**Supporting information**

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**Text S1. Sample selection for the quantitative genetic analysis**

We identified all sibling pairs from each nuclear family from the entire study population. In each pair, one individual defined the exposure (index person), and the other defined the outcome. For example, six pairs of siblings can be identified from three unique siblings. We then constructed a dataset containing sibling pairs and their biological parents, and assigned a random number to each pair of siblings. Next, we sorted the dataset by a combination of mother’s identity number and the random number, and selected the first pair of siblings born to each mother. We then sorted the dataset again by a combination of father’s identity number and the random number, and selected the first pair of siblings born to each father. By doing so, we ensured that each parent contributed only one pair of siblings. Eventually, the sample for the quantitative genetic analysis included 664 721 pairs of full siblings, 68 347 pairs of maternal half siblings, and 69 351 pairs of paternal half siblings.

**Table S1. Bivariate quantitative genetic model comparisons based on likelihood ratio tests**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
|  | **Model Comparison Measures** | | | | |
| **Model** | **No. of parameters** | **AIC** | **-2LL** | **Diff - 2LL** | **P Value** |
| **Bivariate ADCE** | 14 | 53.98 | 115.86 | NA | NA |
| **Bivariate ACE\*** | 11 | 53.86 | 115.86 | 0.00 | 1.000 |
| **Bivariate AE** | 8 | 66.30 | 134.30 | 18.44 | 0.005 |

\* Best fitting model

A: additive genetic component; D: dominant genetic component; C: shared environmental component; E: non-shared environmental correlation;

Diff-2LL: difference in -2×log likelihood between the restricted model and the ADCE model

**Figure S1. Explained variance in ADHDa and clinical obesityb**

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a Variance explained for by genetic, shared environmental, and nonshared environmental effects, either unique for ADHD or in common with clinical obesity.

b Variance explained for by genetic, shared environmental, and nonshared environmental effects, either unique for clinical obesity or in common with ADHD.