrium ($P < 5 \times 10^{-7}$) were removed. After data cleaning, a total of 8,654 individuals (4,225 females and 4,429 males) and 4,054,653 SNPs remained eligible for analyses.

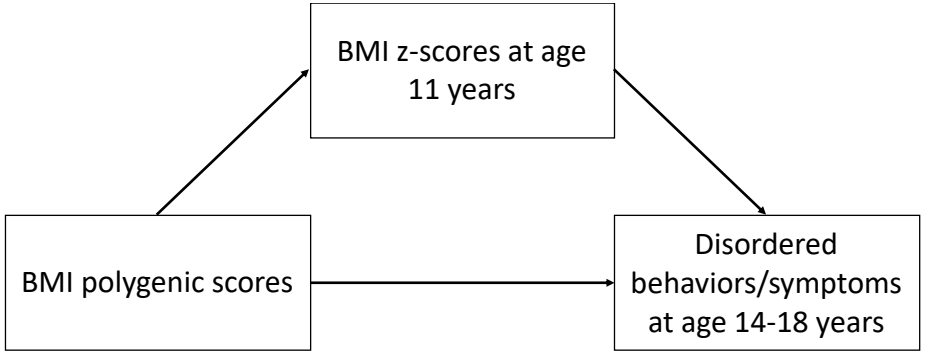


**Figure 1. Directed acyclic graph (DAG) of the causal mediation analyses.** The body mass index polygenic scores (BMI-PGS) was derived from summary statistics of the genome-wide association study (GWAS) carried out by the Genetic Investigation of Anthropometric Traits (GIANT) consortium [8] and were calculated for participants in the Avon Longitudinal Study of Parents and Children [4–7]. The sex and age adjusted BMI z-scores were included as a mediator in the causal mediation analyses that was carried out using the R package ´mediation´ (version 4.4.6; 32). The 'mediation' package is based on concepts proposed in modern causal inference [9]. Prior to the mediation analyses the BMI-PGS was standardized (to mean zero and standard deviation of one) and the analyses were controlled for biological sex and the first four ancestry-informative principal components. Confounders are not included in this DAG for simplicity.

**Table 1.** Age, sex, and ethnicity of the participants in the Avon Longitudinal Study of Parents and Children at all three time points.

| Wave | Age in years (SD) | Female N (%) | Child ancestry/ white ethnicity N (%) |
|---|---|---|---|
| 14 | 14.0 (0.19) | 3,416 (55.5) | 5,372 (87.2) |
| 16 | 16.7 (0.24) | 3,095 (58.7) | 4,482 (86.0) |
| 18 | 18.7 (0.49) | 2,174 (64.2) | 3,010 (88.9) |

**Table 2.** Associations of body mass index polygenic score (BMI-PGS) with BMI in the Avon Longitudinal Study of Parents and Children.

| Outcome | Age outcome measured (years) | N | Threshold[a] | N SNPs | $R^2$ | β (95% CI)[b] | Q[c] |
|---|---|---|---|---|---|---|---|
| BMI | 11 | 5371 | 0.0180001 | 19457 | 0.110480 | 1.14 (1.05, 1.22) | $9.09 \times 10^{-5}$ |
| BMI z-scores | 11 | 4037 | 0.0180001 | 19457 | 0.123235 | 0.40 (1.05, 1.22) | $9.09 \times 10^{-5}$ |
| BMI | 18 | 3931 | 0.0230001 | 21040 | 0.121126 | 1.44 (1.31, 1.56) | $9.09 \times 10^{-5}$ |

SNP = single nucleotide polymorphism; $R^2$, squared multiple correlation; OR, odds ratio; CI, confidence interval. [a] The optimal *P*-value threshold for the inclusion of SNPs in the calculation of the body mass index (BMI) polygenic score (PGS) as determined by PRSice's high-resolution scoring [2]. [b] Standardized betas reflect one standard deviation increase in the standardized (to mean zero and standard deviation of one) BMI-PGS. [c] Benjamini & Hochberg false discovery rate adjusting for the number of phenotypes tested [3].

**Table 3.** Effect size of associations of body mass index polygenic score (BMI-PGS) with disordered eating behaviors and cognitions in the total samples and separately for female participants of the Avon Longitudinal Study of Parents and Children.

| | | Total sample | Females[a] |
|---|---|---|---|
| **Binary outcomes** | **Age outcome measured (years)** | **OR (95% CI)[b]** | **OR (95% CI)[b]** |
| Fasting | 14 | 1.45 (1.28, 1.64) | 1.43 (1.25, 1.64) |
| Fasting | 16 | 1.30 (1.18, 1.43) | 1.27 (1.15, 1.42) |
| Fasting | 18 | 1.26 (1.06, 1.51) | 1.26 (1.05, 1.52) |
| Binge eating | 14 | 1.29 (1.14, 1.47) | 1.27 (1.08, 1.49) |
| Binge eating | 16 | 1.22 (1.10, 1.35) | 1.18 (1.05, 1.33) |
| Binge eating | 18 | 1.23 (1.10, 1.39) | 1.25 (1.09, 1.43) |
| Purging | 16 | 1.24 (1.08, 1.42) | 1.23 (1.07, 1.42) |
| Purging | 18 | 1.21 (1.03, 1.43) | 1.21 (1.02, 1.44) |
| **Continuous outcomes** | **Age outcome measured (years)** | **β (95% CI)[c]** | **β (95% CI)[c]** |
| Thin ideal internalization | 14 | -0.15 (-0.23, -0.07) | -0.15 (-0.24, 0.05) |
| Body dissatisfaction | 14 | 0.99 (0.77, 1.22) | 1.24 (0.92, 1.55) |
| Restrained eating | 14 | 0.14 (0.10, 0.17) | 0.16 (0.11, 0.21) |
| Emotional eating | 14 | 0.21 (0.052, 0.38) | -0.10 (-0.34, 0.14) |
| External eating | 14 | -0.19 (-0.30, -0.09) | -0.18 (-0.32, -0.05) |

OR, odds ratio; CI, confidence interval. [a] Polygenic score analyses only carried out in female participants. [b] ORs reflect one standard deviation change in the standardized (to mean zero and standard deviation of one) BMI-PGS. [c] Standardized betas reflect one standard deviation increase in the standardized (to mean zero and standard deviation of one) BMI-PGS.

**Table 4.** Generalized linear mixed models for the association between the body mass index polygenic score (BMI-PGS) and the disordered eating behaviors with age at self-report of the disordered eating as an interaction term [a].

| Outcome | Threshold [b] | N SNPs | $\chi^2$ | $P_{interaction}$ |
|---|---|---|---|---|
| Fasting | 1 | 81,503 | 4.89 | 0.09 |
| Binge eating | 1 | 81,503 | 5.23 | 0.07 |
| Purging | 1 | 81,503 | 0.19 | 0.66 |

SNP = single nucleotide polymorphism; $\chi^2$, test statistic as measured by chi-square. [a] Analyses were corrected for biological sex and the first four ancestry-informative principal components for the individuals in the Avon Longitudinal Study of Parents and Children. [4–7]. [b] *P*-value threshold for the inclusion of SNPs in the calculation of the BMI-PGS was set to 1 using PRSice [2].

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