ORIGINAL CONTRIBUTION

Parental socioeconomic position and risk of ADHD in offspring: a cohort study of 9648 individuals in Denmark 1976–2013

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Abstract

The strength of the association between parental socioeconomic position (SEP) and risk of attention-deficit/hyperactivity disorder (ADHD) in offspring is found to vary substantially, perhaps due to the negligence of possible changes in parental life course SEP. The present study investigated the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring and whether parental childhood SEP modified this association. The study population included 9648 live-born singletons followed in the Psychiatric Central Register from birth in 1976–1996 until 2013. Cox regression was used to estimate hazard ratios for ADHD diagnosis according to parental SEP in adulthood. The results showed that low parental SEP in adulthood was associated with higher risk of ADHD diagnosis in offspring, also after adjustment for possible confounders. Thus, offspring of parents with low SEP in adulthood had 4.52 (95% CI 2.81–7.26) times higher hazard of ADHD diagnosis compared with offspring of parents with high SEP in adulthood. Further, parental childhood SEP was found to modify the observed association. Thus, offspring of parents with downward social mobility from childhood to adulthood and offspring of parents with stable low SEP experienced the highest risk of ADHD diagnosis, followed by offspring of parents with upward social mobility, compared with offspring of parents with stable high SEP. The results suggest that it is important to take into account the possibility of social mobility as changes in parental life course SEP from childhood to adulthood seem to influence the risk of ADHD diagnosis in offspring.

Keywords ADHD · Socioeconomic factors · Cohort studies · Denmark

Introduction

In recent years, attention-deficit/hyperactivity disorder (ADHD) has received much attention due to its increasing occurrence in the population [1, 2]. The global prevalence of ADHD is now about 2–7% [2]. Whether this prevalence estimate reflects a real increase in the proportion of individuals with ADHD or might be attributed to factors such as earlier diagnosis, changes in diagnostic criteria and diagnostic practices, as well as an increased attention among health professionals and nonprofessionals is a vexed question [1, 3]. Whatever the case, when so many individuals today fulfill the diagnostic criteria for ADHD, there is no doubt that society faces a significant public health problem.

ADHD is often referred to as a disorder belonging to children of the lower social classes [4]. A systematic review and meta-analysis has indeed also found that children of parents with low socioeconomic position (SEP) are more likely to have an ADHD diagnosis than children of parents with high SEP [5]. However, as the systematic review and meta-analysis shows substantial between-study heterogeneity and the strength of the association is found to vary substantially between studies, the authors conclude that there is still a need of further research primarily investigating this association in depth. Therefore, the primary aim of our study was to investigate the association between parental SEP and risk of ADHD diagnosis in offspring.

Since most of the existing studies in the field do not take into account the influence of possible changes in parental life

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course SEP, and since the few existing studies considering social mobility only focus on changes in parental SEP during the upbringing of the child, we speculate if some of the substantial variation in the strength of the association might be explained by possible changes in parental SEP from childhood to adulthood. The few existing studies focusing on changes in parental SEP during the upbringing of the child have found that both upward and downward social mobility influence the risk of ADHD in offspring [6–8]. Thus, it is plausible that social mobility might be associated with individual characteristics such as intelligence and personality [9], which directly or indirectly may be associated with the risk of ADHD diagnosis in offspring [10, 11]. Other studies have found that ADHD is associated with maternal lifestyle factors in the prenatal period such as smoking, alcohol, and drug use [12–14], and since such socially patterned lifestyle habits develop early in life and, once established, are difficult to change [15], it is also plausible that parental childhood SEP might modify the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring. Therefore, a secondary aim of our study was to investigate whether parental childhood SEP modifies the influence of parental SEP in adulthood on risk of ADHD diagnosis in offspring.

In summary, the aims of this study were to investigate the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring and to investigate whether parental childhood SEP modifies the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring.

Methods

Study population

A prospective cohort study was conducted of 9648 live-born singletons whose parents and grandmothers were members of the Copenhagen Perinatal Cohort (CPC).

The CPC consisted of all pregnant women who were admitted to the maternity departments of the Copenhagen University Hospital during the period from 21st October 1959 to 21st December 1961, except for vacation periods, corresponding to 8949 women (generation 1) and their 9125 children (generation 2) [16]. About 7600 women in generation 1 and 8100 men and women in generation 2 have afterwards been identified in the Civil Registration System. Among these, 5032 women in generation 1 (the grandmothers of the population at risk) and 5222 men and women in generation 2 (the parents of the population at risk) were registered with 9648 live-born singleton descendants (generation 3—the population at risk). The study population is illustrated in Fig. 1.

The population at risk was followed from birth in 1976 to 1996 until the first registration of an ADHD diagnosis, attrition or June 30, 2013 (end of follow-up), whichever came first. The observation periods ranged from 0 to 37 years. During the study period, 205 individuals were diagnosed with ADHD and further 350 individuals were censored (114 individuals due to death, 235 individuals due to emigration and 1 individual due to disappearance).

Data were obtained from the Psychiatric Central Register and linked to national registers administered by Statistics Denmark and to information from the CPC. Linkage of individual-level data was possible using the personal registration numbers (CPR-numbers), which are assigned to all citizens in Denmark.

According to Danish legislation, no ethics approval is needed for register-based studies. The present study is covered by permissions from the Danish Data Protection Agency to the authors.

Attention-deficit/hyperactivity disorder

The outcome of interest was the assignment of an ADHD diagnosis to the population at risk. Information regarding ADHD diagnoses was retrieved from the Psychiatric Central Register at the end of follow-up. The Psychiatric Central Register includes information on all psychiatric admissions to Danish hospitals since 1969 and information on all psychiatric outpatient treatments since 1995 [17]. From 1969 to 1994, the International Classification of Diseases—8th revision (ICD-8) was used as diagnostic reference, when

Fig. 1 Overview of the study population
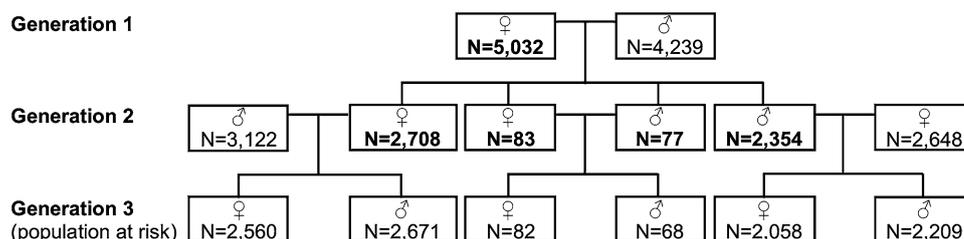
The 5032 women (generation 1) and the 5222 men and women (generation 2) marked in bold type are the members of the Copenhagen Perinatal Cohort

it was replaced by the International Classification of Diseases—10th revision (ICD-10) [18]. Individuals in generation 3 receiving an ICD-8 diagnosis of 308.3 or an ICD-10 diagnosis of F90.0, F90.1, F90.8 or F90.9 following either inpatient or outpatient treatment were categorized as ADHD cases. The validity of these diagnoses in the Psychiatric Central Register has been found to be relatively high (86.8% for hyperkinetic disorder) [19].

Parental socioeconomic position in adulthood

The primary exposure of interest was parental SEP in adulthood measured by the educational level of generation 2. Parental educational level was based on the parents' highest completed education at the time of birth of the population at risk. This indicator was chosen, as we consider it the best SEP indicator at the time in adulthood when most individuals start a family [20]. In addition, parental educational level is a much more stable indicator than for instance occupation and income, which often vary over the life course. Based on information from Statistics Denmark's education register available since 1981, three variables were created for the highest completed maternal education, paternal education and parental education (highest educational level by any of the parents), respectively. Educational level was categorized into three categories: low (primary and lower secondary school), medium (upper secondary school and vocational training) and high (post-school education).

Parental socioeconomic position in childhood

The secondary exposure of interest was parental SEP in childhood. Parental SEP in childhood was based on a composite indicator developed by the Centre International de l'Enfance [21], which was made from information from the CPC's 1-year follow-up examination. This information included the breadwinner's occupation; the breadwinner's type of income; the breadwinner's education; and the character of the living accommodation, its size, the number of persons per room and its location [22]. For each indicator, the individual was assigned 0–5 points, which were added to a total score (range: 0–20). The higher the total score, the higher the SEP. The total score was categorized into three categories, which more or less corresponded to the Registrar General's Social Classes [22]. Because parental childhood SEP was based on information from the CPC's 1-year examination, this indicator was only available for the 2791 mothers and the 2431 fathers in generation 2 who were members of the CPC.

Covariates

Covariates included gender, calendar year, region (Capital Region of Denmark, Region Zealand, North Denmark Region, Central Denmark Region, Region of Southern Denmark), maternal age, paternal age and parental psychiatric history (registered diagnosis in the Psychiatric Central Register, no registered diagnosis). All covariates were measured at birth of the population at risk, except for the time-dependent calendar year.

Calendar year was included due to the secular trends in the occurrence of ADHD diagnoses and region due to geographical differences in the occurrence of ADHD diagnoses [1, 23].

Statistical methods

Descriptive statistics were used to report the characteristics of the study population according to whether the individuals were diagnosed with ADHD. Tests for marginal associations between ADHD diagnosis and each of the characteristics were conducted—binary and ordinal variables were tested by means of γ tests, nominal variables were tested by means of χ^2 tests and continuous variables were tested by means of t tests.

The missing data frequency varied from less than 1% for parental total highest completed education, ADHD diagnosis, gender, calendar year, region, maternal age, paternal age and parental psychiatric history to 2.1% for maternal education, 4.2% for paternal education and 19.4% for parental childhood SEP, for this reason all statistical analyses were conducted using multiple imputation. First, the missing values were replaced with estimated values by drawing from a multivariate normal distribution of the missing data given the observed data in a particular statistical model (categorical variables were replaced by dummy variables). This procedure was repeated until 40 complete datasets were created. Second, the statistical models described below were run within each of the complete datasets. Third, the obtained parameter estimates from all the complete datasets were combined for inference. Consequently, all statistical analyses were conducted using multiple imputation in which missing values were generated from the available values on the included variables in a particular statistical model and analyzed using 40 imputed datasets.

The main analyses investigated the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring by means of Cox regression with age as the underlying timescale. First, the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring was analyzed using three different models. In model 1, the unadjusted associations were estimated. In model 2, the associations were estimated after adjustment for gender, calendar year (quadratic term) and region. In model 3, the associations were estimated with further adjustment for maternal age (quadratic term), paternal age and parental psychiatric history. Results of the statistical analyses were presented

by hazard ratios and 95% confidence intervals. Second, to test whether parental childhood SEP modified the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring, we added an interaction term between parental childhood SEP and parental SEP in adulthood to all statistical models. Separate tests showed no evidence of effect measure modification by either offspring gender or year of birth (results not shown). All statistical models were corrected for intra-cluster dependency. Relevant model assumptions were assessed, but no violations were observed.

In sensitivity analyses, the robustness of the results was investigated. All statistical analyses were thus conducted using non-imputed data and data were analyzed by use of logistic regression. The logistic regression analyses were conducted to investigate whether the association between parental SEP and risk of ADHD diagnosis in offspring changed when the outcome was not time to ADHD diagnosis, but odds for ADHD diagnosis, as time to diagnosis might not be an appropriate outcome measure because the register-based ADHD diagnosis is often delayed, particularly among individuals with low SEP [24].

The statistical analyses were conducted using SAS version 9.4.

Results

The study population included 9648 individuals—4948 males (51.3%) and 4700 females (48.7%). During the study period, 205 individuals were registered with an ADHD diagnosis. All ADHD diagnoses were registered after the implementation of ICD-10 in 1994. The median age at diagnosis was 17 years (range 3–34 years). There was a statistically significant difference in age at diagnosis according to parental SEP in adulthood—the higher the parental SEP, the younger the age at diagnosis (results not shown).

Characteristics of the study population are shown in Table 1. Among individuals registered with an ADHD diagnosis, there was a higher proportion of males, parents with low SEP in adulthood, parents with psychiatric diagnoses, and grandparents with low SEP than among individuals without ADHD diagnoses.

The main analyses showed that low maternal education, paternal education and parental education, respectively, were associated with higher risk of ADHD diagnosis in offspring (Table 2). Adjustment for gender, calendar year and region strengthened the associations considerably, whereas further adjustment for maternal age, paternal age and parental psychiatric history weakened the observed associations slightly. Thus, individuals whose parents had a low educational level had 4.52 (95% CI 2.81–7.26) times higher hazard of ADHD diagnosis, while individuals whose parents had a medium educational level had 2.74 (95% CI 1.77–4.25) times higher hazard of ADHD diagnosis, compared with individuals whose parents had a high educational level.

The interaction analyses showed that parental childhood SEP modified the influence of parental SEP in adulthood on risk of ADHD diagnosis in offspring (Table 3). Among the parents participating in the CPC, 89.5% of the parents with a high SEP in childhood also had a high SEP in adulthood, while 10.6% experienced downward social mobility. Further, 32.9% of the parents with a low SEP in childhood also had a low SEP in adulthood, while 67.1% experienced upward social mobility. This corresponds to γ: 0.61 ($p < 0.001$). As shown in Table 3, individuals whose parents had a stable low SEP from childhood to adulthood had 2.62 (95% CI 1.65–4.14) times higher hazard of ADHD diagnosis compared with individuals whose parents had a stable high SEP. Further, individuals whose parents experienced downward social mobility were found to have 3.14 (95% CI 1.75–5.63) times higher hazard of ADHD diagnosis, while individuals whose parents experienced upward social mobility had 1.66 (95% CI 1.09–2.54) times higher hazard of ADHD diagnosis, compared with individuals whose parents had a stable high SEP.

Sensitivity analyses revealed no noteworthy changes in the associations between parental SEP and risk of ADHD diagnosis in offspring by use of all available cases or by use of logistic regression.

Discussion

Main findings

The results showed that low parental SEP in adulthood was associated with higher risk of ADHD diagnosis in offspring after adjustment for gender, calendar year, region, maternal age, paternal age and parental psychiatric history. Further, parental SEP in childhood seemed to modify the observed association. Thus, individuals whose parents experienced downward social mobility from childhood to adulthood and individuals whose parents had a stable low SEP had the highest risk of ADHD diagnosis, followed by individuals whose parents experienced upward social mobility, compared with individuals whose parents had a stable high SEP.

Comparison with the existing literature

The finding that low parental SEP in adulthood was associated with higher risk of ADHD diagnosis in offspring is consistent with the majority of previous studies, including a recent systematic review and meta-analysis [5].

With regard to the association between maternal educational level and risk of ADHD diagnosis in offspring, we

Table 1 Characteristics of the study population (N = 9648)

	ADHD	
	Diagnosed (N=205)	Not diagnosed (N=9443)
Children		
Gender, N (%)		
Boys	**136 (66.3)**	**4812 (51.0)**
Girls	**69 (33.7)**	**4631 (49.0)**
Year of birth		
Mean (SD)	1988 (5.1)	1988 (4.6)
Region, N (%)		
Capital region of Denmark	143 (69.8)	6553 (69.4)
Region Zealand	29 (14.2)	1469 (15.6)
Jutland and Funen	33 (16.0)	1416 (15.0)
Missing	0 (−)	5 (−)
Parents		
Maternal parity, N (%)		
Primipara	111 (54.2)	5308 (56.2)
Secundipara	69 (33.7)	3190 (33.8)
≥Tertipara	25 (12.2)	945 (10.0)
Maternal age		
Mean (SD)	26 (5.3)	26 (4.6)
Paternal age[a]		
Mean (SD)	28 (6.0)	29 (5.3)
Missing, N	2	73
Maternal education, N (%)		
Low	**120 (61.5)**	**3613 (39.1)**
Medium	**59 (30.3)**	**3480 (37.6)**
High	**16 (8.2)**	**2160 (23.3)**
Missing	10 (−)	190 (−)
Paternal education, N (%)[a]		
Low	**81 (42.6)**	**2965 (32.7)**
Medium	**90 (47.4)**	**4140 (45.7)**
High	**19 (10.0)**	**1952 (21.6)**
Missing	15 (−)	371 (−)
Parental education, N (%)		
Low	**78 (38.1)**	**2033 (21.6)**
Medium	**100 (48.8)**	**4473 (47.5)**
High	**27 (13.2)**	**2919 (31.0)**
Missing	0 (−)	18 (−)
Parental psychiatric history, N (%)		
Psychiatric diagnosis	**17 (8.3)**	**376 (4.0)**
No psychiatric diagnoses	**188 (91.7)**	**9067 (96.0)**
Member of the CPC, N (%)		
Both parents	6 (2.9)	144 (1.5)
Mother	107 (52.2)	5124 (54.3)
Father	92 (44.9)	4175 (44.2)
Grandparents		
Grandparental socioeconomic position, N (%)[b]		
Low	**99 (61.5)**	**3715 (48.8)**
Medium	**54 (33.5)**	**3022 (39.7)**

Table 1 (continued)

	ADHD	
	Diagnosed (*N*=205)	Not diagnosed (*N*=9443)
High	**8 (5.0)**	**874 (11.5)**
Missing	44 (−)	1832 (−)

Variables with distributions marked in bold have been found to differ statistically significantly between individuals with and without ADHD diagnoses. Binary and ordinal variables were tested by use of γ tests, nominal variables were tested by use of χ^2 tests, and continuous variables were tested by use of *t* tests

[a]Characteristics are only shown for individuals with identified fathers—corresponding to 9633 individuals

[b]Grandparental socioeconomic position is the same as parental socioeconomic position in childhood

Table 2 Associations between parental socioeconomic position in adulthood and time to ADHD diagnosis in offspring

	Model 1		Model 2		Model 3	
	HR (95% CI)	*p* value[a]	HR (95% CI)	*p* value[a]	HR (95% CI)	*p* value[a]
Maternal education		0.17		<0.001		<0.001
Low	1.69 (0.97, 2.93)		5.59 (3.20, 9.75)		5.04 (2.90, 8.76)	
Medium	1.61 (0.91, 2.87)		2.68 (1.51, 4.76)		2.59 (1.47, 4.55)	
High	1 (ref.)		1 (ref.)		1 (ref.)	
Paternal education		0.56		<0.001		<0.001
Low	1.32 (0.78, 2.24)		3.46 (2.01, 5.95)		3.07 (1.77, 5.35)	
Medium	1.32 (0.78, 2.23)		2.64 (1.55, 4.51)		2.51 (1.46, 4.30)	
High	1 (ref.)		1 (ref.)		1 (ref.)	
Parental education		0.24		<0.001		<0.001
Low	1.48 (0.94, 2.34)		5.05 (3.18, 8.02)		4.52 (2.81, 7.26)	
Medium	1.37 (0.88, 2.12)		2.85 (1.84, 4.40)		2.74 (1.77, 4.25)	
High	1 (ref.)		1 (ref.)		1 (ref.)	

Model 1: unadjusted

Model 2: adjusted for gender, calendar year and region

Model 3: adjusted for gender, calendar year, region, maternal age, paternal age and parental psychiatric history

HR hazard ratio, *CI* confidence interval

[a]*p* value for Wald test

found that offspring of mothers with low and medium educational levels had 5.04 (95% CI 2.90–8.76) and 2.59 (95% CI 1.47–4.55) higher hazard of ADHD diagnosis, respectively, whereas the meta-analysis found that offspring of mothers with a low educational level had 1.91 (95% CI 1.21–3.03) higher odds of ADHD diagnosis, compared with offspring of mothers with a high educational level. Our much higher risk estimate for the low category might to some extent be explained by differences in the categorization of maternal educational level. The meta-analysis defines a low maternal educational level as no educational qualifications or high school qualifications only, which more or less corresponds to a combination of our low and medium categories. However, as our risk estimates for both the low and medium categories are higher than the pooled risk estimate from the meta-analysis, this cannot be the only explanation. Other explanations might be differences in study design, as the meta-analysis

includes cohort, case–control and cross-sectional studies, or differences in the choice of confounders. However, the meta-analysis does not report which confounders its six studies include. Finally, it should be mentioned that the findings of the studies included vary substantially, for this reason the meta-analysis reports a 95% prediction interval of 0.37–9.75. Therefore, our findings are actually within the prediction interval of the meta-analysis.

With regard to the association between paternal educational level and risk of ADHD diagnosis in offspring, we found that offspring of fathers with low and medium educational levels had 3.07 (95% CI 1.77–5.35) and 2.51 (95% CI 1.46–4.30) higher hazard of ADHD diagnosis, respectively, whereas the meta-analysis found that offspring of fathers with a low educational level had 2.10 (95% CI 1.27–3.47) higher odds of ADHD diagnosis, compared with offspring of fathers with a high educational level. The variance in

Table 3 Association between parental socioeconomic position in adulthood and time to ADHD diagnosis in offspring modified by parental childhood socioeconomic position

	Parental childhood socioeconomic position		p value[a]
	Low	High	
	HR (95% CI)	HR (95% CI)	
Model 1: parental education			0.33
Low	1.15 (0.75, 1.76)	1.71 (0.97, 3.03)	
High	1.21 (0.80, 1.84)	1 (ref.)	
Model 2: parental education			<0.001
Low	2.93 (1.89, 4.55)	3.55 (2.00, 6.28)	
High	1.72 (1.13, 2.62)	1 (ref.)	
Model 3: parental education			<0.001
Low	2.62 (1.65, 4.14)	3.14 (1.75, 5.63)	
High	1.66 (1.09, 2.54)	1 (ref.)	

Model 1: unadjusted

Model 2: adjusted for gender, calendar year and region

Model 3: adjusted for gender, calendar year, region, maternal age, paternal age and parental psychiatric history

HR hazard ratio, *CI* confidence interval

[a] p value for Wald test

the strength of the association might also in this case be explained by differences in study design, choice of confounders and categorization of paternal educational level. However, most importantly, it should be mentioned that the meta-analysis is based on only three studies so the authors have not been able to calculate a prediction interval.

With regard to the association between parental educational level and risk of ADHD diagnosis in offspring, we found that offspring of parents with low and medium educational levels had 4.52 and 2.74 higher hazard of ADHD diagnosis, respectively. The strength of this association is close to the strength of the association between maternal educational level and risk of ADHD diagnosis in offspring, whereas the association between paternal educational level and risk of ADHD diagnosis in offspring was significantly weaker. It is interesting that we find a stronger effect of maternal educational level than paternal educational level, as the meta-analysis finds the opposite tendency. However, at least two explanations might be at work. First, if we believe that our observed associations are mainly explained by strong genetic influences—i.e., if we believe that genetic factors influencing parental educational level also influence the risk of ADHD in offspring—the stronger effect of maternal educational level might be due to the uncertainty of biological paternity [25]. Second, if we believe that our observed associations are mainly explained by environmental influences, the stronger effect of maternal educational level might be due to the fact that the mother usually spends more time with the child and is more important for the upbringing of the child [26]. On the other hand, the meta-analysis' finding of a stronger effect of paternal educational level might be explained by gender-specific effects—whether genetic or

environmental. Consequently, the inconsistent findings of our study and the meta-analysis might be because different mechanisms of action are at work, or—as the associations in both our study and the meta-analysis are marked by wide confidence intervals—they might just be due to low statistical power.

Overall, the finding that low parental SEP in adulthood—measured by parental educational level—was associated with higher risk of ADHD diagnosis in offspring might be explained by possible differences in cognitive and noncognitive characteristics of parents with uneven educational levels [27, 28]. Thus, knowledge attained through education may influence individuals' cognitive functioning, make them more receptive to health messages and more able to communicate with and access appropriate health services [20]. It would be interesting to compare the strength of the association of other indicators of parental SEP in adulthood—such as occupation and income—with risk of ADHD diagnosis in offspring with our study results, since this might tell us something about the underlying mechanisms at work, but this is outside the scope of this study.

The finding that parental childhood SEP modifies the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring is to our knowledge unique and has not previously been investigated. However, the findings of studies investigating the influence of social mobility during the upbringing of the child on risk of ADHD are consistent with our findings [6–8]. The results suggest that both upward and downward social mobility influence the risk of ADHD diagnosis in offspring. A possible explanation is that social mobility is associated with individual characteristics such as intelligence and personality [9],

which directly or indirectly may be associated with the risk of ADHD diagnosis in offspring [10, 11]. Another possible explanation is that maternal lifestyle habits in the prenatal period associated with both parental SEP in adulthood and risk of ADHD in offspring are influenced by parental childhood SEP [12]. In line with this explanation, a previous study has found that maternal childhood SEP modifies the association between maternal SEP in adulthood and two other health outcomes in offspring related to maternal lifestyle: weight and body composition at birth [29]—two factors that are also associated with risk of ADHD [12]. Future studies investigating in depth the joint influence of parental SEP in childhood and adulthood on risk of ADHD diagnosis in offspring, as well as the underlying mechanisms, are needed.

Strengths and limitations

The major strength of this study is the large study population including 9648 individuals who have been followed prospectively from birth in 1976–1996 until June 30, 2013 (end of follow-up). The prospective design makes it unlikely that the diagnosis of ADHD has influenced parental SEP in adulthood. Further, the long follow-up period reduces the risk that individuals who are not registered with an ADHD diagnosis at the end of follow-up will be diagnosed later on. The long follow-up period has also made it possible to investigate whether secular trends in the diagnosis of ADHD have affected the association between parental SEP and risk of ADHD diagnosis in offspring. Another strength of the study is that it is based on descendants of the CPC, which has made it possible to obtain information on parental SEP in childhood and adulthood although national registers on socioeconomic information did not exist in the parents' childhood. Further, the use of information from national administrative registers ensures a systematic collection of data and a limited magnitude of missing information due to the high degree of coverage of the registers.

However, several potential limitations may have influenced the study results and weakened their validity. First, in our statistical analyses we censored 350 individuals due to death, emigration and disappearance during the study period, but as we believe this censoring to be independent, it will probably not have influenced the study results. Second, our study was based on ADHD diagnoses from the Psychiatric Central Register. As the Psychiatric Central Register does not include ADHD diagnoses made in private practice, we probably have misclassified some ADHD cases as non-cases. However, since Denmark has free, universal health care, we do not believe this to be a major problem. It is noteworthy that all ADHD diagnoses in our study were registered after the implementation of the ICD-10 in 1994. This is probably due to the fact that

information on psychiatric outpatient treatments was not included in the register until 1995. However, restricting the sample to individuals born since the implementation of the ICD-10 does not significantly change the study results and we do not find evidence for changes in the association over time. Still, it is important to be aware that parents from different socioeconomic backgrounds might have different attitudes towards whether they wish for their children to receive an ADHD diagnosis or not and this might affect whose children are diagnosed with ADHD. Third, previous studies have shown the composite indicator of parental childhood SEP used in this study to be a strong predictor of offspring development [30–32]. Also, the register information on parental educational level is considered precise at the individual level, but obviously, educational level may not reflect every important aspect of parental SEP in adulthood. The crude categorization of parental educational level might further contribute to loss of information about important aspects of parental SEP in adulthood. Fourth, confounding from unmeasured factors, such as parental intelligence and personality traits, might have resulted in biased estimates of the associations between parental SEP in adulthood and risk of ADHD diagnosis in offspring. Fifth, the generalizability of the study results to other populations with regard to the strength of the investigated associations is to be determined—thus, there is a need of more methodologically similar studies and more studies of the influence of social mobility.

Conclusion

In conclusion, we found that low parental SEP—measured by parental educational level—was associated with higher risk of ADHD diagnosis in offspring. Whether this association is explained by factors such as diagnostic practice or family environment is hard to tell, so there is a need of future studies investigating the wide range of possible explanations. We also found that parental childhood SEP modified the association between parental SEP in adulthood and risk of ADHD diagnosis in offspring. Thus, our findings suggest that it is important to take into account the possibility of social mobility as changes in parental life course SEP from childhood to adulthood seem to influence the risk of ADHD diagnosis in offspring.

Compliance with ethical standards

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