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Childhood obesity is negatively associated with completed educational level independent of socioeconomic status: a prospective cohort study

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4 1 Childhood obesity is negatively associated with completed
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6 2 educational level independent of socioeconomic status: a
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9 3 prospective cohort study
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15 5 Louise Lindberg¹, Martina Persson^{2,3,4}, Pernilla Danielsson¹, Emilia Hagman^{1*}, Claude
16 6 Marcus¹
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23 8 ¹Department of Clinical Science, Intervention and Technology, Division of Pediatrics,
24 9 Karolinska Institutet, Stockholm, Sweden
25

26 10 ²Department of Medicine Solna, Clinical Epidemiology Unit, Karolinska Institutet,
27 11 Stockholm, Sweden
28

29 12 ³Department of Diabetes and Endocrinology, Sachska Children's Hospital Södersjukhuset,
30 13 Stockholm, Sweden
31
32

33 14 ⁴Department of Clinical Science and Education, Södersjukhuset, Karolinska Institutet,
34 15 Stockholm, Sweden
35

36 16 *Corresponding author e-mail: emilia.hagman@ki.se (EH)
37

38 17 Other contributing authors e-mail: louise.lindberg@ki.se; martina.persson@ki.se;
39 18 pernilla.danielsson.liljeqvist@ki.se; claudemarcus@ki.se
40

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21 **Abstract**

22 **Objectives**

23 Children with obesity achieve lower educational level compared with normal-weight peers.
24 Parental socioeconomic status (SES) impacts both a child's academic achievement and risk
25 of obesity. The degree to which the association between obesity and education depend on
26 parental SES is unclear. Therefore, the primary aim is to investigate if individuals with
27 obesity in childhood are less likely to complete ≥ 12 years of schooling, independently of
28 parental SES. The secondary aim is to study how weight loss, level of education, and parental
29 SES are associated.

31 **Methods**

32 In this nationwide prospective cohort study, children aged 10-17 years, recorded in the
33 Swedish Childhood Obesity Treatment Register (BORIS), and aged 20 years or older at
34 follow-up were included (n=3,942). A comparison group was matched by sex, year of birth,
35 and living area (n=18,728). Parental SES was based on maternal and paternal level of
36 education, income, and occupational status. The main outcome, completion of ≥ 12 years of
37 schooling, was analysed with conditional logistic regression, and adjusted for group,
38 migration background, ADHD/ADD, anxiety/depression, and parental SES.

40 **Results**

41 Among those with obesity in childhood, 56.7% completed ≥ 12 years in school compared
42 with 74.4% in the comparison group (p<0.0001). High parental SES compared to low SES

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3 43 was strongly associated with attained level of education in both children with and without
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5 44 obesity, adjusted odds ratio (^aOR) [99% confidence interval (CI)]=5.40 [4.45 to 6.55].
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8 45 However, obesity in childhood remains a strong risk factor of completing ≥ 12 school years,
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10 46 independently of parental SES, ^aOR=0.57 [0.51 to 0.63]. Successful obesity treatment
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12 47 increased the odds of completing ≥ 12 years in school even when taking parental SES into
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14 48 account, ^aOR=1.34 [1.04 to 1.72].
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20 **Conclusions**

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22 51 Individuals with obesity in childhood have lower odds of completing ≥ 12 years of schooling,
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24 52 independently of parental SES. Optimized obesity treatment may improve school results in
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27 53 this group.
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32 **Article summary / Strengths and limitation of this study**

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35 56 • In this prospective cohort study, we have been able to investigate the level of
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37 57 education among a large number of individuals who have obesity in childhood (n=
38
39 58 3,942) in comparison with a matched group (n=18,728).
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42 59 • The study design of using longitudinal data from several national registers provided
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44 60 the opportunity to control for important confounding factors, such as neuropsychiatric
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46 61 disorders, anxiety, depression and family socioeconomic status.
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49 62 • Factors such as free education, school lunches, and students' health care may have an
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51 63 impact on the generalizability of our data to other populations.
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65 Introduction

66 During the last 40 years, the prevalence of childhood obesity has increased exponentially in
67 many parts of the world.¹ Childhood obesity is associated with increased risks of somatic
68 morbidity and risk of premature death in adulthood.²⁻⁵ Psychosocial maladjustment, anxiety
69 and depression are also prevalent^{6 7} and may contribute to the obesity-related long-term
70 morbidity and mortality.⁵ Most studies report that children with obesity more often have
71 lower school grades and reach a lower level of education compared with normal-weight
72 peers,⁸⁻¹⁰ but in a recent study, only girls with obesity were affected.¹¹ We have previously
73 confirmed the association between obesity and lower attained level of education among both
74 girls and boys.⁹ We also found that successful obesity treatment was positively correlated
75 with completing ≥ 12 school years, although without taking socioeconomic status (SES) into
76 account.⁹

77 Low parental SES is a well-established risk factor for both childhood obesity and poorer
78 academic achievement.¹² In particular, parental education has been demonstrated to influence
79 the child's performance at school.¹³⁻¹⁵ In addition, depression¹⁶ and neuropsychiatric
80 disorders, including Attention Deficit Disorder with or without Hyperactivity
81 (ADHD/ADD),¹⁷ may adversely affect school results. Depression and neuropsychiatric
82 disorders are more common in children with obesity compared to the general population.^{9 18}
83 Investigating the impact of obesity alone on attained level of education requires that these
84 and other confounders are considered. The primary aim of this study was to disentangle the
85 association of childhood obesity and parental SES on completed level of education. The

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3 86 secondary aim was to study if positive effects of weight loss on attained level of education is
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5 87 affected by parental SES.
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8 88 **Methods**

9 89 **Study population**

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16 90 This prospective cohort study included children with obesity,¹⁹ aged 10-17 years at the start
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18 91 of obesity treatment (December 1994 to December 2015), and aged 20 years or older and
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21 92 living in Sweden at follow-up (December 31st 2017; n=3,942). Data on subjects receiving
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23 93 treatment for childhood obesity was collected from the Swedish Childhood Obesity
24
25 94 Treatment Register (BORIS). BORIS has been thoroughly described elsewhere,²⁰ but in
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27
28 95 short: The main purpose of the register is quality assessment and long-term evaluation of
29
30 96 childhood obesity treatment. Treatment is individualized and consists primarily of lifestyle
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32 97 modification (i.e. diet and physical activity).
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35 98 A comparison group from the general population was randomly identified using the Swedish
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37 99 Total Population Register and matched by sex, year of birth, and living area at the year
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40 100 obesity treatment was initiated (n=18,728). Using density matching without replacement,
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42 101 five individuals were matched to each individual from the childhood obesity cohort. Siblings
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44 102 of children registered in the childhood obesity cohort were excluded from the comparison
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46 103 group. Children with a diagnosis of mental retardation or genetic syndromes were excluded
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48 104 from both the childhood obesity cohort and the comparison group (Fig 1 and S1 Table).
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55 106 **Fig 1. Participant flowchart.**
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3 107 Families were informed in written or verbal about data collection in the Swedish Childhood
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5 108 Obesity Treatment Register. Post an opt-out approval by parents/guardians, data of the
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7 109 children's weight and height were recorded by the local health care provider during treatment
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9 110 visits. There was no data of weight and height of individuals in the comparison group.
11
12 111 However, less than 1% of the individuals in the comparison group were found in the National
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14 112 Patient Register with a diagnosis of obesity in childhood. In Sweden, health care is free of
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16 113 charge for children and adolescents until 18 years of age. The study was approved by the
17
18 114 regional Ethics Committee in Stockholm, Sweden (No. 2016/922-31/1).

115 **Data sources**

116 Using the Swedish identity number, which is unique to each resident in Sweden, data from
117 several national registers were linked ²¹. Data on education, income, and occupational status
118 were obtained from the Longitudinal Integration Database for Health Insurance and Labour
119 Market Studies. Information on migration background for children and their parents was
120 obtained from the Swedish Total Population Register ²². Both these registers are held by
121 Statistics Sweden, a governmental agency that collects and provide official statistics
122 (www.scb.se/en).

123 Data on diagnoses of mental retardation, genetic syndromes, ADHD/ADD, anxiety, and
124 depression were identified based on codes according the International Classification of
125 Diseases (10th revision; S1 Table) and retrieved from the National Patient Register ²³.
126 Information on prescriptions of psychostimulant drugs for ADHD/ADD as well as
127 antidepressants and tranquilizers for anxiety and depression were identified using the
128 Anatomical Therapeutic Chemical classification system (S1 Table) retrieved from the

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3 129 Swedish Prescribed Drug Register²⁴. Data on deaths were retrieved from the Cause of Death
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5 130 Register²⁵. These registers are held and were linked by the governmental agency the National
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7 131 Board of Health and Welfare (www.socialstyrelsen.se/english).

11 132 **Definition of outcome**

13 133 The main outcome was defined as completion of ≥ 12 years of schooling, and based on the
14
15 134 International Standard Classification of Education²⁶. In Sweden, children start school at the
16
17 135 age of 6 or 7 years and attend compulsory school for 9 years. Upper secondary school
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19 136 includes three additional years of schooling and provides the requirements to attend higher
20
21 137 education. Usually students graduate from secondary school at 18 or 19 years of age. All
22
23 138 education in Sweden is free of charge. Students in compulsory and upper secondary school
24
25 139 are provided school lunches and health care at schools, also free of charge.

31 140 **Definition of main exposures**

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33
34 141 Body Mass Index Standard Deviation Score (BMI SDS) is standardized by sex and age and
35
36 142 used to measure degree of obesity in growing children¹⁹. Baseline measures (continuous)
37
38 143 were used for the variable BMI SDS at start of treatment. Response to obesity treatment was
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40 144 based on the change of BMI SDS from the first to the last clinical visit and categorized into
41
42 145 four groups^{27 28}: good response, a reduction of BMI SDS by 0.25 units or more; no response,
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44 146 a change of BMI SDS by +/- 0.25 units; poor response, an increase of BMI SDS by 0.25 units
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46 147 or more; and dropouts, children with less than one year between their first and last measure
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50 148 or without clinical follow-up after their first registered visit.

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3 149 Parental SES was based on maternal and paternal level of education, income, and
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5 150 occupational status at the year the child turned 15 years, which is about the same time as the
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8 151 child starts upper secondary school. In case of the child was adopted, the SES of adoptive
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10 152 parents was used (childhood obesity cohort n=24 and comparison group n=164). The
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12 153 rationale of treating SES as a composite variable was to capture more of the social context
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14 154 and a potential inequality embedded there. Thus, by taking three variables into account
15
16 155 instead of one, we get a more wide and robust measure of SES. Maternal and paternal
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18 156 educational level was categorized into compulsory school, upper secondary school, or
19
20 157 university degree. Annual disposable income was used to reflect maternal and paternal
21
22 158 economic capacity. The annual disposable income includes all taxable (direct labour income,
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24 159 capital gains from shares etc.) and tax-free income (housing and child benefits, student aid
25
26 160 etc.), minus final tax and other negative transfers such as capital loss from shares and
27
28 161 properties. Disposable income from different years was converted to 2017 prices using the
29
30 162 Consumer Price Index for Sweden provided by Statistics Sweden (www.scb.se/en). Income
31
32 163 was categorized into quartiles based on data from the parents in the comparison group. No
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34 164 occupation was defined as either unemployment 6 months or more, or income from long-
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36 165 term sick leave exceeding any income from the individual's gross salary. Individuals
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38 166 considered to have an occupation included those registered as employed or having an income
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40 167 from student grants/loan equivalent to full-time studies for at least one semester. Parental
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42 168 SES was weighted according to maternal and paternal education (0, 1, 2), income (0, 1, 2, 3),
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44 169 and occupational status (0, 1). The mean parental SES score (i.e. the sum of the mother's and
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46 170 father's all SES indicators divided by two) was applied to their child. The SES-variable was
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3 171 categorized into four levels; low SES (0 to 1.5 points), medium-low SES (2 to 3 points),
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5 172 medium-high SES (3.5 to 4.5 points), and high SES (5 to 6 points).
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8 173 The prevalence of both ADHD/ADD⁹ and depression¹⁸ is higher in children with obesity
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10 174 and may negatively influence attained level of education^{9 16 17}. ADHD/ADD and
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12 175 anxiety/depression in children were identified based on diagnosis or dispensed prescribed
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14 176 medication (S1 Table).
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177 **Definition of covariates**

178 The migration background of children in Sweden may impact school achievements and is
19
20 179 therefore an important factor to control for in the analyses^{9 14}. Migration background was
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22 180 categorized as Nordic or non-Nordic. Nordic was defined as born in a Nordic country
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24 181 (Sweden, Norway, Denmark, Finland, or Iceland) with one or two parents born in the Nordic.
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26 182 Children were classified as non-Nordic if born outside the Nordic or born in the Nordic with
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28 183 two parents born outside the Nordic. Other covariates included sex and age (continuous) at
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30 184 start of obesity treatment.
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39 **Statistical analysis**

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42 186 Descriptive statistics are presented as means and confidence intervals (CI), medians and
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44 187 interquartile ranges (IQR), or frequencies and percentages. Conditional logistic regression
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46 188 was used to calculate odds ratios (OR's) with 99% CI for the main outcome, i.e. completing
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48 189 ≥ 12 school years. Conditional logistic regression was used above ordinary logistic regression
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50 190 as the childhood obesity cohort and the comparison group were matched by several variables.
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53 191 Independent variables included in the adjusted analyses were migration background,
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3 192 ADHD/ADD, anxiety/depression, parental SES, and group (childhood obesity cohort or
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5 193 comparison group) in analyses including both groups, otherwise stratified by group.
6
7 194 Interaction between childhood obesity and parental SES was tested. The association of each
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9 195 SES indicator on the odds of completing ≥ 12 school years was also analysed separately for
10
11 196 mothers and fathers. Sensitivity analyses excluding individuals with ADHD/ADD or
12
13 197 anxiety/depression were performed.
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17 198 Secondary analyses were performed within the childhood obesity cohort to examine
18
19 199 associations between patient characteristics and completed educational level. In ordinary
20
21 200 logistic regressions, odds were adjusted for sex, migration background, parental SES, age
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23 201 and BMI SDS at start of treatment, treatment response, ADHD/ADD, and
24
25 202 anxiety/depression. We tested for possible interaction between parental SES and treatment
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27 203 response for the odds of completing ≥ 12 years of schooling.
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32 204 As missing data on parental SES was rare in both groups (Table 1), records with missing data
33
34 205 were excluded from the analyses, i.e. data were not imputed. P-values < 0.01 were considered
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36 206 statistically significant. All analyses were performed using SAS statistical software (version
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38 207 9.4, Cary, NC, USA).
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Table 1. Characteristics of the participants (n=22,670)

	Childhood obesity cohort (n=3,942)		Comparison group (n=18,728)	
	n	%	n	%
Female sex	1,825	46.3	8,701	46.5
Nordic	2,905	73.7	14,048	75.0
Age at end of follow-up (years)	23.4*	21.4-26.3*	23.4*	21.5-26.3*
ADHD/ADD	617	15.7	1,044	5.6
Anxiety/Depression	831	21.1	2,158	11.5
Parental SES				
Low	867	22.0	2,700	14.4
Medium-low	1,540	39.1	6,023	32.1
Medium-high	1,159	29.4	6,587	35.2
High	355	9.0	3,218	17.2
Missing	21	0.5	200	1.1
Maternal education				
Compulsory school	1,582	40.1	5,615	30.0
Upper secondary school	1,541	39.1	7,167	38.3
University degree	730	18.5	5,383	28.7
Missing	89	2.3	563	3.0
Paternal education				
Compulsory school	1,273	32.3	4,402	23.5
Upper secondary school	1,890	48.0	9,095	48.6
University degree	482	12.2	3,912	20.9
Missing	297	7.5	1,319	7.0
Maternal income				
Q1	1,173	29.8	4,887	26.1
Q2	1,281	32.5	5,843	31.2
Q3	931	23.6	4,585	24.5
Q4	486	12.3	2,988	15.9
Missing	71	1.8	425	2.3
Paternal income				
Q1	1,127	28.6	3,917	20.9
Q2	760	19.3	3,128	16.7
Q3	889	22.5	4,389	23.4
Q4	864	21.9	5,986	32.0
Missing	302	7.7	1,308	7.0
Maternal occupational status				
Occupation	2,963	75.2	15,405	82.2
No occupation	916	23.2	2,956	15.8
Missing	63	1.6	367	2.0
Paternal occupational status				
Occupation	2,868	72.8	14,906	79.6
No occupation	800	20.3	2,627	14.0
Missing	274	6.9	1,195	6.4

Data are n % if not else stated. Abbreviation: SES, socioeconomic status; Q, quartile.

*Median with interquartile ranges

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215 **Results**

216 In total, 3,942 individuals in the childhood obesity cohort and 18,728 individuals in the
217 comparison group were included in the study (Table 1). In both groups, 46% of the
218 participants were girls and the median age at follow-up was 23.4 years. The proportions of
219 individuals of Nordic origin were similar in both groups (73.7% vs. 75.0%; $p=0.02$). Despite
220 the groups being matched for living area, a greater proportion of children with obesity grew
221 up in households with low SES compared with the comparison group (22.0% vs. 14.4%;
222 $p<0.0001$).

224 **Parental SES, childhood obesity and the child's attained level of** 225 **education**

226 In the childhood obesity cohort, 56.7% completed ≥ 12 years in school, compared with 74.4%
227 in the comparison group. Girls more frequently completed ≥ 12 years of schooling than boys
228 in both groups (S2 Table).

229 Higher parental SES was positively associated with completion of ≥ 12 years of schooling in
230 both the childhood obesity cohort and in the comparison group (Fig 2). The odds were more
231 than five times higher among children growing up in high SES households compared with
232 those growing up in low SES households (Table 2). However, even when taking parental
233 SES and other risk factors into account, individuals in the childhood obesity cohort were
234 almost half as likely to complete ≥ 12 school years compared with those in the comparison
235 group (Table 2). The adjusted odds ratio (^aOR) [99% CI] to complete ≥ 12 years of schooling

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3 236 for the childhood obesity cohort versus the comparison group were lower in the higher level
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5 237 of SES: ^aOR low parental SES = 0.69 [0.50 to 0.95], p=0.0026; ^aOR medium-low parental
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7 238 SES = 0.59 [0.48 to 0.72], p<0.0001; ^aOR medium-high parental SES = 0.46 [0.35 to 0.60],
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9 239 p<0.0001; ^aOR high parental SES = 0.27 [0.14 to 0.54], p<0.0001. P-value for interaction
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11 240 test for childhood obesity and parental SES reaches a p of 0.0015.
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15 241 The association of parental SES on school performance was more pronounced in the
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17 242 comparison group than in the childhood obesity cohort. For example, stratified analyses show
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19 243 that adjusted OR of completing ≥ 12 years in school in low SES compared with high SES in
20
21 244 the comparison group was 0.17 (99% CI 0.14 to 0.21, p<0.0001) and in the childhood obesity
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23 245 cohort 0.31 (99% CI 0.22 to 0.45, p<0.0001). Regardless of how we divide the childhood
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25 246 obesity population (into age- or calendar year at start of obesity treatment, SES sub-scores),
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27 247 large differences between the two groups remain (Table 2 and S3 Table).
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34 249 **Fig 2. Percentage of individuals completing ≥ 12 years of schooling by SES level in the**
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36 250 **childhood obesity cohort (n=3,921, solid bars) and the comparison group (n=18,528,**
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38 251 **dotted bars).**
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Table 2. Crude and adjusted odds ratio (99% CI); p-value of subjects completing ≥12 years of schooling

	Crude estimates	Model 1, n=22 449	Model 2, n=20 610	Model 3, n=20 691	Model 4, n=20 885	Model 5, n=20 420
Childhood obesity cohort vs. comparison group	0.44 (0.40 to 0.48); <0.0001	0.57 (0.51 to 0.63); <0.0001	0.55 (0.50 to 0.62); <0.0001	0.54 (0.48 to 0.61); <0.0001	0.53 (0.48 to 0.59); <0.0001	0.59 (0.52 to 0.66); <0.0001
Non-Nordic vs. Nordic	0.70 (0.63 to 0.78); <0.0001	0.85 (0.75 to 0.97); 0.001	0.77 (0.67 to 0.88); <0.0001	0.87 (0.76 to 0.99); 0.0068	0.81 (0.71 to 0.92); <0.0001	0.97 (0.84 to 1.12); 0.5687
ADHD/ADD vs. non-ADHD/ADD	0.19 (0.17 to 0.23); <0.0001	0.28 (0.24 to 0.33); <0.0001	0.27 (0.22 to 0.33); <0.0001	0.27 (0.22 to 0.32); <0.0001	0.26 (0.22 to 0.31); <0.0001	0.28 (0.23 to 0.34); <0.0001
Anxiety/depression vs. no anxiety/depression	0.31 (0.27 to 0.35); <0.0001	0.39 (0.34 to 0.45); <0.0001	0.36 (0.31 to 0.41); <0.0001	0.38 (0.33 to 0.44); <0.0001	0.39 (0.33 to 0.44); <0.0001	0.36 (0.31 to 0.42); <0.0001
Parental SES						
Medium-low vs. low parental SES	1.74 (1.54 to 1.97); <0.0001	1.69 (1.48 to 1.93); <0.0001				
Medium-high vs. low parental SES	3.33 (2.92 to 3.81); <0.0001	2.99 (2.59 to 3.45); <0.0001				
High vs. low parental SES	6.21 (5.19 to 7.44); <0.0001	5.40 (4.45 to 6.55); <0.0001				
Maternal education						
Upper secondary school vs. compulsory school	1.88 (1.69 to 2.08); <0.0001		1.63 (1.45 to 1.83); <0.0001			1.51 (1.34 to 1.71); <0.0001
University degree vs. compulsory school	3.23 (2.86 to 3.66); <0.0001		2.45 (2.12 to 2.83); <0.0001			2.17 (1.87 to 2.53); <0.0001
Paternal education						
Upper secondary school vs. compulsory school	1.88 (1.69 to 2.09); <0.0001		1.58 (1.40 to 1.77); <0.0001			1.42 (1.26 to 1.60); <0.0001
University degree vs. compulsory school	3.07 (2.66 to 3.55); <0.0001		2.09 (1.77 to 2.45); <0.0001			1.81 (1.53 to 2.15); <0.0001
Maternal income						
Q2 vs. Q1	1.28 (1.15 to 1.43); <0.0001			1.25 (1.10 to 1.41); <0.0001		1.07 (0.93 to 1.23); 0.2065
Q3 vs. Q1	1.45 (1.28 to 1.63); <0.0001			1.35 (1.17 to 1.55); <0.0001		1.05 (0.90 to 1.23); 0.4224
Q4 vs. Q1	1.86 (1.60 to 2.15); <0.0001			1.52 (1.28 to 1.81); <0.0001		1.07 (0.88 to 1.28); 0.3831
Paternal income						
Q2 vs. Q1	1.36 (1.19 to 1.55); <0.0001			1.31 (1.13 to 1.51); <0.0001		1.07 (0.91 to 1.26); 0.2740
Q3 vs. Q1	1.90 (1.67 to 2.17); <0.0001			1.70 (1.48 to 1.96); <0.0001		1.24 (1.05 to 1.46); 0.0009
Q4 vs. Q1	2.95 (2.58 to 3.36); <0.0001			2.47 (2.13 to 2.86); <0.0001		1.58 (1.33 to 1.88); <0.0001
Maternal occupational status						
Occupation vs. no occupation	2.15 (1.92 to 2.40); <0.0001				1.72 (1.51 to 1.96); <0.0001	1.38 (1.19 to 1.61); <0.0001
Paternal occupational status						
Occupation vs. no occupation	2.24 (1.99 to 2.52); <0.0001				1.79 (1.57 to 2.04); <0.0001	1.41 (1.20 to 1.65); <0.0001

Abbreviations: CI, confidence interval; SES, socioeconomic status.

Model 1: Variables included were group (childhood obesity cohort vs. comparison group), migration background, ADHD/ADD, anxiety/depression, and parental SES

Model 2: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal education

Model 3: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal income

Model 4: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal occupational status.

Model 5: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal education, income, and occupational status.

253 Of note, having a non-Nordic-, compared to a Nordic background, was associated with
254 reduced odds to complete 12 or more years of schooling (Table 2). This was however not
255 observed in boys in the childhood obesity cohort ($p=0.68$). Further, excluding individuals
256 with ADHD/ADD or anxiety/depression in sensitivity analyses, did not alter the association
257 between childhood obesity and attained level of education.

258 **Degree of obesity and treatment response on completed** 259 **educational level in children with obesity**

260 In the childhood obesity cohort, the mean age at start of treatment was 13.5 years (99% CI:
261 13.45 to 13.62) and the mean BMI SDS was +2.91 (99% CI: 2.89 to 2.92). The median
262 treatment duration was 2.83 years (IQR: 1.86 to 4.42) and the mean treatment response,
263 calculated using BMI SDS from the first to the last clinical visit, was -0.13 (99% CI: -0.15
264 to -0.10, $n=2,709$, 68.7%, dropouts excluded). At baseline, 50.7% had obesity and 49.3% had
265 morbid obesity. At last measured weight and height, 33.4% had obesity and 47.2% had
266 morbid obesity, while 19.4% of the children no longer had obesity (dropouts excluded).

267 A greater degree of obesity at start of treatment lowered the odds of completing ≥ 12 years of
268 schooling, ^aOR [99% CI] = 0.51 [0.40 to 0.64], $p<0.0001$, per one unit increase in BMI SDS,
269 while age at start of obesity treatment did not influence the outcome (Table 3). Treatment
270 response was categorized as good response ($n=847$), no response ($n=1\ 315$), poor response
271 ($n=547$), and dropouts ($n=1,233$). Of those with good treatment response, 67% completed
272 ≥ 12 years in school compared with 58%, 52%, and 50% in the groups with no or poor
273 response, and dropouts, respectively ($p<0.0001$). Within all SES groups, except for high SES,
274 greater treatment response resulted in higher odds of completing ≥ 12 years in school (Fig 3).

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3 275 Dropouts were less likely to complete ≥ 12 years in school compared with non-responders
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5 276 (Table 3) but this was not observed in the high SES group (Fig 3). Duration of treatment
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7 277 differed between children with good and poor treatment response (3.5 vs. 4.0 years;
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9 278 $p < 0.0001$). In the childhood obesity cohort, the association between parental SES and odds
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11 279 of completing ≥ 12 years of schooling was not modified by treatment response ($p = 0.603$).
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Table 3. Crude and adjusted odds ratios of completing ≥ 12 years of schooling in the childhood obesity cohort

	Odds ratio (99% CI) performed with ordinary logistic regression; p value		
	Crude estimates	Model 1, n=3 921	Model 2, n=3 575
Sex (girls vs. boys)	1.26 (1.06 to 1.48); 0.0004	1.30 (1.08 to 1.56); 0.0002	1.29 (1.06 to 1.56); 0.0008
Migration background (non-Nordic vs. Nordic)	0.78 (0.65 to 0.94); 0.0006	0.74 (0.60 to 0.91); 0.0002	0.69 (0.55 to 0.87); <0.0001
ADHD/ADD (yes vs. no)	0.31 (0.25 to 0.40); <0.0001	0.35 (0.27 to 0.45); <0.0001	0.33 (0.26 to 0.44); <0.0001
Anxiety/depression (yes vs. no)	0.43 (0.35 to 0.53); <0.0001	0.45 (0.36 to 0.57); <0.0001	0.43 (0.34 to 0.55); <0.0001
BMI SDS at start of treatment	0.47 (0.38 to 0.58); <0.0001	0.51 (0.40 to 0.64); <0.0001	0.48 (0.38 to 0.62); <0.0001
Age at start of treatment	0.96 (0.93 to 1.01); 0.0234	1.01 (0.96 to 1.06); 0.60	1.01 (0.96 to 1.06); 0.805
Treatment response			
Good response vs. no response	1.46 (1.15 to 1.85); <0.0001	1.34 (1.04 to 1.72); 0.0031	1.28 (0.98 to 1.67); 0.0182
Poor response vs. no response	0.78 (0.60 to 1.02); 0.0167	0.72 (0.54 to 0.96); 0.0028	0.71 (0.53 to 0.96); 0.003
Dropouts vs. no response	0.71 (0.58 to 0.87); <0.0001	0.69 (0.55 to 0.86); <0.0001	0.68 (0.54 to 0.87); <0.0001
Parental SES			
Medium-low vs. low parental SES	1.63 (1.31 to 2.03); <0.0001	1.56 (1.24 to 1.98); <0.0001	
Medium-high vs. low parental SES	2.40 (1.90 to 3.05); <0.0001	2.19 (1.69 to 2.83); <0.0001	
High vs. low parental SES	2.99 (2.11 to 4.22); <0.0001	2.83 (1.95 to 4.12); <0.0001	
Maternal education			
Upper secondary school vs. compulsory school	1.74 (1.44 to 2.09); <0.0001		1.57 (1.27 to 1.94); <0.0001
University degree vs. compulsory school	2.47 (1.94 to 3.16); <0.0001		2.23 (1.67 to 2.98); <0.0001
Paternal education			
Upper secondary school vs. compulsory school	1.48 (1.23 to 1.79); <0.0001		1.26 (1.03 to 1.55); 0.004
University degree vs. compulsory school	1.92 (1.44 to 2.56); <0.0001		1.41 (1.01 to 1.95); 0.0076

Abbreviations: CI, confidence interval; BMI SDS, BMI standard deviation score; SES, socioeconomic status.

Model 1: Variables included were sex, migration background, ADHD/ADD, anxiety/depression, BMI SDS- and age at start of treatment, treatment response, and parental SES.

Model 2: Variables included were sex, migration background, ADHD/ADD, anxiety/depression, BMI SDS- and age at start of treatment, treatment response, maternal-, and paternal education.

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3 281 **Fig 3. Crude percentage of individuals with obesity in childhood (n=3,921) completing ≥ 12**
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5 282 **school years by parental SES and treatment response (black bars=dropouts; white**
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7 283 **bars=poor response; striped bars=no response; dotted bars=good response). p=p for**
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9 284 **trend; n=refers to number of individuals in each category.**
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16 286 **Discussion**

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20 287 In this prospective cohort study, we have compared level of education among individuals
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22 288 recorded in the Swedish Childhood Obesity Treatment Register, and a comparison group
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24 289 matched by sex, year of birth, and living area. Individuals in the childhood obesity cohort were
25
26 290 half as likely to complete 12 or more years of schooling, independently of parental
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28 291 socioeconomic status (SES).

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32 292 Among individuals from high SES families, those in the childhood obesity cohort were
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34 293 approximately one third as likely to complete ≥ 12 years of schooling as individuals from high
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36 294 SES families in the comparison group. Furthermore, our results indicate that parental SES was
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38 295 more important to complete ≥ 12 school years in the comparison group than in the childhood
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40 296 obesity cohort. An association between obesity and impaired academic achievements has been
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42 297 demonstrated before.^{8 9} To which extent other psychosocial factors contribute to this finding
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44 298 has not been evaluated. It is well established that low parental SES,¹³⁻¹⁵ immigrant background,⁹
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46 299 ¹⁴ ADHD/ADD,^{9 17} and depression,^{9 14 16} may negatively affect performance in school. These
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48 300 factors are also overrepresented in the paediatric population with obesity. In this study, we can
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50 301 confirm that low parental SES, neuropsychiatric disorders, and anxiety/depression are more
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52 302 common in children with obesity and contribute to the decreased odds of completing ≥ 12 school
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54 303 years. However, we found that obesity in childhood is a considerable risk factor for not
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3 304 completing ≥ 12 years of schooling even after taking these and other important risk factors into
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5 305 account.

306 **Obesity treatment outcome and educational level**

307 We identified a positive association between weight-loss during obesity treatment and
308 completed educational level, which confirms our previously reported data unadjusted for SES
309 ⁹. Children with good treatment response, compared with those with no response, were more
310 likely to complete ≥ 12 school years in all SES groups. A decrease of 0.25 BMI SDS, that has
311 previously been shown to improve metabolic health ²⁷, we can now also show may have a
312 positive association on completing ≥ 12 years of schooling. However, the positive effects of
313 successful obesity treatment did not compensate for the observed SES differences on attained
314 level of education.

315 In Sweden, 12 years of schooling is not mandatory, but the long-term social effects are
316 considerable for those who fail. It has been estimated that 50% of those who fail to complete
317 ≥ 12 school years face a situation of being left outside the society with poor psychosocial health
318 and large costs for the society ²⁹. Thus, it is possible that school failure will further worsen the
319 health perspective of adolescents with obesity.

320 **Potential mechanisms between obesity, weight loss, and school** 321 **performance**

322 The mechanisms by which obesity influences school performance are unclear but are likely
323 multifactorial and complex. Possible mechanisms may include psychosocial aspects such as
324 stigma³⁰ and increased risk for anxiety and depression.⁷ Physiological mechanisms may be
325 mediated by anatomical changes such as atrophy of the frontal lobe and reduced hippocampal
326 volume have been demonstrated in individuals with obesity.³¹ Some of the reported cognitive

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3 327 dysfunctions in children and adolescents with obesity include slower response times when
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5 328 performing visuospatial attention tasks,³² diminished executive functions, such as working
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7 329 memory,³³ and slower cognitive performance speed³⁴. In addition, obesity-related
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9 330 comorbidities such as insulin resistance,³⁵ type 2 diabetes mellitus,³⁶ chronic low-grade
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11 331 inflammation,³⁷ and the metabolic syndrome³⁸ have also been associated with impaired
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13 332 executive function, memory performance, attention, and cognitive flexibility. A high fat diet in
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15 333 rodents demonstrated detrimental effects on memory and executive functions.³¹

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17 334 At least some of these effects seem to be reversible, which may add to the positive associations
18
19 335 we observed of weight loss on school outcome. Weight loss may have a direct positive effect
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21 336 on cognitive functions.³⁹ Extensive weight loss via bariatric surgery improves insulin sensitivity
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23 337 and decreases systematic inflammation,⁴⁰ and it has been suggested that these factors affect
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25 338 cognitive functions.⁴¹

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27 339 However, children with obesity are often stigmatized,⁴² have a low self-esteem, and are exposed
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29 340 to bullying and social exclusion.⁴³ All these factors have most likely a negative impact on school
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31 341 performance. In contrast, a strong social network is most probably an important factor both for
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33 342 good treatment response and achievement in school. An inverse association has been observed
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35 343 between familial social support and child weight status⁴⁴ Thus, it is likely that both negative
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37 344 social effects of obesity and obesity-related morbidity, as well as genetic factors, contribute to
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39 345 the adverse association of childhood obesity on completed educational level.

346 **Limitations**

347 Using longitudinal data from several national registers provided an opportunity to assess the
348 impact of obesity on completed educational level, adjusted for several important confounders.
349 Data on both exposure and outcome were prospectively collected and defined according to
350 standardized international classifications.^{19 26}

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3 351 However, some important limitations should be recognized. We did not have anthropometric
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5 352 data on children in the comparison group. There is no representative national data on children
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7 353 with obesity in Sweden. As our comparison group includes children with obesity, although
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9 354 likely less than 1% according to obesity diagnoses found in the National Patient Register, odds
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11 355 of lower level of education associated with obesity might be underestimated. It is also important
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13 356 to consider that children receiving obesity treatment may not be representative of all children
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15 357 with obesity. However, such bias is reduced as a large proportion of children are referred to
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17 358 treatment from school. It should be noted that parental SES was based on data from one specific
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19 359 year in the child's life and not over the child's entire adolescent lifetime, and that the SES
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21 360 indicators used may not reflect the whole SES spectrum. In addition, the impact of anxiety and
22
23 361 depression on educational level may be underestimated since these conditions often are under-
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25 362 diagnosed. Lastly, factors such as free education, school lunches, and students' health care may
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27 363 have an impact on the generalizability of our data to other populations.
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34 **Conclusion**

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37 365 In this longitudinal, population-based study, individuals with obesity in childhood were less
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39 366 likely to complete 12 or more years of schooling, compared with a group from the general
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41 367 population. The odds associated with obesity remained significantly increased even after taking
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43 368 parental SES and other important risk factors, such as ADHD, into consideration. The
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45 369 underlying mechanisms are unclear but previous studies indicate that the effects of obesity both
46
47 370 involve psychosocial effects and cognitive functions. The negative impact of childhood obesity
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49 371 on educational level could partly be reversed by successful obesity treatment in childhood.
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51 372 Results from this study underline the wide effects of childhood obesity on public health and the
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53 373 importance of continued efforts to reduce the prevalence of obesity in children. It is of clinical
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3 374 importance to increase awareness of the potential need for extra support at school of children
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5 375 and adolescents with obesity.
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7

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11
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13
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16
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21 22 23 381 **Author contributions**

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27 382 Conceptualization: Louise Lindberg, Pernilla Danielsson, Martina Persson, Claude Marcus,
28
29 383 Emilia Hagman.

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31
32 384 Data curation: Louise Lindberg.

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34 385 Formal analysis: Louise Lindberg, Emilia Hagman.

35
36 386 Funding acquisition: Louise Lindberg, Claude Marcus, Emilia Hagman.

37
38 387 Methodology: Louise Lindberg, Emilia Hagman.

39
40 388 Project administration: Louise Lindberg, Emilia Hagman.

41
42
43 389 Supervision: Martina Persson, Emilia Hagman, Pernilla Danielsson, Claude Marcus.

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45 390 Writing – original draft: Louise Lindberg.

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47
48 391 Writing – review & editing: Louise Lindberg, Pernilla Danielsson, Martina Persson, Claude

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50 392 Marcus, Emilia Hagman.

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402 **Data Sharing statement**

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16 403 The data that support the findings of this study contains sensitive information. Restrictions
17
18 404 therefore apply to the availability of these data, which were used under license for the current
19
20 405 study, and so are not publicly available. According to Swedish law and the General Data
21
22 406 Protection Regulation, the authors are not permitted to share the datasets used in this study with
23
24 407 third parties. Given that an ethical approval is obtained, any individual may apply for data from
25
26 408 Statistics Sweden via information@scb.se, the Swedish National Board of Health and Welfare
27
28 409 via registerservice@socialstyrelsen.se, and the Swedish Childhood Obesity Treatment Register
29
30 410 via <http://www.e-boris.se/kontaktuppgifter/>.

36 411 **Competing interests**

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40
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42
43 414 submitted work; no financial relationships with any organisations that might have an interest in
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45 415 the submitted work in the previous three years; no other relationships or activities that could
46
47 416 appear to have influenced the submitted.

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4 547 **Supporting information**
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7 548 **S1 Table. International Classification of Diseases (ICD 10th revision) codes and**
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9 549 **Anatomical Therapeutic Chemical (ATC) classification system codes used.**
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12 550 **S2 Table. Proportion of children completing ≥ 12 years of schooling.**
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15 551 **S3 Table. Proportion of individuals who have undergone obesity treatment in childhood**
16 **who completed ≥ 12 years of schooling by calendar year and by age at start of obesity**
17 **treatment (n=3,942).**
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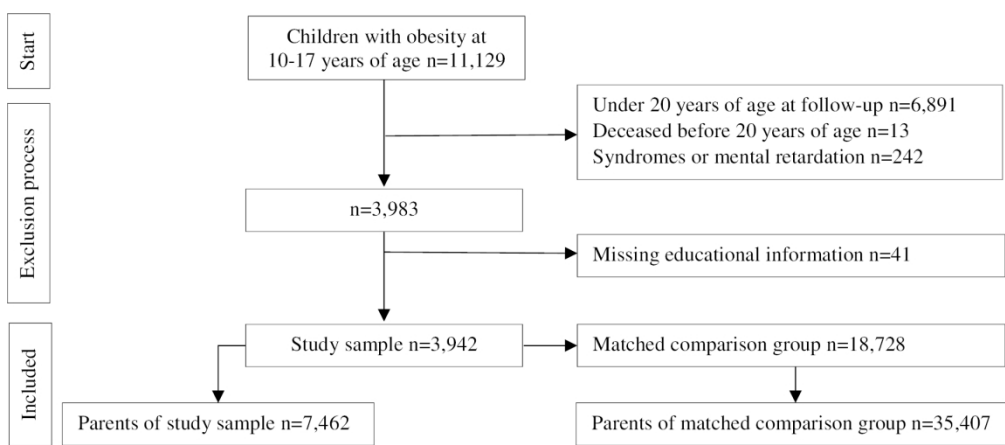
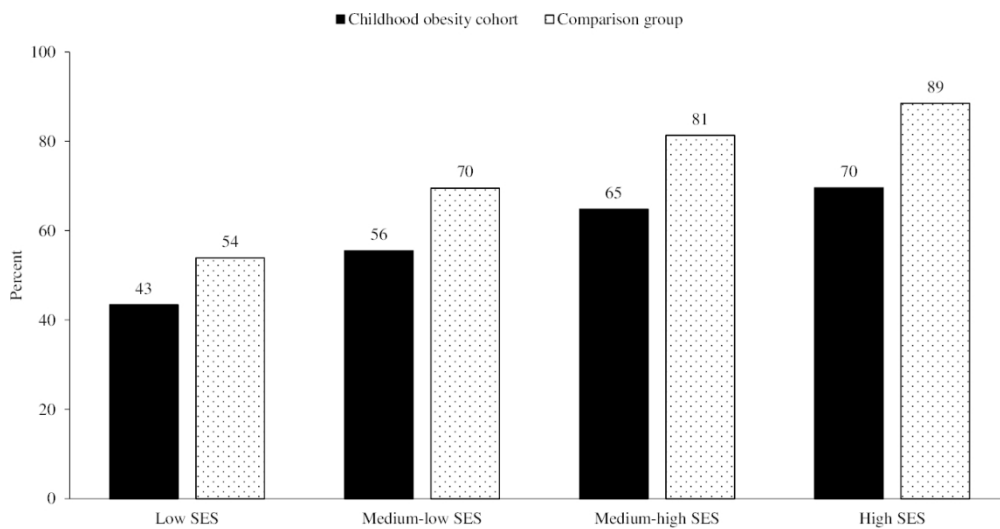


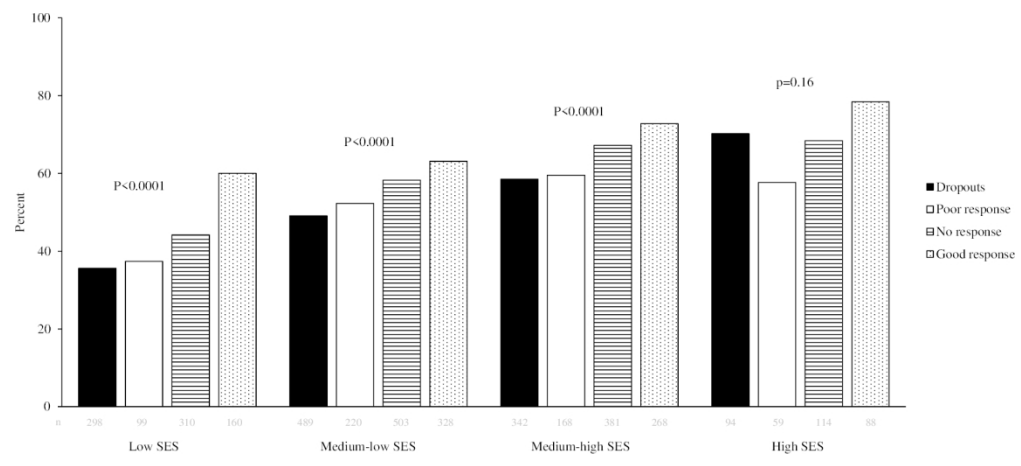
Figure 1

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S1 Table. International Classification of Diseases (ICD 10th revision) codes and Anatomical Therapeutic Chemical (ATC) classification system codes used.

Condition	ICD-10	ATC Code
Anxiety/sedatives	F40.0-F40.1, F41-F42, F51.4-F51.9, F93.0-F93.2	N05B
Depression/antidepressants	F32-F39, F92	N06A
ADHD/ADD	F90	N06B
Mental retardation	F70-F79	
Genetic syndromes		
Fragile X	Q99.2	
Klinefelter	Q98	
Laurence-Moon-Bardet-Biedl	Q87.8B	
Mb Down	Q90	
Noonan	Q87.1E	
Prader-Willi	Q87.1F	
Silver Russell	Q87.1G	
Turner	Q96	

S2 Table. Proportion of children completing ≥ 12 years of schooling

	Childhood obesity cohort		Comparison group		p-value [¶]
	n	%	n	%	
Total	2,236	56.7	13,939	74.4	<0.0001
Sex					
Girl	1,090	59.7	6,843	78.7	<0.0001
Boy	1,146	54.1	7,096	70.8	<0.0001
Migration background					
Nordic	1,695	58.4	10,974	78.1	<0.0001
Non-Nordic	541	52.2	2,965	63.4	<0.0001
ADHD/ADD					
Non-ADHD/ADD	2,032	61.1	13,528	76.5	<0.0001
ADHD/ADD	204	33.1	411	39.4	0.01
Anxiety/depression					
No anxiety/depression	1,902	61.1	12,754	77.0	<0.0001
Anxiety/depression	334	40.2	1,185	54.9	<0.0001
Parental SES					
Low	376	43.4	1,455	53.9	<0.0001
Medium-low	855	55.5	4,187	69.5	<0.0001
Medium-high	751	64.8	5,358	81.3	<0.0001
High	247	69.6	2,848	88.5	<0.0001
Maternal education					
Compulsory school	755	47.7	3,474	61.9	<0.0001
Upper secondary school	945	61.3	5,582	77.9	<0.0001
University degree	506	69.3	4,589	85.3	<0.0001
Paternal education					
Compulsory school	639	50.2	2,775	63.0	<0.0001
Upper secondary school	1,132	59.9	7,085	77.9	<0.0001
University degree	318	66.0	3,333	85.2	<0.0001
Maternal income					
Q1	614	52.3	3,327	68.1	<0.0001
Q2	750	58.6	4,391	75.2	<0.0001
Q3	542	58.2	3,534	77.1	<0.0001
Q4	304	62.6	2,429	81.3	<0.0001
Paternal income					
Q1	556	49.3	2,453	62.6	<0.0001
Q2	410	54.0	2,260	72.3	<0.0001
Q3	549	61.8	3,404	77.6	<0.0001
Q4	572	66.2	5,056	84.5	<0.0001
Maternal occupational status					
No occupation	438	47.8	1,722	58.3	<0.0001
Occupation	1,777	60.0	12,005	77.9	<0.0001
Paternal occupational status					
No occupation	363	45.4	1,543	58.7	<0.0001
Occupation	1,736	60.5	11,703	78.5	<0.0001

Abbreviations: SES, socioeconomic status; Q, quartile. [¶]Performed with chi-square.

1 EUR \approx 9.83 SEK December 31st 2017. Q1: <18,652 EUR; Q2: 18,652 to 25,025 EUR; Q3: 25,026 to 32,309 EUR; Q4: >32,309 EUR.

S3 Table. Proportion of individuals who have undergone obesity treatment in childhood who completed ≥ 12 years of schooling by calendar year and by age at start of obesity treatment (n=3,942).

Individuals completing ≥ 12 years of schooling		
	<u>n</u>	<u>%</u>
Calendar year at start of obesity treatment		
1995-2001	229	62.6
2002-2008	1,262	58.1
2009-2014	745	53.1
Age at start of obesity treatment		
10 - 12.99	960	59.0
13 - 16.99	1,276	55.1

STROBE Statement—Checklist of items that should be included in reports of *cohort studies*

	Item No	Recommendation	Page No
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract (b) Provide in the abstract an informative and balanced summary of what was done and what was found	1-2 2-3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4
Objectives	3	State specific objectives, including any prespecified hypotheses	4.5
Methods			
Study design	4	Present key elements of study design early in the paper	5
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5-9
Participants	6	(a) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up (b) For matched studies, give matching criteria and number of exposed and unexposed	5 5
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	7-9
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	7-9
Bias	9	Describe any efforts to address potential sources of bias	7-9
Study size	10	Explain how the study size was arrived at	5
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	9-10
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding (b) Describe any methods used to examine subgroups and interactions (c) Explain how missing data were addressed (d) If applicable, explain how loss to follow-up was addressed (e) Describe any sensitivity analyses	9-10
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed (b) Give reasons for non-participation at each stage (c) Consider use of a flow diagram	12 + fig
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders (b) Indicate number of participants with missing data for each variable of interest (c) Summarise follow-up time (eg, average and total amount)	11
Outcome data	15*	Report numbers of outcome events or summary measures over time	12

1	Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	12-17
2			(b) Report category boundaries when continuous variables were categorized	
3			(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	
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9	Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	12-17
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11	Discussion			
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13	Key results	18	Summarise key results with reference to study objectives	18
14	Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	20-21
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16	Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	18-21
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19	Generalisability	21	Discuss the generalisability (external validity) of the study results	21
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21	Other information			
22	Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	22-23
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*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at <http://www.plosmedicine.org/>, Annals of Internal Medicine at <http://www.annals.org/>, and Epidemiology at <http://www.epidem.com/>). Information on the STROBE Initiative is available at <http://www.strobe-statement.org>.

BMJ Open

Obesity in childhood, socioeconomic status, and completion of 12 or more school years: a prospective cohort study

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4 1 Obesity in childhood, socioeconomic status, and completion of 12 or
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7 2 more school years: a prospective cohort study
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11 4 Louise Lindberg¹, Martina Persson^{2,3,4}, Pernilla Danielsson¹, Emilia Hagman^{1*}, Claude
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19
20 7 ¹Department of Clinical Science, Intervention and Technology, Division of Pediatrics,
21 8 Karolinska Institutet, Stockholm, Sweden
22

23 9 ²Department of Medicine Solna, Clinical Epidemiology Unit, Karolinska Institutet,
24 10 Stockholm, Sweden
25

26 11 ³Department of Diabetes and Endocrinology, Sachska Children's Hospital Södersjukhuset,
27 12 Stockholm, Sweden
28

29
30 13 ⁴Department of Clinical Science and Education, Södersjukhuset, Karolinska Institutet,
31 14 Stockholm, Sweden
32

33 15 *Corresponding author e-mail: emilia.hagman@ki.se (EH)
34

35 16 Other contributing authors e-mail: louise.lindberg@ki.se; martina.persson@ki.se;
36 17 pernilla.danielsson.liljeqvist@ki.se; claudio.marcus@ki.se
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38 18 Keywords: Pediatric obesity, schools, epidemiology, social class, cohort study
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40 19 Word count: 3722
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20 Abstract

21 **Objectives:** Children with obesity achieve lower educational level compared with normal-
22 weight peers. Parental socioeconomic status (SES) impacts both a child's academic
23 achievement and risk of obesity. The degree to which the association between obesity and
24 education depend on parental SES is unclear. Therefore, the primary aim is to investigate if
25 individuals with obesity in childhood are less likely to complete ≥ 12 years of schooling,
26 independently of parental SES. The secondary aim is to study how weight loss, level of
27 education, and parental SES are associated.

28 **Design:** Nationwide prospective cohort study.

29 **Setting:** Swedish national register data.

30 **Participants:** Children aged 10-17 years, recorded in the Swedish Childhood Obesity
31 Treatment Register (BORIS), and aged 20 years or older at follow-up were included
32 (n=3,942). A comparison group was matched by sex, year of birth, and living area
33 (n=18,728). Parental SES was based on maternal and paternal level of education, income,
34 and occupational status.

35 **Primary outcome measure:** Completion of ≥ 12 years of schooling was analysed with
36 conditional logistic regression, and adjusted for group, migration background, ADHD/ADD,
37 anxiety/depression, and parental SES.

38 **Results:** Among those with obesity in childhood, 56.7% completed ≥ 12 school years
39 compared with 74.4% in the comparison group ($p < 0.0001$). High parental SES compared
40 with low SES was strongly associated with attained level of education in both children with
41 and without obesity, adjusted odds ratio (^aOR) [99% confidence interval (CI)]=5.40 [4.45 to
42 6.55]. However, obesity in childhood remains a strong risk factor of not completing ≥ 12

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3 43 school years, independently of parental SES, ^aOR=0.57 [0.51 to 0.63]. Successful obesity
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5 44 treatment increased the odds of completing ≥ 12 years in school even when taking parental
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8 45 SES into account, ^aOR=1.34 [1.04 to 1.72].
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10 46 **Conclusions:** Individuals with obesity in childhood have lower odds of completing ≥ 12
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12 47 school years, independently of parental SES. Optimized obesity treatment may improve
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15 48 school results in this group.
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20 50 **Strengths and limitation of this study**

- 23 51 • In this prospective cohort study, we have been able to investigate the level of
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25 52 education among a large number of individuals who have obesity in childhood (n=
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27 53 3,942) in comparison with a matched group (n=18,728).
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- 30 54 • The study design of using longitudinal data from several national registers provided
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32 55 the opportunity to control for important confounding factors, such as neuropsychiatric
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34 56 disorders, anxiety, depression, and family socioeconomic status.
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- 37 57 • Factors such as free education, school lunches, and students' health care may have an
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39 58 impact on the generalizability of our data to other populations.
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60 Introduction

61 During the last 40 years, the prevalence of childhood obesity has increased exponentially in
62 many parts of the world.¹ Childhood obesity is associated with increased risks of somatic
63 morbidity and risk of premature death in adulthood.²⁻⁵ Psychosocial maladjustment, anxiety
64 and depression are also prevalent^{6 7} and may contribute to the obesity-related long-term
65 morbidity and mortality.⁵ Most studies report that children with obesity more often have
66 lower school grades and reach a lower level of education compared with normal-weight
67 peers,⁸⁻¹⁰ but in a recent study, only girls with obesity were affected.¹¹ We have previously
68 confirmed the association between obesity and lower attained level of education among both
69 girls and boys.⁹ We also found that successful obesity treatment was positively correlated
70 with completing ≥ 12 school years, although without taking socioeconomic status (SES) into
71 account.⁹

72 Low parental SES is a well-established risk factor for both childhood obesity and poorer
73 academic achievement.¹² In particular, parental education has been demonstrated to influence
74 the child's performance at school.¹³⁻¹⁵ In addition, depression¹⁶ and neuropsychiatric
75 disorders, including Attention Deficit Disorder with or without Hyperactivity
76 (ADHD/ADD),¹⁷ may adversely affect school results. Depression and neuropsychiatric
77 disorders are more common in children with obesity compared to the general population.^{9 18}
78 Investigating the impact of obesity alone on attained level of education requires that these
79 and other confounders are considered. The primary aim of this study was to disentangle the
80 association of childhood obesity and parental SES on completed level of education. The

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3 81 secondary aim was to study if positive effects of weight loss on attained level of education is
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5 82 affected by parental SES.
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8 83 **Methods**

9 84 **Study population**

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16 85 This prospective cohort study included children with obesity,¹⁹ aged 10-17 years at the start
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18 86 of obesity treatment (December 1994 to December 2015), and aged 20 years or older and
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21 87 living in Sweden at follow-up (December 31st 2017; n=3,942). Data on subjects receiving
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23 88 treatment for childhood obesity was collected from the Swedish Childhood Obesity
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25 89 Treatment Register (BORIS). BORIS has been thoroughly described elsewhere,²⁰ but in
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28 90 short: The main purpose of the register is quality assessment and long-term evaluation of
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30 91 childhood obesity treatment. Treatment is individualized and consists primarily of lifestyle
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32 92 modification (i.e. diet and physical activity).
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35 93 A comparison group from the general population was randomly identified using the Swedish
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37 94 Total Population Register and matched by sex, year of birth, and living area at the year
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39 95 obesity treatment was initiated (n=18,728). Using density matching without replacement,
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41 96 five individuals were matched to each individual from the childhood obesity cohort. Siblings
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43 97 of children registered in the childhood obesity cohort were excluded from the comparison
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45 98 group. Children with a diagnosis of mental retardation or genetic syndromes were excluded
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48 99 from both the childhood obesity cohort and the comparison group (Fig 1 and S1 Table).
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3 101 Families were informed in written or verbal about data collection in the Swedish Childhood
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5 102 Obesity Treatment Register. Post an opt-out approval by parents/guardians, data of the
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7 103 children's weight and height were recorded by the local health care provider during treatment
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9 104 visits. There was no data of weight and height of individuals in the comparison group.
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11 105 However, less than 1% of the individuals in the comparison group were found in the National
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13 106 Patient Register with a diagnosis of obesity in childhood. In Sweden, health care is free of
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15 107 charge for children and adolescents until 18 years of age. The study was approved by the
16
17 108 regional Ethics Committee in Stockholm, Sweden (No. 2016/922-31/1).

109 **Data sources**

110 Using the Swedish identity number, which is unique to each resident in Sweden, data from
111 several national registers were linked.²¹ Data on education, income, and occupational status
112 were obtained from the Longitudinal Integration Database for Health Insurance and Labour
113 Market Studies. Information on migration background for children and their parents was
114 obtained from the Swedish Total Population Register.²² Both these registers are held by
115 Statistics Sweden, a governmental agency that collects and provide official statistics
116 (www.scb.se/en).

117 Data on diagnoses of mental retardation, genetic syndromes, ADHD/ADD, anxiety, and
118 depression were identified based on codes according the International Classification of
119 Diseases (10th revision; S1 Table) and retrieved from the National Patient Register.²³
120 Information on prescriptions of psychostimulant drugs for ADHD/ADD as well as
121 antidepressants and tranquilizers for anxiety and depression were identified using the
122 Anatomical Therapeutic Chemical classification system (S1 Table) retrieved from the

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3 123 Swedish Prescribed Drug Register.²⁴ Data on deaths were retrieved from the Cause of Death
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5 124 Register.²⁵ These registers are held and were linked by the governmental agency the National
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8 125 Board of Health and Welfare (www.socialstyrelsen.se/english).

11 126 **Definition of outcome**

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14 127 The main outcome was defined as completion of ≥ 12 years of schooling, and based on the
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16 128 International Standard Classification of Education.²⁶ In Sweden, children start school at the
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18 129 age of 6 or 7 years and attend compulsory school for 9 years. Upper secondary school
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20 130 includes three additional years of schooling and provides the requirements to attend higher
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22 131 education. Usually students graduate from upper secondary school at 18 or 19 years of age.
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24 132 All education in Sweden is free of charge. Students in compulsory and upper secondary
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26 133 school are provided school lunches and health care at schools, also free of charge.

31 134 **Definition of exposure variables**

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35 135 Degree of obesity in children from BORIS was assessed with Body Mass Index Standard
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37 136 Deviation Score (BMI SDS), which is standardized by sex and age and used to measure
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39 137 degree of obesity in growing children.¹⁹ Baseline measures (continuous) were used for the
40
41 138 variable BMI SDS at start of treatment. Response to obesity treatment was based on the
42
43 139 change of BMI SDS from the first to the last clinical visit and categorized into four groups:³
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45 140 ²⁷ good response, a reduction of BMI SDS by 0.25 units or more; no response, a change of
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47 141 BMI SDS by +/- 0.24 units; poor response, an increase of BMI SDS by 0.25 units or more;
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49 142 and dropouts, children with less than one year between their first and last measure or without
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51 143 clinical follow-up after their first registered visit.
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3 144 Parental SES was based on maternal and paternal level of education, income, and
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5 145 occupational status at the year the child turned 15 years, which is about the same time as the
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7 146 child starts upper secondary school. In case of the child was adopted, the SES of adoptive
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10 147 parents was used (childhood obesity cohort n=24 and comparison group n=164). The
11
12 148 rationale of treating SES as a composite variable was to capture more of the social context
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14 149 and a potential inequality embedded there. Thus, by taking three variables into account
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16 150 instead of one, we get a more wide and robust measure of SES. Maternal and paternal
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18 151 educational level was categorized into compulsory school, upper secondary school, or
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20 152 university degree. Annual disposable income was used to reflect maternal and paternal
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22 153 economic capacity. The annual disposable income includes all taxable (direct labour income,
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24 154 capital gains from shares etc.) and tax-free income (housing and child benefits, student aid
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26 155 etc.), minus final tax and other negative transfers such as capital loss from shares and
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28 156 properties. Disposable income from different years was converted to 2017 prices using the
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30 157 Consumer Price Index for Sweden provided by Statistics Sweden (www.scb.se/en). Income
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32 158 was categorized into quartiles based on data from the parents in the comparison group. No
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34 159 occupation was defined as either unemployment 6 months or more, or income from long-
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36 160 term sick leave exceeding any income from the individual's gross salary. Individuals
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38 161 considered to have an occupation included those registered as employed or having an income
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40 162 from student grants/loan equivalent to full-time studies for at least one semester. Parental
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42 163 SES was weighted according to maternal and paternal education (0, 1, 2), income (0, 1, 2, 3),
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44 164 and occupational status (0, 1). The mean parental SES score (i.e. the sum of the mother's and
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46 165 father's all SES indicators divided by two) was applied to their child. The SES-variable was
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3 166 categorized into four levels; low SES (0 to 1.5 points), medium-low SES (2 to 3 points),
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5 167 medium-high SES (3.5 to 4.5 points), and high SES (5 to 6 points).
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8 168 The prevalence of both ADHD/ADD⁹ and depression¹⁸ is higher in children with obesity and
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10 169 may negatively influence attained level of education.^{9 16 17} ADHD/ADD and
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12 170 anxiety/depression in children were identified based on diagnosis or dispensed prescribed
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14 171 medication (S1 Table).
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18 172 **Definition of covariates**

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22 173 The migration background of children in Sweden may impact school achievements and is
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24 174 therefore an important factor to control for in the analyses.^{9 14} Migration background was
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26 175 categorized as Nordic or non-Nordic. Nordic was defined as born in a Nordic country
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28 176 (Sweden, Norway, Denmark, Finland, or Iceland) with one or two parents born in the Nordic.
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30 177 Children were classified as non-Nordic if born outside the Nordic or born in the Nordic with
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32 178 two parents born outside the Nordic. Other covariates included sex and age (continuous) at
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34 179 start of obesity treatment.
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39 180 **Patient and public involvement**

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42 181 No patient involved.
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49 183 **Statistical analysis**

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53 184 Descriptive statistics are presented as means and confidence intervals (CI), medians and
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55 185 interquartile ranges (IQR), or frequencies and percentages. Conditional logistic regression
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3 186 was used to calculate odds ratios (OR's) with 99% CI for the main outcome, i.e. completing
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5 187 ≥ 12 school years. Conditional logistic regression was used above ordinary logistic regression
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7 188 as the childhood obesity cohort and the comparison group were matched by several variables.
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10 189 Independent variables included in the adjusted analyses were migration background,
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12 190 ADHD/ADD, anxiety/depression, parental SES, and group (childhood obesity cohort or
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14 191 comparison group) in analyses including both groups, otherwise stratified by group.
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17 192 Interaction between childhood obesity and parental SES was tested. The association of each
18
19 193 SES indicator on the odds of completing ≥ 12 school years was also analysed separately for
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21 194 mothers and fathers. Sensitivity analyses excluding individuals with a non-Swedish
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23 195 background (defined as child born in Sweden with at least one parent also born in Sweden),
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25
26 196 or ADHD/ADD, or anxiety/depression were performed.
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29 197 Secondary analyses were performed within the childhood obesity cohort to examine
30
31 198 associations between patient characteristics and completed educational level. In ordinary
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33 199 logistic regressions, odds were adjusted for sex, migration background, parental SES, age
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35 200 and BMI SDS at start of treatment, treatment response, ADHD/ADD, and
36
37 201 anxiety/depression. We tested for possible interaction between parental SES and treatment
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39 202 response for the odds of completing ≥ 12 years of schooling.
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43 203 As missing data on parental SES was rare in both groups (Table 1), records with missing data
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45 204 were excluded from the analyses, i.e. data were not imputed. P-values < 0.01 were considered
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47 205 statistically significant. All analyses were performed using SAS statistical software (version
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49 206 9.4, Cary, NC, USA).
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Table 1. Characteristics of the participants (n=22,670)

	Childhood obesity cohort (n=3,942)		Comparison group (n=18,728)	
	n	%	n	%
Female sex	1,825	46.3	8,701	46.5
Nordic	2,905	73.7	14,048	75.0
Age at end of follow-up (years)	23.4*	21.4-26.3*	23.4*	21.5-26.3*
ADHD/ADD	617	15.7	1,044	5.6
Anxiety/Depression	831	21.1	2,158	11.5
Parental SES				
Low	867	22.0	2,700	14.4
Medium-low	1,540	39.1	6,023	32.1
Medium-high	1,159	29.4	6,587	35.2
High	355	9.0	3,218	17.2
Missing	21	0.5	200	1.1
Maternal education				
Compulsory school	1,582	40.1	5,615	30.0
Upper secondary school	1,541	39.1	7,167	38.3
University degree	730	18.5	5,383	28.7
Missing	89	2.3	563	3.0
Paternal education				
Compulsory school	1,273	32.3	4,402	23.5
Upper secondary school	1,890	48.0	9,095	48.6
University degree	482	12.2	3,912	20.9
Missing	297	7.5	1,319	7.0
Maternal income				
Q1	1,173	29.8	4,887	26.1
Q2	1,281	32.5	5,843	31.2
Q3	931	23.6	4,585	24.5
Q4	486	12.3	2,988	15.9
Missing	71	1.8	425	2.3
Paternal income				
Q1	1,127	28.6	3,917	20.9
Q2	760	19.3	3,128	16.7
Q3	889	22.5	4,389	23.4
Q4	864	21.9	5,986	32.0
Missing	302	7.7	1,308	7.0
Maternal occupational status				
Occupation	2,963	75.2	15,405	82.2
No occupation	916	23.2	2,956	15.8
Missing	63	1.6	367	2.0
Paternal occupational status				
Occupation	2,868	72.8	14,906	79.6
No occupation	800	20.3	2,627	14.0
Missing	274	6.9	1,195	6.4

Data are n % if not else stated. Abbreviation: SES, socioeconomic status; Q, quartile.

*Median with interquartile ranges

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211 **Results**

212 In total, 3,942 individuals in the childhood obesity cohort and 18,728 individuals in the
213 comparison group were included in the study (Table 1). In both groups, 46% of the
214 participants were girls and the median age at follow-up was 23.4 years. The proportions of
215 individuals of Nordic origin were similar in both groups (73.7% vs. 75.0%; $p=0.02$). Despite
216 the groups being matched for living area, a greater proportion of children with obesity grew
217 up in households with low SES compared with the comparison group (22.0% vs. 14.4%;
218 $p<0.0001$).

219 **Parental SES, childhood obesity and the child's attained level of** 220 **education**

221 In the childhood obesity cohort, 56.7% completed ≥ 12 years in school, compared with 74.4%
222 in the comparison group. Girls more frequently completed ≥ 12 years of schooling than boys
223 in both groups (S2 Table).

224 Higher parental SES was positively associated with completion of ≥ 12 years of schooling in
225 both the childhood obesity cohort and in the comparison group (Fig 2). The odds were more
226 than five times higher among children growing up in high SES households compared with
227 those growing up in low SES households (Table 2). However, even when taking parental
228 SES and other risk factors into account, individuals in the childhood obesity cohort were
229 almost half as likely to complete ≥ 12 school years compared with those in the comparison
230 group (Table 2). The adjusted odds ratio (^aOR) [99% CI] to complete ≥ 12 years of schooling
231 stratified by parental SES, when comparing the childhood obesity cohort with the comparison

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3 232 group, showed a trend towards lower OR in the higher level of SES: low parental SES = 0.69
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5 233 [0.50 to 0.95], $p=0.0026$; medium-low parental SES = 0.59 [0.48 to 0.72], $p<0.0001$;
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7 234 medium-high parental SES = 0.46 [0.35 to 0.60], $p<0.0001$; high parental SES = 0.27 [0.14
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10 235 to 0.54], $p<0.0001$. P-value for interaction test for childhood obesity and parental SES
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12 236 reaches a p of 0.0015.

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15 237 The association of parental SES on school performance was more pronounced in the
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17 238 comparison group than in the childhood obesity cohort. For example, stratified analyses show
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19 239 that adjusted OR of completing ≥ 12 years in school in low SES compared with high SES in
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21 240 the comparison group was 0.17 (99% CI 0.14 to 0.21, $p<0.0001$) and in the childhood obesity
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23 241 cohort 0.31 (99% CI 0.22 to 0.45, $p<0.0001$). Regardless of how we divide the childhood
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25 242 obesity population (into age- or calendar year at start of obesity treatment, SES sub-scores),
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27 243 large differences between the two groups remain (Table 2 and S3 Table).

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Table 2. Crude and adjusted odds ratio (99% CI); p-value of subjects completing ≥12 years of schooling

	Crude estimates	Model 1, n=22 449	Model 2, n=20 610	Model 3, n=20 691	Model 4, n=20 885	Model 5, n=20 420
Childhood obesity cohort vs. comparison group	0.44 (0.40 to 0.48); <0.0001	0.57 (0.51 to 0.63); <0.0001	0.55 (0.50 to 0.62); <0.0001	0.54 (0.48 to 0.61); <0.0001	0.53 (0.48 to 0.59); <0.0001	0.59 (0.52 to 0.66); <0.0001
Non-Nordic vs. Nordic	0.70 (0.63 to 0.78); <0.0001	0.85 (0.75 to 0.97); 0.001	0.77 (0.67 to 0.88); <0.0001	0.87 (0.76 to 0.99); 0.0068	0.81 (0.71 to 0.92); <0.0001	0.97 (0.84 to 1.12); 0.5687
ADHD/ADD vs. non-ADHD/ADD	0.19 (0.17 to 0.23); <0.0001	0.28 (0.24 to 0.33); <0.0001	0.27 (0.22 to 0.33); <0.0001	0.27 (0.22 to 0.32); <0.0001	0.26 (0.22 to 0.31); <0.0001	0.28 (0.23 to 0.34); <0.0001
Anxiety/depression vs. no anxiety/depression	0.31 (0.27 to 0.35); <0.0001	0.39 (0.34 to 0.45); <0.0001	0.36 (0.31 to 0.41); <0.0001	0.38 (0.33 to 0.44); <0.0001	0.39 (0.33 to 0.44); <0.0001	0.36 (0.31 to 0.42); <0.0001
Parental SES						
Medium-low vs. low parental SES	1.74 (1.54 to 1.97); <0.0001	1.69 (1.48 to 1.93); <0.0001				
Medium-high vs. low parental SES	3.33 (2.92 to 3.81); <0.0001	2.99 (2.59 to 3.45); <0.0001				
High vs. low parental SES	6.21 (5.19 to 7.44); <0.0001	5.40 (4.45 to 6.55); <0.0001				
Maternal education						
Upper secondary school vs. compulsory school	1.88 (1.69 to 2.08); <0.0001		1.63 (1.45 to 1.83); <0.0001			1.51 (1.34 to 1.71); <0.0001
University degree vs. compulsory school	3.23 (2.86 to 3.66); <0.0001		2.45 (2.12 to 2.83); <0.0001			2.17 (1.87 to 2.53); <0.0001
Paternal education						
Upper secondary school vs. compulsory school	1.88 (1.69 to 2.09); <0.0001		1.58 (1.40 to 1.77); <0.0001			1.42 (1.26 to 1.60); <0.0001
University degree vs. compulsory school	3.07 (2.66 to 3.55); <0.0001		2.09 (1.77 to 2.45); <0.0001			1.81 (1.53 to 2.15); <0.0001
Maternal income						
Q2 vs. Q1	1.28 (1.15 to 1.43); <0.0001			1.25 (1.10 to 1.41); <0.0001		1.07 (0.93 to 1.23); 0.2065
Q3 vs. Q1	1.45 (1.28 to 1.63); <0.0001			1.35 (1.17 to 1.55); <0.0001		1.05 (0.90 to 1.23); 0.4224
Q4 vs. Q1	1.86 (1.60 to 2.15); <0.0001			1.52 (1.28 to 1.81); <0.0001		1.07 (0.88 to 1.28); 0.3831
Paternal income						
Q2 vs. Q1	1.36 (1.19 to 1.55); <0.0001			1.31 (1.13 to 1.51); <0.0001		1.07 (0.91 to 1.26); 0.2740
Q3 vs. Q1	1.90 (1.67 to 2.17); <0.0001			1.70 (1.48 to 1.96); <0.0001		1.24 (1.05 to 1.46); 0.0009
Q4 vs. Q1	2.95 (2.58 to 3.36); <0.0001			2.47 (2.13 to 2.86); <0.0001		1.58 (1.33 to 1.88); <0.0001
Maternal occupational status						
Occupation vs. no occupation	2.15 (1.92 to 2.40); <0.0001				1.72 (1.51 to 1.96); <0.0001	1.38 (1.19 to 1.61); <0.0001
Paternal occupational status						
Occupation vs. no occupation	2.24 (1.99 to 2.52); <0.0001				1.79 (1.57 to 2.04); <0.0001	1.41 (1.20 to 1.65); <0.0001

Abbreviations: CI, confidence interval; SES, socioeconomic status.

Model 1: Variables included were group (childhood obesity cohort vs. comparison group), migration background, ADHD/ADD, anxiety/depression, and parental SES

Model 2: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal education

Model 3: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal income

Model 4: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal occupational status.

Model 5: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal education, income, and occupational status.

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3 246 Of note, having a non-Nordic-, compared to a Nordic background, was associated with
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5 247 reduced odds to complete 12 or more years of schooling (Table 2). This was however not
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7 248 observed in boys in the childhood obesity cohort ($p=0.68$). Further, excluding individuals
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9 249 with a non-Swedish background, or ADHD/ADD, or anxiety/depression in sensitivity
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11 250 analyses, did not alter the association between childhood obesity and attained level of
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13 251 education. For example, when excluding individuals with a non-Swedish background, the
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15 252 ^aOR to complete ≥ 12 years in school for the obesity cohort vs. the comparison group was
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17 253 0.55 [0.48 to 0.63], $p < 0.0001$.

22 254 **Degree of obesity and treatment response on completed**

23 255 **educational level in children with obesity**

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29 256 In the childhood obesity cohort, the mean age at start of treatment was 13.5 years (99% CI:
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31 257 13.45 to 13.62) and the mean BMI SDS was +2.91 (99% CI: 2.89 to 2.92). The median
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33 258 treatment duration was 2.83 years (IQR: 1.86 to 4.42) and the mean treatment response,
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35 259 calculated using BMI SDS from the first to the last clinical visit, was -0.13 (99% CI: -0.15
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37 260 to -0.10, $n=2,709$, 68.7%, dropouts excluded). At baseline, 50.7% had obesity and 49.3% had
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39 261 morbid obesity. At last measured weight and height, 33.4% had obesity and 47.2% had
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41 262 morbid obesity, while 19.4% of the children no longer had obesity (dropouts excluded).

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46 263 A greater degree of obesity at start of treatment lowered the odds of completing ≥ 12 years of
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48 264 schooling, ^aOR [99% CI] = 0.51 [0.40 to 0.64], $p < 0.0001$, per one unit increase in BMI SDS,
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50 265 while age at start of obesity treatment did not influence the outcome (Table 3). Treatment
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52 266 response was categorized as good response ($n=847$), no response ($n=1\ 315$), poor response
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54 267 ($n=547$), and dropouts ($n=1,233$). Of those with good treatment response, 67% completed

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3 268 ≥ 12 years in school compared with 58%, 52%, and 50% in the groups with no or poor
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5 269 response, and dropouts, respectively ($p < 0.0001$). Within all SES groups, except for high SES,
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7 270 greater treatment response resulted in higher odds of completing ≥ 12 years in school (Fig 3).
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9 271 Dropouts were less likely to complete ≥ 12 years in school compared with non-responders
10
11 272 (Table 3) but this was not observed in the high SES group (Fig 3). Duration of treatment
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13 273 differed between children with good and poor treatment response (3.5 vs. 4.0 years;
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15 274 $p < 0.0001$). In the childhood obesity cohort, the association between parental SES and odds
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17 275 of completing ≥ 12 years of schooling was not modified by treatment response ($p = 0.603$).
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Table 3. Crude and adjusted odds ratios of completing ≥ 12 years of schooling in the childhood obesity cohort

	Odds ratio (99% CI) performed with ordinary logistic regression; p value		
	Crude estimates	Model 1, n=3 921	Model 2, n=3 575
Sex (girls vs. boys)	1.26 (1.06 to 1.48); 0.0004	1.30 (1.08 to 1.56); 0.0002	1.29 (1.06 to 1.56); 0.0008
Migration background (non-Nordic vs. Nordic)	0.78 (0.65 to 0.94); 0.0006	0.74 (0.60 to 0.91); 0.0002	0.69 (0.55 to 0.87); <0.0001
ADHD/ADD (yes vs. no)	0.31 (0.25 to 0.40); <0.0001	0.35 (0.27 to 0.45); <0.0001	0.33 (0.26 to 0.44); <0.0001
Anxiety/depression (yes vs. no)	0.43 (0.35 to 0.53); <0.0001	0.45 (0.36 to 0.57); <0.0001	0.43 (0.34 to 0.55); <0.0001
BMI SDS at start of treatment	0.47 (0.38 to 0.58); <0.0001	0.51 (0.40 to 0.64); <0.0001	0.48 (0.38 to 0.62); <0.0001
Age at start of treatment	0.96 (0.93 to 1.01); 0.0234	1.01 (0.96 to 1.06); 0.60	1.01 (0.96 to 1.06); 0.805
Treatment response*			
Good response vs. no response	1.46 (1.15 to 1.85); <0.0001	1.34 (1.04 to 1.72); 0.0031	1.28 (0.98 to 1.67); 0.0182
Poor response vs. no response	0.78 (0.60 to 1.02); 0.0167	0.72 (0.54 to 0.96); 0.0028	0.71 (0.53 to 0.96); 0.003
Dropouts vs. no response	0.71 (0.58 to 0.87); <0.0001	0.69 (0.55 to 0.86); <0.0001	0.68 (0.54 to 0.87); <0.0001
Parental SES			
Medium-low vs. low parental SES	1.63 (1.31 to 2.03); <0.0001	1.56 (1.24 to 1.98); <0.0001	
Medium-high vs. low parental SES	2.40 (1.90 to 3.05); <0.0001	2.19 (1.69 to 2.83); <0.0001	
High vs. low parental SES	2.99 (2.11 to 4.22); <0.0001	2.83 (1.95 to 4.12); <0.0001	
Maternal education			
Upper secondary school vs. compulsory school	1.74 (1.44 to 2.09); <0.0001		1.57 (1.27 to 1.94); <0.0001
University degree vs. compulsory school	2.47 (1.94 to 3.16); <0.0001		2.23 (1.67 to 2.98); <0.0001
Paternal education			
Upper secondary school vs. compulsory school	1.48 (1.23 to 1.79); <0.0001		1.26 (1.03 to 1.55); 0.004
University degree vs. compulsory school	1.92 (1.44 to 2.56); <0.0001		1.41 (1.01 to 1.95); 0.0076

Abbreviations: CI, confidence interval; BMI SDS, BMI standard deviation score; SES, socioeconomic status.

* Good response=decrease of BMI SDS ≥ 0.25 units; No response=BMI SDS +/- 0.24 units; Poor response=increase of BMI SDS ≥ 0.25 units; Dropouts=no follow-up measure or less than one year between their first and last measure.

Model 1: Variables included were sex, migration background, ADHD/ADD, anxiety/depression, BMI SDS- and age at start of treatment, treatment response, and parental SES.

Model 2: Variables included were sex, migration background, ADHD/ADD, anxiety/depression, BMI SDS- and age at start of treatment, treatment response, maternal-, and paternal education.

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278 Discussion

279 In this prospective cohort study, we have compared level of education among individuals
280 recorded in the Swedish Childhood Obesity Treatment Register, and a comparison group
281 matched by sex, year of birth, and living area. Individuals in the childhood obesity cohort were
282 half as likely to complete 12 or more years of schooling, independently of parental
283 socioeconomic status (SES).

284 Among individuals from high SES families, those in the childhood obesity cohort were
285 approximately one third as likely to complete ≥ 12 years of schooling as individuals from high
286 SES families in the comparison group. Furthermore, our results indicate that parental SES was
287 more important to complete ≥ 12 school years in the comparison group than in the childhood
288 obesity cohort. An association between obesity and impaired academic achievements has been
289 demonstrated before.^{8 9} To which extent other psychosocial factors contribute to this finding
290 has not been evaluated. It is well established that low parental SES,¹³⁻¹⁵ immigrant background,⁹
291 ¹⁴ ADHD/ADD,^{9 17} and depression,^{9 14 16} may negatively affect performance in school. These
292 factors are also overrepresented in the paediatric population with obesity. In this study, we can
293 confirm that low parental SES, neuropsychiatric disorders, and anxiety/depression are more
294 common in children with obesity and contribute to the decreased odds of completing ≥ 12 school
295 years. However, we found that obesity in childhood is a considerable risk factor for not
296 completing ≥ 12 years of schooling even after taking these and other important risk factors into
297 account.

298 Obesity treatment outcome and educational level

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3 299 We identified a positive association between weight-loss during obesity treatment and
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5 300 completed educational level, which confirms our previously reported data unadjusted for SES.⁹
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7 301 Children with good treatment response, compared with those with no response, were more
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9 302 likely to complete ≥ 12 school years in all SES groups. A decrease of 0.25 BMI SDS, that has
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11 303 previously been shown to improve metabolic health,²⁷ we can now also show may have a
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13 304 positive association on completing ≥ 12 years of schooling. However, the positive effects of
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15 305 successful obesity treatment did not compensate for the observed SES differences on attained
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17 306 level of education.

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22 307 In Sweden, 12 years of schooling is not mandatory, but the long-term social effects are
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24 308 considerable for those who fail. It has been estimated that 50% of those who fail to complete
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26 309 ≥ 12 school years face a situation of being left outside the society with poor psychosocial health
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28 310 and large costs for the society.²⁸ Thus, it is possible that school failure will further worsen the
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30 311 health perspective of adolescents with obesity.

312 **Potential mechanisms between obesity, weight loss, and school** 313 **performance**

314 Several parameters may interplay when measuring school completion, e.g. resourcefulness,
315 intelligence, or ability to conform. Nevertheless, the mechanisms by which obesity influences
316 school performance are unclear but are likely multifactorial and complex. Possible mechanisms
317 may include psychosocial aspects such as stigma²⁹ and increased risk for anxiety and
318 depression.⁷ Physiological mechanisms may be mediated by anatomical changes such as
319 atrophy of the frontal lobe and reduced hippocampal volume have been demonstrated in
320 individuals with obesity.³⁰ Some of the reported cognitive dysfunctions in children and
321 adolescents with obesity include slower response times when performing visuospatial attention
322 tasks,³¹ diminished executive functions, such as working memory,³² and slower cognitive

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3 323 performance speed.³³ In addition, obesity-related comorbidities such as insulin resistance,³⁴
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5 324 type 2 diabetes mellitus,³⁵ chronic low-grade inflammation,³⁶ and the metabolic syndrome³⁷
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7 325 have also been associated with impaired executive function, memory performance, attention,
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9 326 and cognitive flexibility. A high fat diet in rodents demonstrated detrimental effects on memory
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11 327 and executive functions.³⁰
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15 328 At least some of these effects seem to be reversible, which may add to the positive associations
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17 329 we observed of weight loss on school outcome. Weight loss may have a direct positive effect
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19 330 on cognitive functions.³⁸ Extensive weight loss via bariatric surgery improves insulin sensitivity
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21 331 and decreases systematic inflammation,³⁹ and it has been suggested that these factors affect
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23 332 cognitive functions.⁴⁰
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27 333 However, children with obesity are often stigmatized,⁴¹ have a low self-esteem, and are exposed
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29 334 to bullying and social exclusion.⁴² All these factors have most likely a negative impact on school
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31 335 performance. In contrast, a strong social network is most probably an important factor both for
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33 336 good treatment response and achievement in school. An inverse association has been observed
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35 337 between familial social support and child weight status.⁴³ The relationship between BMI and
36
37 338 school completion may also be biased from e.g. assortative mating and dynastic effects which
38
39 339 have shown to reduce causal effects.⁴⁴ There is also a suggested interplay between genetic
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41 340 variants and environmental factors that may affect intelligence.⁴⁵ Thus, it is likely that both
42
43 341 negative social effects of obesity and obesity-related morbidity, as well as genetic factors,
44
45 342 contribute to the adverse association of childhood obesity on completed educational level.^{7 29 41}
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53 344 **Limitations**

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57 345 Using longitudinal data from several national registers provided an opportunity to assess the
58
59 346 impact of obesity on completed educational level, adjusted for several important confounders.
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3 347 Data on both exposure and outcome were prospectively collected and defined according to
4
5 348 standardized international classifications.^{19 26}
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7

8 349 However, some important limitations should be recognized. We did not have anthropometric
9
10 350 data on children in the comparison group. There is no representative national data on children
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12 351 with obesity in Sweden. As our comparison group includes children with obesity, although
13
14 352 likely less than 1% according to obesity diagnoses found in the National Patient Register, odds
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16 353 of lower level of education associated with obesity might be underestimated. It is also important
17
18 354 to consider that children receiving obesity treatment may not be representative of all children
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20 355 with obesity. It should further be noted that parental SES was based on data from one specific
21
22 356 year in the child's life and not over the child's entire adolescent lifetime, and that the SES
23
24 357 indicators used may not reflect the whole SES spectrum. In addition, the impact of anxiety and
25
26 358 depression on educational level may be underestimated since these conditions often are under-
27
28 359 diagnosed. We urge the reader to bear in mind that despite several possible mechanisms have
29
30 360 been proposed, causal relationships of obesity and the effect of treatment on the outcome
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32 361 remains to be established. Lastly, factors such as free education, school lunches, and students'
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34 362 health care may have an impact on the generalizability of our data to other populations.
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41 363 **Conclusion**

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45 364 In this longitudinal, population-based study, individuals with obesity in childhood were less
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47 365 likely to complete 12 or more years of schooling, compared with a group from the general
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49 366 population. The odds associated with obesity remained significantly increased even after taking
50
51 367 parental SES and other important risk factors, such as ADHD, into consideration. The
52
53 368 underlying mechanisms are unclear but previous studies indicate that the effects of obesity on
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55 369 school completion both involve psychosocial effects and cognitive functions.^{16 17 32} The
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57 370 negative impact of childhood obesity on educational level may partly be reversed by successful
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3 371 obesity treatment in childhood. Results from this study underline effects of childhood obesity
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5 372 on schooling. For the sake of an optimal educational environment, it is of great importance to
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7 373 increase awareness both in schools and among decision makers to allocate resources for
8
9 374 potential extra support, e.g. reduce stigma and increase educational support.
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14
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16
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18
19 378 steering committee for establishing and maintaining the register.
20
21
22

23 379 **Author contributions**

24
25 380 Conceptualization: Louise Lindberg, Pernilla Danielsson, Martina Persson, Claude Marcus,
26
27 381 Emilia Hagman.

28
29 382 Data curation: Louise Lindberg.

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31 383 Formal analysis: Louise Lindberg, Emilia Hagman.

32
33 384 Funding acquisition: Louise Lindberg, Claude Marcus, Emilia Hagman.

34
35 385 Methodology: Louise Lindberg, Emilia Hagman.

36
37 386 Project administration: Louise Lindberg, Emilia Hagman.

38
39 387 Supervision: Martina Persson, Emilia Hagman, Pernilla Danielsson, Claude Marcus.

40
41 388 Writing – original draft: Louise Lindberg.

42
43 389 Writing – review & editing: Louise Lindberg, Pernilla Danielsson, Martina Persson, Claude
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8
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13 400 **Data Sharing statement**

15 401 The data that support the findings of this study contains sensitive information. Restrictions
16
17 402 therefore apply to the availability of these data, which were used under license for the current
18
19 403 study, and so are not publicly available. According to Swedish law and the General Data
20
21 404 Protection Regulation, the authors are not permitted to share the datasets used in this study with
22
23 405 third parties. Given that an ethical approval is obtained, any individual may apply for data from
24
25 406 Statistics Sweden via information@scb.se, the Swedish National Board of Health and Welfare
26
27 407 via registerservice@socialstyrelsen.se, and the Swedish Childhood Obesity Treatment Register
28
29 408 via <http://www.e-boris.se/kontaktuppgifter/>.

34 409 **Competing interests**

36 410 All authors have completed the ICMJE uniform disclosure form at
37
38 411 www.icmje.org/coi_disclosure.pdf and declare: no support from any organisation for the
39
40 412 submitted work; no financial relationships with any organisations that might have an interest in
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43
44 414 appear to have influenced the submitted.

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549 **Supporting information**

550 **S1 Table. International Classification of Diseases (ICD 10th revision) codes and**
551 **Anatomical Therapeutic Chemical (ATC) classification system codes used.**

552 **S2 Table. Proportion of children completing ≥ 12 years of schooling.**

553 **S3 Table. Proportion of individuals who have undergone obesity treatment in childhood**
554 **who completed ≥ 12 years of schooling by calendar year and by age at start of obesity**
555 **treatment (n=3,942).**

556

557

558 **Figure Legends**

559 **Fig 1. Participant flowchart.**

560

561 **Fig 2. Percentage of individuals completing ≥ 12 years of schooling by SES level in the**
562 **childhood obesity cohort (n=3,921,) and the comparison group (n=18,528).**

563

564 **Fig 3. Crude percentage of individuals with obesity in childhood (n=3,921) completing ≥ 12**
565 **school years by parental SES and treatment response. p=p for trend; n=refers to number**
566 **of individuals in each category.**

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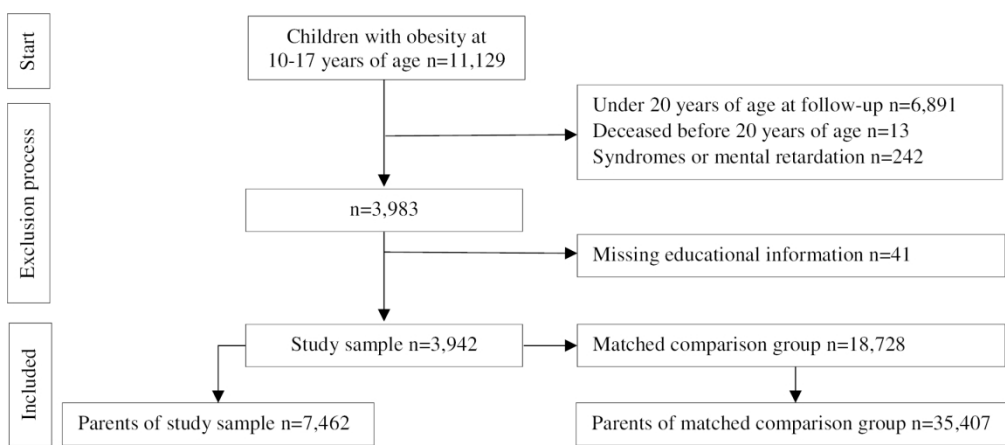


Figure 1

169x73mm (300 x 300 DPI)

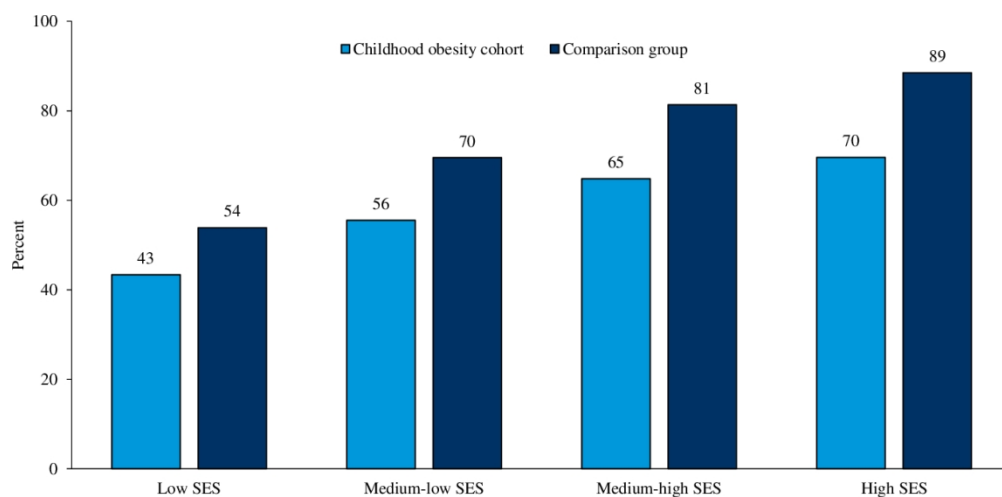


Fig 2. Percentage of individuals completing ≥ 12 years of schooling by SES level in the childhood obesity cohort (n=3,921) and the comparison group (n=18,528).

118x57mm (300 x 300 DPI)

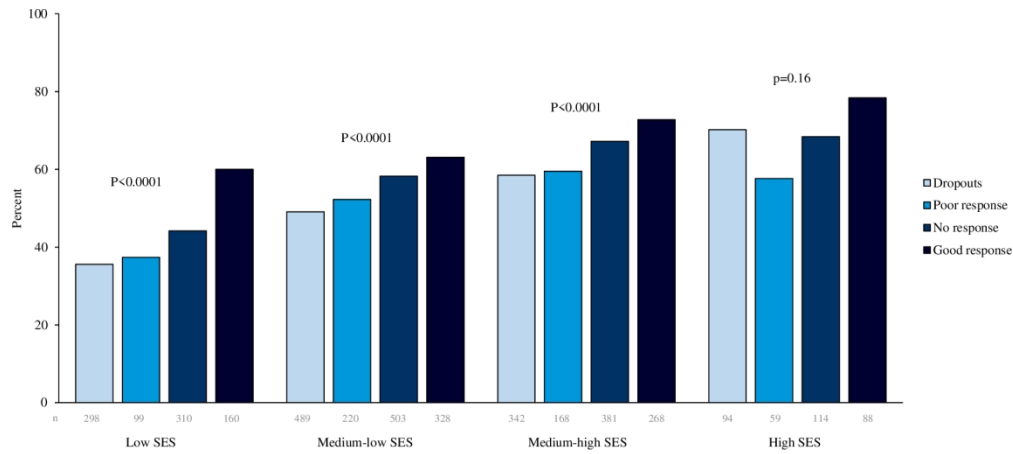


Fig 3. Crude percentage of individuals with obesity in childhood (n=3,921) completing ≥ 12 school years by parental SES and treatment response. p=p for trend; n=refers to number of individuals in each category.

150x66mm (300 x 300 DPI)

S1 Table. International Classification of Diseases (ICD 10th revision) codes and Anatomical Therapeutic Chemical (ATC) classification system codes used.

Condition	ICD-10	ATC Code
Anxiety/sedatives	F40.0-F40.1, F41-F42, F51.4-F51.9, F93.0-F93.2	N05B
Depression/antidepressants	F32-F39, F92	N06A
ADHD/ADD	F90	N06B
Mental retardation	F70-F79	
Genetic syndromes		
Fragile X	Q99.2	
Klinefelter	Q98	
Laurence-Moon-Bardet-Biedl	Q87.8B	
Mb Down	Q90	
Noonan	Q87.1E	
Prader-Willi	Q87.1F	
Silver Russell	Q87.1G	
Turner	Q96	

S2 Table. Proportion of children completing ≥ 12 years of schooling

	Childhood obesity cohort		Comparison group		p-value [¶]
	n	%	n	%	
Total	2,236	56.7	13,939	74.4	<0.0001
Sex					
Girl	1,090	59.7	6,843	78.7	<0.0001
Boy	1,146	54.1	7,096	70.8	<0.0001
Migration background					
Nordic	1,695	58.4	10,974	78.1	<0.0001
Non-Nordic	541	52.2	2,965	63.4	<0.0001
ADHD/ADD					
Non-ADHD/ADD	2,032	61.1	13,528	76.5	<0.0001
ADHD/ADD	204	33.1	411	39.4	0.01
Anxiety/depression					
No anxiety/depression	1,902	61.1	12,754	77.0	<0.0001
Anxiety/depression	334	40.2	1,185	54.9	<0.0001
Parental SES					
Low	376	43.4	1,455	53.9	<0.0001
Medium-low	855	55.5	4,187	69.5	<0.0001
Medium-high	751	64.8	5,358	81.3	<0.0001
High	247	69.6	2,848	88.5	<0.0001
Maternal education					
Compulsory school	755	47.7	3,474	61.9	<0.0001
Upper secondary school	945	61.3	5,582	77.9	<0.0001
University degree	506	69.3	4,589	85.3	<0.0001
Paternal education					
Compulsory school	639	50.2	2,775	63.0	<0.0001
Upper secondary school	1,132	59.9	7,085	77.9	<0.0001
University degree	318	66.0	3,333	85.2	<0.0001
Maternal income					
Q1	614	52.3	3,327	68.1	<0.0001
Q2	750	58.6	4,391	75.2	<0.0001
Q3	542	58.2	3,534	77.1	<0.0001
Q4	304	62.6	2,429	81.3	<0.0001
Paternal income					
Q1	556	49.3	2,453	62.6	<0.0001
Q2	410	54.0	2,260	72.3	<0.0001
Q3	549	61.8	3,404	77.6	<0.0001
Q4	572	66.2	5,056	84.5	<0.0001
Maternal occupational status					
No occupation	438	47.8	1,722	58.3	<0.0001
Occupation	1,777	60.0	12,005	77.9	<0.0001
Paternal occupational status					
No occupation	363	45.4	1,543	58.7	<0.0001
Occupation	1,736	60.5	11,703	78.5	<0.0001

Abbreviations: SES, socioeconomic status; Q, quartile. [¶]Performed with chi-square.

1 EUR \approx 9.83 SEK December 31st 2017. Q1: <18,652 EUR; Q2: 18,652 to 25,025 EUR; Q3: 25,026 to 32,309 EUR; Q4: >32,309 EUR.

S3 Table. Proportion of individuals who have undergone obesity treatment in childhood who completed ≥ 12 years of schooling by calendar year and by age at start of obesity treatment (n=3,942).

Individuals completing ≥ 12 years of schooling		
	n	%
Calendar year at start of obesity treatment		
1995-2001	229	62.6
2002-2008	1,262	58.1
2009-2014	745	53.1
Age at start of obesity treatment		
10 - 12.99	960	59.0
13 - 16.99	1,276	55.1

STROBE Statement—Checklist of items that should be included in reports of *cohort studies*

	Item No	Recommendation	Page No
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract (b) Provide in the abstract an informative and balanced summary of what was done and what was found	1-2 2-3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4
Objectives	3	State specific objectives, including any prespecified hypotheses	4.5
Methods			
Study design	4	Present key elements of study design early in the paper	5
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5-9
Participants	6	(a) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up (b) For matched studies, give matching criteria and number of exposed and unexposed	5 5
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	7-9
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	7-9
Bias	9	Describe any efforts to address potential sources of bias	7-9
Study size	10	Explain how the study size was arrived at	5
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	9-10
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding (b) Describe any methods used to examine subgroups and interactions (c) Explain how missing data were addressed (d) If applicable, explain how loss to follow-up was addressed (e) Describe any sensitivity analyses	9-10
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed (b) Give reasons for non-participation at each stage (c) Consider use of a flow diagram	12 + fig
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders (b) Indicate number of participants with missing data for each variable of interest (c) Summarise follow-up time (eg, average and total amount)	11
Outcome data	15*	Report numbers of outcome events or summary measures over time	12

1	Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	12-17
2			(b) Report category boundaries when continuous variables were categorized	
3			(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	
4				
5				
6				
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8				
9	Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	12-17
10				
11	Discussion			
12				
13	Key results	18	Summarise key results with reference to study objectives	18
14	Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	20-21
15				
16	Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	18-21
17				
18				
19	Generalisability	21	Discuss the generalisability (external validity) of the study results	21
20				
21	Other information			
22	Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	22-23
23				
24				

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at <http://www.plosmedicine.org/>, Annals of Internal Medicine at <http://www.annals.org/>, and Epidemiology at <http://www.epidem.com/>). Information on the STROBE Initiative is available at <http://www.strobe-statement.org>.

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Obesity in childhood, socioeconomic status, and completion of 12 or more school years: a prospective cohort study

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4 1 Obesity in childhood, socioeconomic status, and completion of 12 or
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7 2 more school years: a prospective cohort study
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11 4 Louise Lindberg¹, Martina Persson^{2,3,4}, Pernilla Danielsson¹, Emilia Hagman^{1*}, Claude
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13
14 5 Marcus¹
15
16
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19
20 7 ¹Department of Clinical Science, Intervention and Technology, Division of Pediatrics,
21 8 Karolinska Institutet, Stockholm, Sweden

22
23 9 ²Department of Medicine Solna, Clinical Epidemiology Unit, Karolinska Institutet,
24 10 Stockholm, Sweden

25
26 11 ³Department of Diabetes and Endocrinology, Sachska Children's Hospital Södersjukhuset,
27 12 Stockholm, Sweden

28
29
30 13 ⁴Department of Clinical Science and Education, Södersjukhuset, Karolinska Institutet,
31 14 Stockholm, Sweden

32
33 15 *Corresponding author e-mail: emilia.hagman@ki.se (EH)
34

35 16 Other contributing authors e-mail: louise.lindberg@ki.se; martina.persson@ki.se;
36 17 pernilla.danielsson.liljeqvist@ki.se; claudio.marcus@ki.se
37

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20 Abstract

21 **Objectives:** Children with obesity achieve lower educational level compared with normal-
22 weight peers. Parental socioeconomic status (SES) impacts both a child's academic
23 achievement and risk of obesity. The degree to which the association between obesity and
24 education depend on parental SES is unclear. Therefore, the primary aim is to investigate if
25 individuals with obesity in childhood are less likely to complete ≥ 12 years of schooling,
26 independently of parental SES. The secondary aim is to study how weight loss, level of
27 education, and parental SES are associated.

28 **Design:** Nationwide prospective cohort study.

29 **Setting:** Swedish national register data.

30 **Participants:** Children aged 10-17 years, recorded in the Swedish Childhood Obesity
31 Treatment Register (BORIS), and aged 20 years or older at follow-up were included
32 (n=3,942). A comparison group was matched by sex, year of birth, and living area
33 (n=18,728). Parental SES was based on maternal and paternal level of education, income,
34 and occupational status.

35 **Primary outcome measure:** Completion of ≥ 12 years of schooling was analysed with
36 conditional logistic regression, and adjusted for group, migration background, ADHD/ADD,
37 anxiety/depression, and parental SES.

38 **Results:** Among those with obesity in childhood, 56.7% completed ≥ 12 school years
39 compared with 74.4% in the comparison group (p<0.0001). High parental SES compared
40 with low SES was strongly associated with attained level of education in both children with
41 and without obesity, adjusted odds ratio (aOR) [99% confidence interval (CI)]=5.40 [4.45 to
42 6.55]. However, obesity in childhood remains a strong risk factor of not completing ≥ 12

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3 43 school years, independently of parental SES, ^aOR=0.57 [0.51 to 0.63]. Successful obesity
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5 44 treatment increased the odds of completing ≥ 12 years in school even when taking parental
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8 45 SES into account, ^aOR=1.34 [1.04 to 1.72].
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10 46 **Conclusions:** Individuals with obesity in childhood have lower odds of completing ≥ 12
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12 47 school years, independently of parental SES. Optimized obesity treatment may improve
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15 48 school results in this group.
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20 50 **Strengths and limitation of this study**

- 23 51 • In this prospective cohort study, we have been able to investigate the level of
24
25 52 education among a large number of individuals who have obesity in childhood (n=
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27
28 53 3,942) in comparison with a matched group (n=18,728).
29
- 30 54 • The study design of using longitudinal data from several national registers provided
31
32 55 the opportunity to control for important confounding factors, such as neuropsychiatric
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35 56 disorders, anxiety, depression, and family socioeconomic status.
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- 37 57 • Factors such as free education, school lunches, and students' health care may have an
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39 58 impact on the generalizability of our data to other populations.
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60 Introduction

61 During the last 40 years, the prevalence of childhood obesity has increased exponentially in
62 many parts of the world.¹ Childhood obesity is associated with increased risks of somatic
63 morbidity and risk of premature death in adulthood.²⁻⁵ Psychosocial maladjustment, anxiety
64 and depression are also prevalent^{6 7} and may contribute to the obesity-related long-term
65 morbidity and mortality.⁵ Most studies report that children with obesity more often have
66 lower school grades and reach a lower level of education compared with normal-weight
67 peers,⁸⁻¹⁰ but in a recent study, only girls with obesity were affected.¹¹ We have previously
68 confirmed the association between obesity and lower attained level of education among both
69 girls and boys.⁹ We also found that successful obesity treatment was positively correlated
70 with completing ≥ 12 school years, although without taking socioeconomic status (SES) into
71 account.⁹

72 Low parental SES is a well-established risk factor for both childhood obesity and poorer
73 academic achievement.¹² In particular, parental education has been demonstrated to influence
74 the child's performance at school.¹³⁻¹⁵ In addition, depression¹⁶ and neuropsychiatric
75 disorders, including Attention Deficit Disorder with or without Hyperactivity
76 (ADHD/ADD),¹⁷ may adversely affect school results. Depression and neuropsychiatric
77 disorders are more common in children with obesity compared to the general population.^{9 18}
78 Investigating the impact of obesity alone on attained level of education requires that these
79 and other confounders are considered. The primary aim of this study was to disentangle the
80 association of childhood obesity and parental SES on completed level of education. The

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3 81 secondary aim was to study if positive effects of weight loss on attained level of education is
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5 82 affected by parental SES.
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8 83 **Methods**

9 84 **Study population**

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16 85 This prospective cohort study included children with obesity,¹⁹ aged 10-17 years at the start
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18 86 of obesity treatment (December 1994 to December 2015), and aged 20 years or older and
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21 87 living in Sweden at follow-up (December 31st 2017; n=3,942). Data on subjects receiving
22
23 88 treatment for childhood obesity was collected from the Swedish Childhood Obesity
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25 89 Treatment Register (BORIS). BORIS has been thoroughly described elsewhere,²⁰ but in
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27
28 90 short: The main purpose of the register is quality assessment and long-term evaluation of
29
30 91 childhood obesity treatment. Treatment is individualized and consists primarily of lifestyle
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32 92 modification (i.e. diet and physical activity).
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35 93 A comparison group from the general population was randomly identified using the Swedish
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37 94 Total Population Register and matched by sex, year of birth, and living area at the year
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39 95 obesity treatment was initiated (n=18,728). Using density matching without replacement,
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41 96 five individuals were matched to each individual from the childhood obesity cohort. Siblings
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43 97 of children registered in the childhood obesity cohort were excluded from the comparison
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45 98 group. Children with a diagnosis of mental retardation or genetic syndromes were excluded
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48 99 from both the childhood obesity cohort and the comparison group (Fig 1 and S1 Table).
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3 101 Families were informed in written or verbal about data collection in the Swedish Childhood
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5 102 Obesity Treatment Register. Post an opt-out approval by parents/guardians, data of the
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7 103 children's weight and height were recorded by the local health care provider during treatment
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9 104 visits. There was no data of weight and height of individuals in the comparison group.
10
11 105 However, less than 1% of the individuals in the comparison group were found in the National
12
13 106 Patient Register with a diagnosis of obesity in childhood. In Sweden, health care is free of
14
15 107 charge for children and adolescents until 18 years of age. The study was approved by the
16
17 108 regional Ethics Committee in Stockholm, Sweden (No. 2016/922-31/1).

109 **Data sources**

110 Using the Swedish identity number, which is unique to each resident in Sweden, data from
111 several national registers were linked.²¹ Data on education, income, and occupational status
112 were obtained from the Longitudinal Integration Database for Health Insurance and Labour
113 Market Studies. Information on migration background for children and their parents was
114 obtained from the Swedish Total Population Register.²² Both these registers are held by
115 Statistics Sweden, a governmental agency that collects and provide official statistics
116 (www.scb.se/en).

117 Data on diagnoses of mental retardation, genetic syndromes, ADHD/ADD, anxiety, and
118 depression were identified based on codes according the International Classification of
119 Diseases (10th revision; S1 Table) and retrieved from the National Patient Register.²³
120 Information on prescriptions of psychostimulant drugs for ADHD/ADD as well as
121 antidepressants and tranquilizers for anxiety and depression were identified using the
122 Anatomical Therapeutic Chemical classification system (S1 Table) retrieved from the

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3 123 Swedish Prescribed Drug Register.²⁴ Data on deaths were retrieved from the Cause of Death
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5 124 Register.²⁵ These registers are held and were linked by the governmental agency the National
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8 125 Board of Health and Welfare (www.socialstyrelsen.se/english).

11 126 **Definition of outcome**

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14 127 The main outcome was defined as completion of ≥ 12 years of schooling, and based on the
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16 128 International Standard Classification of Education.²⁶ In Sweden, children start school at the
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18
19 129 age of 6 or 7 years and attend compulsory school for 9 years. Upper secondary school
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21 130 includes three additional years of schooling and provides the requirements to attend higher
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23 131 education. Usually students graduate from upper secondary school at 18 or 19 years of age.
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26 132 All education in Sweden is free of charge. Students in compulsory and upper secondary
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28 133 school are provided school lunches and health care at schools, also free of charge.

31 134 **Definition of exposure variables**

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35 135 Degree of obesity in children from BORIS was assessed with Body Mass Index Standard
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37 136 Deviation Score (BMI SDS), which is standardized by sex and age and used to measure
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39 137 degree of obesity in growing children.¹⁹ Baseline measures (continuous) were used for the
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42 138 variable BMI SDS at start of treatment. Response to obesity treatment was based on the
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44 139 change of BMI SDS from the first to the last clinical visit and categorized into four groups:³
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46 140 ²⁷ good response, a reduction of BMI SDS by 0.25 units or more; no response, a change of
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48 141 BMI SDS by +/- 0.24 units; poor response, an increase of BMI SDS by 0.25 units or more;
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51 142 and dropouts, children with less than one year between their first and last measure or without
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53 143 clinical follow-up after their first registered visit.

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3 144 Parental SES was based on maternal and paternal level of education, income, and
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5 145 occupational status at the year the child turned 15 years, which is about the same time as the
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7 146 child starts upper secondary school. In case of the child was adopted, the SES of adoptive
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10 147 parents was used (childhood obesity cohort n=24 and comparison group n=164). The
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12 148 rationale of treating SES as a composite variable was to capture more of the social context
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15 149 and a potential inequality embedded there. Thus, by taking three variables into account
16
17 150 instead of one, we get a more wide and robust measure of SES. Maternal and paternal
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19 151 educational level was categorized into compulsory school, upper secondary school, or
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21 152 university degree. Annual disposable income was used to reflect maternal and paternal
22
23 153 economic capacity. The annual disposable income includes all taxable (direct labour income,
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25 154 capital gains from shares etc.) and tax-free income (housing and child benefits, student aid
26
27 155 etc.), minus final tax and other negative transfers such as capital loss from shares and
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30 156 properties. Disposable income from different years was converted to 2017 prices using the
31
32
33 157 Consumer Price Index for Sweden provided by Statistics Sweden (www.scb.se/en). Income
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35 158 was categorized into quartiles based on data from the parents in the comparison group. No
36
37 159 occupation was defined as either unemployment 6 months or more, or income from long-
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39 160 term sick leave exceeding any income from the individual's gross salary. Individuals
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42 161 considered to have an occupation included those registered as employed or having an income
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44 162 from student grants/loan equivalent to full-time studies for at least one semester. Parental
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47 163 SES was weighted according to maternal and paternal education (0, 1, 2), income (0, 1, 2, 3),
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49 164 and occupational status (0, 1). The mean parental SES score (i.e. the sum of the mother's and
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51 165 father's all SES indicators divided by two) was applied to their child. The SES-variable was
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3 166 categorized into four levels; low SES (0 to 1.5 points), medium-low SES (2 to 3 points),
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5 167 medium-high SES (3.5 to 4.5 points), and high SES (5 to 6 points).
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8 168 The prevalence of both ADHD/ADD⁹ and depression¹⁸ is higher in children with obesity and
9
10 169 may negatively influence attained level of education.^{9 16 17} ADHD/ADD and
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12 170 anxiety/depression in children were identified based on diagnosis or dispensed prescribed
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14 171 medication (S1 Table).
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18 172 **Definition of covariates**

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22 173 The migration background of children in Sweden may impact school achievements and is
23
24 174 therefore an important factor to control for in the analyses.^{9 14} Migration background was
25
26 175 categorized as Nordic or non-Nordic. Nordic was defined as born in a Nordic country
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28 176 (Sweden, Norway, Denmark, Finland, or Iceland) with one or two parents born in the Nordic.
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30 177 Children were classified as non-Nordic if born outside the Nordic or born in the Nordic with
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32 178 two parents born outside the Nordic. Other covariates included sex and age (continuous) at
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34 179 start of obesity treatment.
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39 180 **Patient and public involvement**

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42 181 No patient involved.
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49 183 **Statistical analysis**

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53 184 Descriptive statistics are presented as means and confidence intervals (CI), medians and
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55 185 interquartile ranges (IQR), or frequencies and percentages. Conditional logistic regression
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3 186 was used to calculate odds ratios (OR's) with 99% CI for the main outcome, i.e. completing
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5 187 ≥ 12 school years. Conditional logistic regression was used above ordinary logistic regression
6
7 188 as the childhood obesity cohort and the comparison group were matched by several variables.
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10 189 Independent variables included in the adjusted analyses were migration background,
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12 190 ADHD/ADD, anxiety/depression, parental SES, and group (childhood obesity cohort or
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14 191 comparison group) in analyses including both groups, otherwise stratified by group.
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17 192 Interaction between childhood obesity and parental SES was tested. The association of each
18
19 193 SES indicator on the odds of completing ≥ 12 school years was also analysed separately for
20
21 194 mothers and fathers. Sensitivity analyses excluding individuals with a non-Swedish
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23 195 background (defined as child born in Sweden with at least one parent also born in Sweden),
24
25
26 196 or ADHD/ADD, or anxiety/depression were performed.
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29 197 Secondary analyses were performed within the childhood obesity cohort to examine
30
31 198 associations between patient characteristics and completed educational level. In ordinary
32
33 199 logistic regressions, odds were adjusted for sex, migration background, parental SES, age
34
35 200 and BMI SDS at start of treatment, treatment response, ADHD/ADD, and
36
37 201 anxiety/depression. We tested for possible interaction between parental SES and treatment
38
39 202 response for the odds of completing ≥ 12 years of schooling.
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44 203 As missing data on parental SES was rare in both groups (Table 1), records with missing data
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46 204 were excluded from the analyses, i.e. data were not imputed. P-values < 0.01 were considered
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48 205 statistically significant. All analyses were performed using SAS statistical software (version
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50 206 9.4, Cary, NC, USA).
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Table 1. Characteristics of the participants (n=22,670)

	Childhood obesity cohort (n=3,942)		Comparison group (n=18,728)	
	n	%	n	%
Female sex	1,825	46.3	8,701	46.5
Nordic	2,905	73.7	14,048	75.0
Age at end of follow-up (years)	23.4*	21.4-26.3*	23.4*	21.5-26.3*
ADHD/ADD	617	15.7	1,044	5.6
Anxiety/Depression	831	21.1	2,158	11.5
Parental SES				
Low	867	22.0	2,700	14.4
Medium-low	1,540	39.1	6,023	32.1
Medium-high	1,159	29.4	6,587	35.2
High	355	9.0	3,218	17.2
Missing	21	0.5	200	1.1
Maternal education				
Compulsory school	1,582	40.1	5,615	30.0
Upper secondary school	1,541	39.1	7,167	38.3
University degree	730	18.5	5,383	28.7
Missing	89	2.3	563	3.0
Paternal education				
Compulsory school	1,273	32.3	4,402	23.5
Upper secondary school	1,890	48.0	9,095	48.6
University degree	482	12.2	3,912	20.9
Missing	297	7.5	1,319	7.0
Maternal income				
Q1	1,173	29.8	4,887	26.1
Q2	1,281	32.5	5,843	31.2
Q3	931	23.6	4,585	24.5
Q4	486	12.3	2,988	15.9
Missing	71	1.8	425	2.3
Paternal income				
Q1	1,127	28.6	3,917	20.9
Q2	760	19.3	3,128	16.7
Q3	889	22.5	4,389	23.4
Q4	864	21.9	5,986	32.0
Missing	302	7.7	1,308	7.0
Maternal occupational status				
Occupation	2,963	75.2	15,405	82.2
No occupation	916	23.2	2,956	15.8
Missing	63	1.6	367	2.0
Paternal occupational status				
Occupation	2,868	72.8	14,906	79.6
No occupation	800	20.3	2,627	14.0
Missing	274	6.9	1,195	6.4

Data are n % if not else stated. Abbreviation: SES, socioeconomic status; Q, quartile.

*Median with interquartile ranges

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210

211 **Results**

212 In total, 3,942 individuals in the childhood obesity cohort and 18,728 individuals in the
213 comparison group were included in the study (Table 1). In both groups, 46% of the
214 participants were girls and the median age at follow-up was 23.4 years. The proportions of
215 individuals of Nordic origin were similar in both groups (73.7% vs. 75.0%; $p=0.02$). Despite
216 the groups being matched for living area, a greater proportion of children with obesity grew
217 up in households with low SES compared with the comparison group (22.0% vs. 14.4%;
218 $p<0.0001$).

219 **Parental SES, childhood obesity and the child's attained level of** 220 **education**

221 In the childhood obesity cohort, 56.7% completed ≥ 12 years in school, compared with 74.4%
222 in the comparison group. Girls more frequently completed ≥ 12 years of schooling than boys
223 in both groups (S2 Table).

224 Higher parental SES was positively associated with completion of ≥ 12 years of schooling in
225 both the childhood obesity cohort and in the comparison group (Fig 2). The odds were more
226 than five times higher among children growing up in high SES households compared with
227 those growing up in low SES households (Table 2). However, even when taking parental
228 SES and other risk factors into account, individuals in the childhood obesity cohort were
229 almost half as likely to complete ≥ 12 school years compared with those in the comparison
230 group (Table 2). The adjusted odds ratio (^aOR) [99% CI] to complete ≥ 12 years of schooling
231 stratified by parental SES, when comparing the childhood obesity cohort with the comparison

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3 232 group, showed a trend towards lower OR in the higher level of SES: low parental SES = 0.69
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5 233 [0.50 to 0.95], $p=0.0026$; medium-low parental SES = 0.59 [0.48 to 0.72], $p<0.0001$;
6
7 234 medium-high parental SES = 0.46 [0.35 to 0.60], $p<0.0001$; high parental SES = 0.27 [0.14
8
9
10 235 to 0.54], $p<0.0001$. P-value for interaction test for childhood obesity and parental SES
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12 236 reaches a p of 0.0015.

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14
15 237 The association of parental SES on school performance was more pronounced in the
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17 238 comparison group than in the childhood obesity cohort (S3 Table). For example, stratified
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19 239 analyses show that adjusted OR of completing ≥ 12 years in school in low SES compared
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21 240 with high SES in the comparison group was 0.17 (99% CI 0.14 to 0.21, $p<0.0001$) and in the
22
23 241 childhood obesity cohort 0.31 (99% CI 0.22 to 0.45, $p<0.0001$). Regardless of how we divide
24
25 242 the childhood obesity population (into age- or calendar year at start of obesity treatment, SES
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27 243 sub-scores), large differences between the two groups remain (Table 2 and S4 Table).

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Table 2. Crude and adjusted odds ratio (99% CI); p-value of subjects completing ≥12 years of schooling

	Crude estimates	Model 1, n=22 449	Model 2, n=20 610	Model 3, n=20 691	Model 4, n=20 885	Model 5, n=20 420
Childhood obesity cohort vs. comparison group	0.44 (0.40 to 0.48); <0.0001	0.57 (0.51 to 0.63); <0.0001	0.55 (0.50 to 0.62); <0.0001	0.54 (0.48 to 0.61); <0.0001	0.53 (0.48 to 0.59); <0.0001	0.59 (0.52 to 0.66); <0.0001
Non-Nordic vs. Nordic	0.70 (0.63 to 0.78); <0.0001	0.85 (0.75 to 0.97); 0.001	0.77 (0.67 to 0.88); <0.0001	0.87 (0.76 to 0.99); 0.0068	0.81 (0.71 to 0.92); <0.0001	0.97 (0.84 to 1.12); 0.5687
ADHD/ADD vs. non-ADHD/ADD	0.19 (0.17 to 0.23); <0.0001	0.28 (0.24 to 0.33); <0.0001	0.27 (0.22 to 0.33); <0.0001	0.27 (0.22 to 0.32); <0.0001	0.26 (0.22 to 0.31); <0.0001	0.28 (0.23 to 0.34); <0.0001
Anxiety/depression vs. no anxiety/depression	0.31 (0.27 to 0.35); <0.0001	0.39 (0.34 to 0.45); <0.0001	0.36 (0.31 to 0.41); <0.0001	0.38 (0.33 to 0.44); <0.0001	0.39 (0.33 to 0.44); <0.0001	0.36 (0.31 to 0.42); <0.0001
Parental SES						
Medium-low vs. low parental SES	1.74 (1.54 to 1.97); <0.0001	1.69 (1.48 to 1.93); <0.0001				
Medium-high vs. low parental SES	3.33 (2.92 to 3.81); <0.0001	2.99 (2.59 to 3.45); <0.0001				
High vs. low parental SES	6.21 (5.19 to 7.44); <0.0001	5.40 (4.45 to 6.55); <0.0001				
Maternal education						
Upper secondary school vs. compulsory school	1.88 (1.69 to 2.08); <0.0001		1.63 (1.45 to 1.83); <0.0001			1.51 (1.34 to 1.71); <0.0001
University degree vs. compulsory school	3.23 (2.86 to 3.66); <0.0001		2.45 (2.12 to 2.83); <0.0001			2.17 (1.87 to 2.53); <0.0001
Paternal education						
Upper secondary school vs. compulsory school	1.88 (1.69 to 2.09); <0.0001		1.58 (1.40 to 1.77); <0.0001			1.42 (1.26 to 1.60); <0.0001
University degree vs. compulsory school	3.07 (2.66 to 3.55); <0.0001		2.09 (1.77 to 2.45); <0.0001			1.81 (1.53 to 2.15); <0.0001
Maternal income						
Q2 vs. Q1	1.28 (1.15 to 1.43); <0.0001			1.25 (1.10 to 1.41); <0.0001		1.07 (0.93 to 1.23); 0.2065
Q3 vs. Q1	1.45 (1.28 to 1.63); <0.0001			1.35 (1.17 to 1.55); <0.0001		1.05 (0.90 to 1.23); 0.4224
Q4 vs. Q1	1.86 (1.60 to 2.15); <0.0001			1.52 (1.28 to 1.81); <0.0001		1.07 (0.88 to 1.28); 0.3831
Paternal income						
Q2 vs. Q1	1.36 (1.19 to 1.55); <0.0001			1.31 (1.13 to 1.51); <0.0001		1.07 (0.91 to 1.26); 0.2740
Q3 vs. Q1	1.90 (1.67 to 2.17); <0.0001			1.70 (1.48 to 1.96); <0.0001		1.24 (1.05 to 1.46); 0.0009
Q4 vs. Q1	2.95 (2.58 to 3.36); <0.0001			2.47 (2.13 to 2.86); <0.0001		1.58 (1.33 to 1.88); <0.0001
Maternal occupational status						
Occupation vs. no occupation	2.15 (1.92 to 2.40); <0.0001				1.72 (1.51 to 1.96); <0.0001	1.38 (1.19 to 1.61); <0.0001
Paternal occupational status						
Occupation vs. no occupation	2.24 (1.99 to 2.52); <0.0001				1.79 (1.57 to 2.04); <0.0001	1.41 (1.20 to 1.65); <0.0001

Abbreviations: CI, confidence interval; SES, socioeconomic status.

Model 1: Variables included were group (childhood obesity cohort vs. comparison group), migration background, ADHD/ADD, anxiety/depression, and parental SES

Model 2: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal education

Model 3: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal income

Model 4: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal occupational status.

Model 5: Variables included were group, migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal education, income, and occupational status.

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3 246 Of note, having a non-Nordic-, compared to a Nordic background, was associated with
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5 247 reduced odds to complete 12 or more years of schooling (Table 2). This was however not
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7 248 observed in boys in the childhood obesity cohort ($p=0.68$). Further, excluding individuals
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9 249 with a non-Swedish background, or ADHD/ADD, or anxiety/depression in sensitivity
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11 250 analyses, did not alter the association between childhood obesity and attained level of
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13 251 education. For example, when excluding individuals with a non-Swedish background, the
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15 252 ^aOR to complete ≥ 12 years in school for the obesity cohort vs. the comparison group was
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17 253 0.55 [0.48 to 0.63], $p < 0.0001$.

22 254 **Degree of obesity and treatment response on completed**

23 255 **educational level in children with obesity**

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29 256 In the childhood obesity cohort, the mean age at start of treatment was 13.5 years (99% CI:
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31 257 13.45 to 13.62) and the mean BMI SDS was +2.91 (99% CI: 2.89 to 2.92). The median
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33 258 treatment duration was 2.83 years (IQR: 1.86 to 4.42) and the mean treatment response,
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35 259 calculated using BMI SDS from the first to the last clinical visit, was -0.13 (99% CI: -0.15
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37 260 to -0.10, $n=2,709$, 68.7%, dropouts excluded). At baseline, 50.7% had obesity and 49.3% had
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39 261 morbid obesity. At last measured weight and height, 33.4% had obesity and 47.2% had
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41 262 morbid obesity, while 19.4% of the children no longer had obesity (dropouts excluded).

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46 263 A greater degree of obesity at start of treatment lowered the odds of completing ≥ 12 years of
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48 264 schooling, ^aOR [99% CI] = 0.51 [0.40 to 0.64], $p < 0.0001$, per one unit increase in BMI SDS,
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50 265 while age at start of obesity treatment did not influence the outcome (Table 3). Treatment
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52 266 response was categorized as good response ($n=847$), no response ($n=1\ 315$), poor response
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54 267 ($n=547$), and dropouts ($n=1,233$). Of those with good treatment response, 67% completed

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3 268 ≥ 12 years in school compared with 58%, 52%, and 50% in the groups with no or poor
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5 269 response, and dropouts, respectively ($p < 0.0001$). Within all SES groups, except for high SES,
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7 270 greater treatment response resulted in higher odds of completing ≥ 12 years in school (Fig 3).
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9 271 Dropouts were less likely to complete ≥ 12 years in school compared with non-responders
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11 272 (Table 3) but this was not observed in the high SES group (Fig 3). Duration of treatment
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13 273 differed between children with good and poor treatment response (3.5 vs. 4.0 years;
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15 274 $p < 0.0001$). In the childhood obesity cohort, the association between parental SES and odds
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17 275 of completing ≥ 12 years of schooling was not modified by treatment response ($p = 0.603$).
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Table 3. Crude and adjusted odds ratios of completing ≥ 12 years of schooling in the childhood obesity cohort

	Odds ratio (99% CI) performed with ordinary logistic regression; p value		
	Crude estimates	Model 1, n=3 921	Model 2, n=3 575
Sex (girls vs. boys)	1.26 (1.06 to 1.48); 0.0004	1.30 (1.08 to 1.56); 0.0002	1.29 (1.06 to 1.56); 0.0008
Migration background (non-Nordic vs. Nordic)	0.78 (0.65 to 0.94); 0.0006	0.74 (0.60 to 0.91); 0.0002	0.69 (0.55 to 0.87); <0.0001
ADHD/ADD (yes vs. no)	0.31 (0.25 to 0.40); <0.0001	0.35 (0.27 to 0.45); <0.0001	0.33 (0.26 to 0.44); <0.0001
Anxiety/depression (yes vs. no)	0.43 (0.35 to 0.53); <0.0001	0.45 (0.36 to 0.57); <0.0001	0.43 (0.34 to 0.55); <0.0001
BMI SDS at start of treatment	0.47 (0.38 to 0.58); <0.0001	0.51 (0.40 to 0.64); <0.0001	0.48 (0.38 to 0.62); <0.0001
Age at start of treatment	0.96 (0.93 to 1.01); 0.0234	1.01 (0.96 to 1.06); 0.60	1.01 (0.96 to 1.06); 0.805
Treatment response*			
Good response vs. no response	1.46 (1.15 to 1.85); <0.0001	1.34 (1.04 to 1.72); 0.0031	1.28 (0.98 to 1.67); 0.0182
Poor response vs. no response	0.78 (0.60 to 1.02); 0.0167	0.72 (0.54 to 0.96); 0.0028	0.71 (0.53 to 0.96); 0.003
Dropouts vs. no response	0.71 (0.58 to 0.87); <0.0001	0.69 (0.55 to 0.86); <0.0001	0.68 (0.54 to 0.87); <0.0001
Parental SES			
Medium-low vs. low parental SES	1.63 (1.31 to 2.03); <0.0001	1.56 (1.24 to 1.98); <0.0001	
Medium-high vs. low parental SES	2.40 (1.90 to 3.05); <0.0001	2.19 (1.69 to 2.83); <0.0001	
High vs. low parental SES	2.99 (2.11 to 4.22); <0.0001	2.83 (1.95 to 4.12); <0.0001	
Maternal education			
Upper secondary school vs. compulsory school	1.74 (1.44 to 2.09); <0.0001		1.57 (1.27 to 1.94); <0.0001
University degree vs. compulsory school	2.47 (1.94 to 3.16); <0.0001		2.23 (1.67 to 2.98); <0.0001
Paternal education			
Upper secondary school vs. compulsory school	1.48 (1.23 to 1.79); <0.0001		1.26 (1.03 to 1.55); 0.004
University degree vs. compulsory school	1.92 (1.44 to 2.56); <0.0001		1.41 (1.01 to 1.95); 0.0076

Abbreviations: CI, confidence interval; BMI SDS, BMI standard deviation score; SES, socioeconomic status.

* Good response=decrease of BMI SDS ≥ 0.25 units; No response=BMI SDS +/- 0.24 units; Poor response=increase of BMI SDS ≥ 0.25 units; Dropouts=no follow-up measure or less than one year between their first and last measure.

Model 1: Variables included were sex, migration background, ADHD/ADD, anxiety/depression, BMI SDS- and age at start of treatment, treatment response, and parental SES.

Model 2: Variables included were sex, migration background, ADHD/ADD, anxiety/depression, BMI SDS- and age at start of treatment, treatment response, maternal-, and paternal education.

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278 **Discussion**

279 In this prospective cohort study, we have compared level of education among individuals
280 recorded in the Swedish Childhood Obesity Treatment Register, and a comparison group
281 matched by sex, year of birth, and living area. Individuals in the childhood obesity cohort were
282 half as likely to complete 12 or more years of schooling, independently of parental
283 socioeconomic status (SES).

284 Among individuals from high SES families, those in the childhood obesity cohort were
285 approximately one third as likely to complete ≥ 12 years of schooling as individuals from high
286 SES families in the comparison group. Furthermore, our results indicate that parental SES was
287 more important to complete ≥ 12 school years in the comparison group than in the childhood
288 obesity cohort. An association between obesity and impaired academic achievements has been
289 demonstrated before.^{8 9} To which extent other psychosocial factors contribute to this finding
290 has not been evaluated. It is well established that low parental SES,¹³⁻¹⁵ immigrant background,⁹
291 ¹⁴ ADHD/ADD,^{9 17} and depression,^{9 14 16} may negatively affect performance in school. These
292 factors are also overrepresented in the paediatric population with obesity. In this study, we can
293 confirm that low parental SES, neuropsychiatric disorders, and anxiety/depression are more
294 common in children with obesity and contribute to the decreased odds of completing ≥ 12 school
295 years. However, we found that obesity in childhood is a considerable risk factor for not
296 completing ≥ 12 years of schooling even after taking these and other important risk factors into
297 account.

298 **Obesity treatment outcome and educational level**

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3 299 We identified a positive association between weight-loss during obesity treatment and
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5 300 completed educational level, which confirms our previously reported data unadjusted for SES.⁹
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7 301 Children with good treatment response, compared with those with no response, were more
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9 302 likely to complete ≥ 12 school years in all SES groups. A decrease of 0.25 BMI SDS, that has
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11 303 previously been shown to improve metabolic health,²⁷ we can now also show may have a
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13 304 positive association on completing ≥ 12 years of schooling. However, the positive effects of
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15 305 successful obesity treatment did not compensate for the observed SES differences on attained
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17 306 level of education.

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22 307 In Sweden, 12 years of schooling is not mandatory, but the long-term social effects are
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24 308 considerable for those who fail. It has been estimated that 50% of those who fail to complete
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26 309 ≥ 12 school years face a situation of being left outside the society with poor psychosocial health
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28 310 and large costs for the society.²⁸ Thus, it is possible that school failure will further worsen the
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30 311 health perspective of adolescents with obesity.

312 **Potential mechanisms between obesity, weight loss, and school** 313 **performance**

314 Several parameters may interplay when measuring school completion, e.g. resourcefulness,
315 intelligence, or ability to conform. Nevertheless, the mechanisms by which obesity influences
316 school performance are unclear but are likely multifactorial and complex. Possible mechanisms
317 may include psychosocial aspects such as stigma²⁹ and increased risk for anxiety and
318 depression.⁷ Physiological mechanisms may be mediated by anatomical changes such as
319 atrophy of the frontal lobe and reduced hippocampal volume have been demonstrated in
320 individuals with obesity.³⁰ Some of the reported cognitive dysfunctions in children and
321 adolescents with obesity include slower response times when performing visuospatial attention
322 tasks,³¹ diminished executive functions, such as working memory,³² and slower cognitive

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3 323 performance speed.³³ In addition, obesity-related comorbidities such as insulin resistance,³⁴
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5 324 type 2 diabetes mellitus,³⁵ chronic low-grade inflammation,³⁶ and the metabolic syndrome³⁷
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7 325 have also been associated with impaired executive function, memory performance, attention,
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9 326 and cognitive flexibility. A high fat diet in rodents demonstrated detrimental effects on memory
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11 327 and executive functions.³⁰
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15 328 At least some of these effects seem to be reversible, which may add to the positive associations
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17 329 we observed of weight loss on school outcome. Weight loss may have a direct positive effect
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19 330 on cognitive functions.³⁸ Extensive weight loss via bariatric surgery improves insulin sensitivity
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21 331 and decreases systematic inflammation,³⁹ and it has been suggested that these factors affect
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23 332 cognitive functions.⁴⁰
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27 333 However, children with obesity are often stigmatized,⁴¹ have a low self-esteem, and are exposed
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29 334 to bullying and social exclusion.⁴² All these factors have most likely a negative impact on school
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31 335 performance. In contrast, a strong social network is most probably an important factor both for
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33 336 good treatment response and achievement in school. An inverse association has been observed
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35 337 between familial social support and child weight status.⁴³ The relationship between BMI and
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37 338 school completion may also be biased from e.g. assortative mating and dynastic effects which
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39 339 have shown to reduce causal effects.⁴⁴ There is also a suggested interplay between genetic
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41 340 variants and environmental factors that may affect intelligence.⁴⁵ Thus, it is likely that both
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43 341 negative social effects of obesity and obesity-related morbidity, as well as genetic factors,
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45 342 contribute to the adverse association of childhood obesity on completed educational level.^{7 29 41}
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53 344 **Limitations**

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57 345 Using longitudinal data from several national registers provided an opportunity to assess the
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59 346 impact of obesity on completed educational level, adjusted for several important confounders.
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3 347 Data on both exposure and outcome were prospectively collected and defined according to
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5 348 standardized international classifications.^{19 26}
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8 349 However, some important limitations should be recognized. We did not have anthropometric
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10 350 data on children in the comparison group. There is no representative national data on children
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12 351 with obesity in Sweden. As our comparison group includes children with obesity, although
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14 352 likely less than 1% according to obesity diagnoses found in the National Patient Register, odds
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16 353 of lower level of education associated with obesity might be underestimated. It is also important
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18 354 to consider that children receiving obesity treatment may not be representative of all children
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20 355 with obesity. It should further be noted that parental SES was based on data from one specific
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22 356 year in the child's life and not over the child's entire adolescent lifetime, and that the SES
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24 357 indicators used may not reflect the whole SES spectrum. After providing specific ORs for each
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26 358 SES indicator (Table 2) we can hypothesize that additive, or perhaps synergistic effects, of
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28 359 different SES indicators may affect the outcome. Nevertheless, the OR for the childhood obesity
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30 360 cohort vs. comparison group remained within a narrow interval. This may indicate that there is
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32 361 an overlap between the measured SES domains and/or that the effect of obesity is a robust
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34 362 variable, independently of SES. In addition, the impact of anxiety and depression on educational
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36 363 level may be underestimated since these conditions often are under-diagnosed. It is furthermore
37
38 364 plausible that controlling for anxiety and depression may lead to an underestimation of the
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40 365 association found between obesity in childhood and completion of 12 or more school years,
41
42 366 since the variable may act as a modifier/mediator.⁷ We urge the reader to bear in mind that
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44 367 despite several possible mechanisms have been proposed, causal relationships of obesity and
45
46 368 the effect of treatment on the outcome remains to be established. Lastly, factors such as free
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48 369 education, school lunches, and students' health care may have an impact on the generalizability
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50 370 of our data to other populations.
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371 **Conclusion**

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3 372 In this longitudinal, population-based study, individuals with obesity in childhood were less
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5 373 likely to complete 12 or more years of schooling, compared with a group from the general
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7 374 population. The odds associated with obesity remained significantly increased even after taking
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9 375 parental SES and other important risk factors, such as ADHD, into consideration. The
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11 376 underlying mechanisms are unclear but previous studies indicate that the effects of obesity on
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13 377 school completion both involve psychosocial effects and cognitive functions.^{16 17 32} The
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15 378 negative impact of childhood obesity on educational level may partly be reversed by successful
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17 379 obesity treatment in childhood. Results from this study underline effects of childhood obesity
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19 380 on schooling. For the sake of an optimal educational environment, it is of great importance to
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21 381 increase awareness both in schools and among decision makers to allocate resources for
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23 382 potential extra support, e.g. reduce stigma and increase educational support.
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32
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34
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39 387 **Author contributions**

40
41 388 Conceptualization: Louise Lindberg, Pernilla Danielsson, Martina Persson, Claude Marcus,
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43 389 Emilia Hagman.

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45 390 Data curation: Louise Lindberg.

46
47 391 Formal analysis: Louise Lindberg, Emilia Hagman.

48
49 392 Funding acquisition: Louise Lindberg, Claude Marcus, Emilia Hagman.

50
51 393 Methodology: Louise Lindberg, Emilia Hagman.

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53 394 Project administration: Louise Lindberg, Emilia Hagman.

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55 395 Supervision: Martina Persson, Emilia Hagman, Pernilla Danielsson, Claude Marcus.

56
57 396 Writing – original draft: Louise Lindberg.
58
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3 397 Writing – review & editing: Louise Lindberg, Pernilla Danielsson, Martina Persson, Claude
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29 408 **Data Sharing statement**

30 409 The data that support the findings of this study contains sensitive information. Restrictions
31
32 410 therefore apply to the availability of these data, which were used under license for the current
33
34 411 study, and so are not publicly available. According to Swedish law and the General Data
35
36 412 Protection Regulation, the authors are not permitted to share the datasets used in this study with
37
38 413 third parties. Given that an ethical approval is obtained, any individual may apply for data from
39
40 414 Statistics Sweden via information@scb.se, the Swedish National Board of Health and Welfare
41
42 415 via registerservice@socialstyrelsen.se, and the Swedish Childhood Obesity Treatment Register
43
44 416 via <http://www.e-boris.se/kontaktuppgifter/>.
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50 417 **Competing interests**

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422 appear to have influenced the submitted.

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557 **Supporting information**

558 **S1 Table. International Classification of Diseases (ICD 10th revision) codes and**
559 **Anatomical Therapeutic Chemical (ATC) classification system codes used.**

560 **S2 Table. Proportion of children completing ≥ 12 years of schooling.**

561 **S3 Table. Adjusted odds ratio (99% CI); p-value of subjects completing ≥ 12 years of**
562 **schooling stratified by group.**

563 **S4 Table. Proportion of individuals who have undergone obesity treatment in childhood**
564 **who completed ≥ 12 years of schooling by calendar year and by age at start of obesity**
565 **treatment (n=3,942).**

567 **Figure Legends**

568 **Fig 1. Participant flowchart.**

570 **Fig 2. Percentage of individuals completing ≥ 12 years of schooling by SES level in the**
571 **childhood obesity cohort (n=3,921,) and the comparison group (n=18,528).**

573 **Fig 3. Crude percentage of individuals with obesity in childhood (n=3,921) completing ≥ 12**
574 **school years by parental SES and treatment response. p=p for trend; n=refers to number**
575 **of individuals in each category.**

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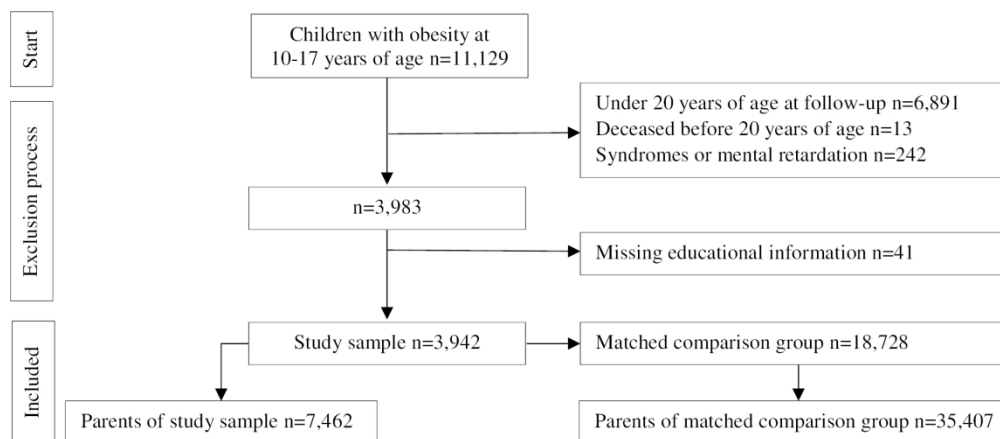


Figure 1

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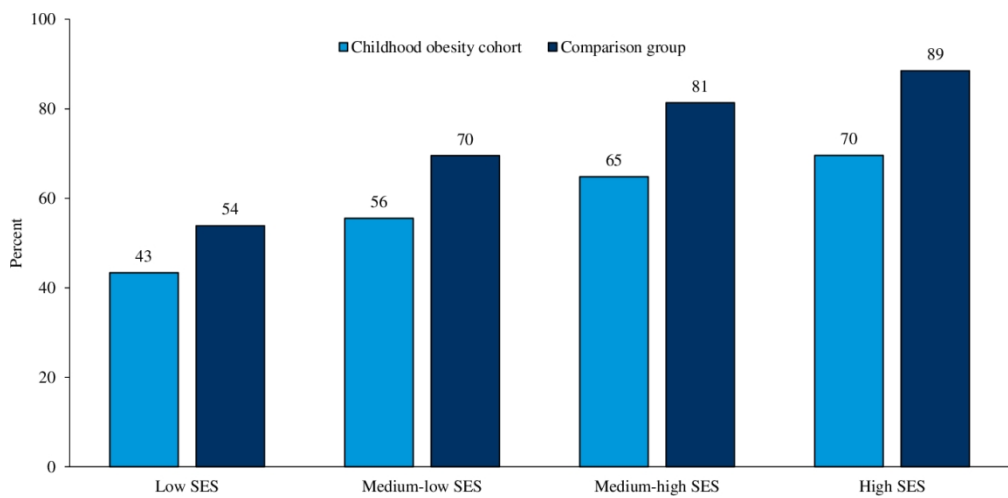


Fig 2. Percentage of individuals completing ≥ 12 years of schooling by SES level in the childhood obesity cohort (n=3,921) and the comparison group (n=18,528).

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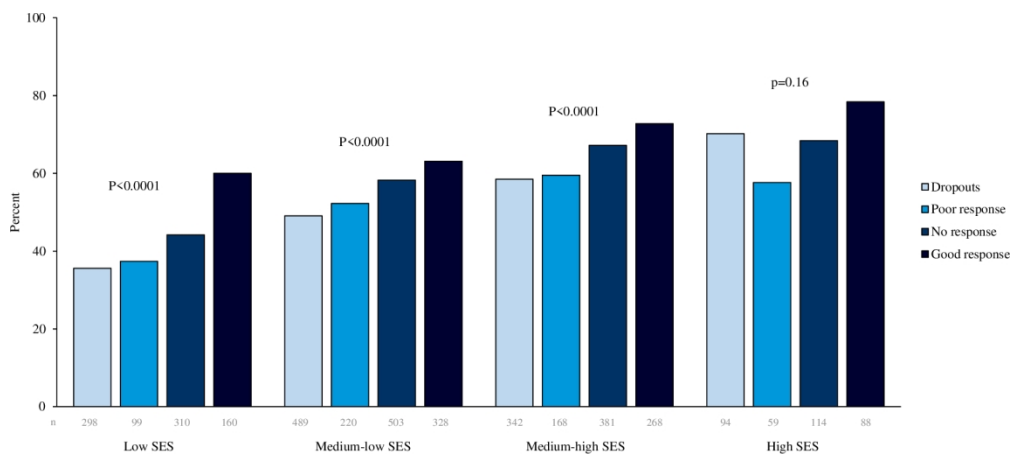


Fig 3. Crude percentage of individuals with obesity in childhood (n=3,921) completing ≥12 school years by parental SES and treatment response. p=p for trend; n=refers to number of individuals in each category.

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S1 Table. International Classification of Diseases (ICD 10th revision) codes and Anatomical Therapeutic Chemical (ATC) classification system codes used.

Condition	ICD-10	ATC Code
Anxiety/sedatives	F40.0-F40.1, F41-F42, F51.4-F51.9, F93.0-F93.2	N05B
Depression/antidepressants	F32-F39, F92	N06A
ADHD/ADD	F90	N06B
Mental retardation	F70-F79	
Genetic syndromes		
Fragile X	Q99.2	
Klinefelter	Q98	
Laurence-Moon-Bardet-Biedl	Q87.8B	
Mb Down	Q90	
Noonan	Q87.1E	
Prader-Willi	Q87.1F	
Silver Russell	Q87.1G	
Turner	Q96	

S2 Table. Proportion of children completing ≥ 12 years of schooling

	Childhood obesity cohort		Comparison group		p-value [¶]
	n	%	n	%	
Total	2,236	56.7	13,939	74.4	<0.0001
Sex					
Girl	1,090	59.7	6,843	78.7	<0.0001
Boy	1,146	54.1	7,096	70.8	<0.0001
Migration background					
Nordic	1,695	58.4	10,974	78.1	<0.0001
Non-Nordic	541	52.2	2,965	63.4	<0.0001
ADHD/ADD					
Non-ADHD/ADD	2,032	61.1	13,528	76.5	<0.0001
ADHD/ADD	204	33.1	411	39.4	0.01
Anxiety/depression					
No anxiety/depression	1,902	61.1	12,754	77.0	<0.0001
Anxiety/depression	334	40.2	1,185	54.9	<0.0001
Parental SES					
Low	376	43.4	1,455	53.9	<0.0001
Medium-low	855	55.5	4,187	69.5	<0.0001
Medium-high	751	64.8	5,358	81.3	<0.0001
High	247	69.6	2,848	88.5	<0.0001
Maternal education					
Compulsory school	755	47.7	3,474	61.9	<0.0001
Upper secondary school	945	61.3	5,582	77.9	<0.0001
University degree	506	69.3	4,589	85.3	<0.0001
Paternal education					
Compulsory school	639	50.2	2,775	63.0	<0.0001
Upper secondary school	1,132	59.9	7,085	77.9	<0.0001
University degree	318	66.0	3,333	85.2	<0.0001
Maternal income					
Q1	614	52.3	3,327	68.1	<0.0001
Q2	750	58.6	4,391	75.2	<0.0001
Q3	542	58.2	3,534	77.1	<0.0001
Q4	304	62.6	2,429	81.3	<0.0001
Paternal income					
Q1	556	49.3	2,453	62.6	<0.0001
Q2	410	54.0	2,260	72.3	<0.0001
Q3	549	61.8	3,404	77.6	<0.0001
Q4	572	66.2	5,056	84.5	<0.0001
Maternal occupational status					
No occupation	438	47.8	1,722	58.3	<0.0001
Occupation	1,777	60.0	12,005	77.9	<0.0001
Paternal occupational status					
No occupation	363	45.4	1,543	58.7	<0.0001
Occupation	1,736	60.5	11,703	78.5	<0.0001

Abbreviations: SES, socioeconomic status; Q, quartile. [¶]Performed with chi-square.

1 EUR \approx 9.83 SEK December 31st 2017. Q1: <18,652 EUR; Q2: 18,652 to 25,025 EUR; Q3: 25,026 to 32,309 EUR; Q4: >32,309 EUR.

S3 Table. Adjusted odds ratio (99% CI); p-value of subjects completing ≥12 years of schooling stratified by group

	Childhood obesity cohort	Comparison group
Parental SES (ref=low) ^a		
Medium-low	1.64 (1.30 to 2.07); <0.0001	1.81 (1.59 to 2.07); <0.0001
Medium-high	2.43 (1.88 to 3.12); <0.0001	3.25 (2.82 to 3.75); <0.0001
High	3.22 (2.24 to 4.64); <0.0001	5.80 (4.81 to 6.99); <0.0001
Maternal education (ref=compulsory school) ^b		
Upper secondary school	1.64 (1.33 to 2.01); <0.0001	1.75 (1.56 to 1.96); <0.0001
University degree	2.33 (1.76 to 3.09); <0.0001	2.54 (2.20 to 2.93); <0.0001
Paternal education (ref=compulsory school) ^b		
Upper secondary school	1.29 (1.05 to 1.58); 0.0012	1.59 (1.42 to 1.79); <0.0001
University degree	1.46 (1.06 to 2.01); 0.0025	2.28 (1.94 to 2.67); <0.0001
Maternal income (ref=Q1) ^c		
Q2	1.28 (1.01 to 1.61); 0.0064	1.26 (1.11 to 1.42); <0.0001
Q3	1.26 (0.98 to 1.62); 0.019	1.30 (1.13 to 1.49); <0.0001
Q4	1.42 (1.04 to 1.95); 0.004	1.47 (1.25 to 1.73); <0.0001
Paternal income (ref=Q1) ^c		
Q2	1.19 (0.92 to 1.54); 0.075	1.44 (1.25 to 1.66); <0.0001
Q3	1.57 (1.22 to 2.02); <0.0001	1.79 (1.56 to 2.05); <0.0001
Q4	1.96 (1.51 to 2.55); <0.0001	2.63 (2.29 to 3.03); <0.0001
Maternal occupational status (ref=no occupation) ^d		
Occupation	1.42 (1.14 to 1.77); <0.0001	1.83 (1.61 to 2.08); <0.0001
Paternal occupational status (ref=no occupation) ^d		
Occupation	1.63 (1.30 to 2.04); <0.0001	1.90 (1.67 to 2.16); <0.0001

^aAdjusted for migration background, ADHD/ADD, anxiety/depression, parental SES

^bAdjusted for migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal education

^cAdjusted for migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal income

^dAdjusted for migration background, ADHD/ADD, anxiety/depression, maternal-, and paternal occupational status

Abbreviations: SES, socioeconomic status; Q, quartile.

Q1: <18,652 EUR; Q2: 18,652 to 25,025 EUR; Q3: 25,026 to 32,309 EUR; Q4: >32,309 EUR. 1 EUR ≈ 9.83 SEK December 31st 2017.

S4 Table. Proportion of individuals who have undergone obesity treatment in childhood who completed ≥ 12 years of schooling by calendar year and by age at start of obesity treatment (n=3,942).

Individuals completing ≥ 12 years of schooling		
	n	%
Calendar year at start of obesity treatment		
1995-2001	229	62.6
2002-2008	1,262	58.1
2009-2014	745	53.1
Age at start of obesity treatment		
10 - 12.99	960	59.0
13 - 16.99	1,276	55.1

STROBE Statement—Checklist of items that should be included in reports of *cohort studies*

	Item No	Recommendation	Page No
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract (b) Provide in the abstract an informative and balanced summary of what was done and what was found	1-2 2-3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4
Objectives	3	State specific objectives, including any prespecified hypotheses	4.5
Methods			
Study design	4	Present key elements of study design early in the paper	5
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5-9
Participants	6	(a) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up (b) For matched studies, give matching criteria and number of exposed and unexposed	5 5
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	7-9
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	7-9
Bias	9	Describe any efforts to address potential sources of bias	7-9
Study size	10	Explain how the study size was arrived at	5
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	9-10
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding (b) Describe any methods used to examine subgroups and interactions (c) Explain how missing data were addressed (d) If applicable, explain how loss to follow-up was addressed (e) Describe any sensitivity analyses	9-10
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed (b) Give reasons for non-participation at each stage (c) Consider use of a flow diagram	12 + fig
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders (b) Indicate number of participants with missing data for each variable of interest (c) Summarise follow-up time (eg, average and total amount)	11
Outcome data	15*	Report numbers of outcome events or summary measures over time	12

1	Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	12-17
2			(b) Report category boundaries when continuous variables were categorized	
3			(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	
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9	Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	12-17
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11	Discussion			
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13	Key results	18	Summarise key results with reference to study objectives	18
14	Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	20-21
15				
16	Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	18-21
17				
18				
19	Generalisability	21	Discuss the generalisability (external validity) of the study results	21
20				
21	Other information			
22	Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	22-23
23				
24				

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at <http://www.plosmedicine.org/>, Annals of Internal Medicine at <http://www.annals.org/>, and Epidemiology at <http://www.epidem.com/>). Information on the STROBE Initiative is available at <http://www.strobe-statement.org>.