



Parental socioeconomic position and risk of autism spectrum disorders in offspring: A cohort study of 9,648 individuals in Denmark 1976-2013

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ABSTRACT

Background: The results of studies of the association between parental socioeconomic position (SEP) and risk of autism spectrum disorder (ASD) in offspring are inconsistent, perhaps due to contextual differences in health care systems and their influence on risk of ASD diagnosis among different socioeconomic groups. The present study investigated the association between parental SEP in adulthood and risk of ASD diagnosis in offspring in a Nordic welfare state and whether this association was modified by parental childhood SEP.

Method: The study population comprised 9648 live-born singletons who were followed in the Psychiatric Central Register from birth in 1976–1996 until 2013. Cox regression was used to estimate hazard ratios for ASD diagnosis according to parental SEP in adulthood.

Results: The crude results showed a tendency towards higher parental SEP in adulthood being associated with higher risk of ASD diagnosis in offspring. However, the association was reversed after adjustment for possible confounders. The reversion of the direction of the association was entirely attributable to the strong confounding effect of calendar year. Further, the results showed that parental childhood SEP modified the association between parental SEP in adulthood and risk of ASD diagnosis in offspring.

Conclusions: Both methodological and contextual issues may be of great importance for the observed association between parental SEP and risk of ASD diagnosis in offspring. Particularly, the secular trends in ASD diagnoses seem to be of great importance suggesting that changes in diagnostic patterns may influence the association between parental SEP and risk of being diagnosed with ASD.

1. Introduction

Autism spectrum disorders (ASDs) have received much attention during the last years due to their increasing occurrence in the population. From being relatively rare disorders few decades ago, the worldwide prevalence is now about 1% (Lai, Lombardo, & Baron-Cohen, 2014). Whether the increasing prevalence estimate reflects a real increase in the proportion of individuals with ASDs or

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may be ascribed to factors such as earlier diagnosis, changes in diagnostic criteria and diagnostic practice, and a rise in public awareness is a much-debated question (Atladdottir et al., 2015; Elsabbagh et al., 2012). Whatever the case, there is no doubt that when so many individuals meet the diagnostic criteria for ASDs, society faces a significant public health problem.

It is a widely held belief that this public health problem is not evenly distributed among members of society: Thus, ASDs are commonly referred to as disorders of upper-class children (Hoffmann, 2012). Results from the majority of cross-sectional studies in the field support that parental socioeconomic position (SEP) is a significant risk marker for ASD in offspring finding a higher occurrence of ASD among children of parents with high SEP (Durkin et al., 2010; Liptak et al., 2008). Likewise, several cohort and case-control studies have found that children of parents with high SEP have a higher likelihood of being diagnosed with ASD (Bilder, Pinborough-Zimmerman, Miller, & McMahon, 2009; Chen, Liu, Su, Huang, & Lin, 2007; Croen, Grether, & Selvin, 2002; King & Bearman, 2011; Windham et al., 2011). However, not all cohort and case-control studies support this finding. Some cohort and case-control studies have not been able to find an association between parental SEP and risk of ASD diagnosis in offspring (Bhasin & Schendel, 2007; Larsson et al., 2005; Pinborough-Zimmerman et al., 2011; Rai et al., 2012; Windham et al., 2011), while others have found that children of parents with low SEP have a higher likelihood of being diagnosed with ASD (Burd, Severud, Kerbeshian, & Klug, 1999; Dodds et al., 2011; Lehti et al., 2015; Rai et al., 2012).

Whether the studies' inconsistent findings are due to methodological or contextual issues has been widely discussed. It is possible that methodological variations with respect to the choice of SEP indicators (e.g. parental education, occupation or income), diagnostic criteria (e.g. ICD or DSM criteria), and confounders can explain some of the discrepancies among the studies' findings. Likewise, it is possible that contextual variations can explain some of the discrepancies among the studies' findings. In particular, it has been questioned whether the many American studies' finding of positive associations can be generalized to the Nordic welfare states where all citizens have free and equal access to health services (Delobel-Ayoub et al., 2015; Durkin et al., 2017; Rai et al., 2012). The Nordic welfare states are not only interesting contexts because of their free and equal access to health services, but also because they are considered some of the most socially equal countries in the world. Thus, the rather limited number of Nordic studies in the field is surprising.

It is also surprising that in spite of the recent focus on the life course's influence on individuals' health and wellbeing, previous studies have only investigated the association between parental SEP in adulthood and risk of ASD in offspring. With the life course epidemiology's conceptual model of social mobility in mind, it seems obvious to expand this narrow perspective and investigate whether changes in parental life course SEP influence the risk of ASD diagnosis in offspring. In particular, it seems obvious to investigate whether parental SEP in childhood may modify the influence of parental SEP in adulthood on the risk of ASD diagnosis in offspring.

The objectives of this study were therefore to investigate the association between parental SEP in adulthood and risk of ASD diagnosis in offspring in a Nordic welfare state context and to investigate whether parental childhood SEP modified the association between parental SEP in adulthood and risk of ASD diagnosis in offspring.

2. Methods

2.1. Study population

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

The CPC consisted of all pregnant women who were admitted to the maternity departments of the Copenhagen University Hospital from 21st October 1959 to 21st December 1961 - corresponding to 8949 women (generation 1) and their 9125 children (generation 2) (Mortensen, 1997). About 7600 women in generation 1 and 8100 men and women in generation 2 could be identified in the Civil Registration System. Among these, 5032 women in generation 1 and 5222 men and women in generation 2 (2431 men and 2791 women) were registered with one or more live-born singleton descendants (generation 3). The 9648 individuals in generation 3 constitute the population at risk. The total 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 ASD diagnosis, attrition or June

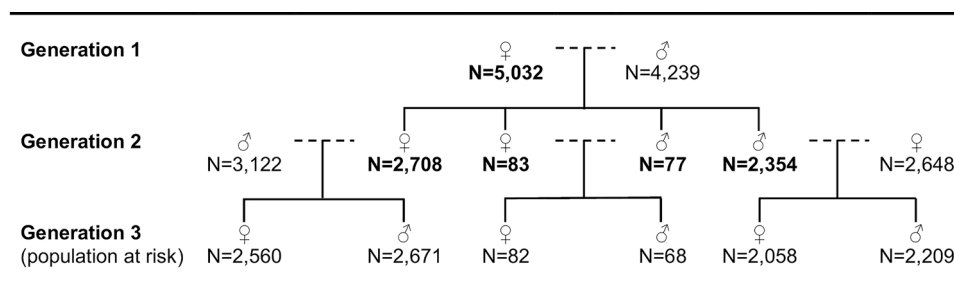


Fig. 1. Overview of the study population^a.

^aThe 5032 women (generation 1) and the 5222 men and women (generation 2) marked in bold type are the members of the Copenhagen Perinatal Cohort.

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