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Current changes in the occurrence of Autism Spectrum Disorders in Stockholm

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ABSTRACT

The overall objective of this thesis is to estimate recent changes in and current prevalence of autism spectrum disorders (ASD) among young people in Stockholm County. An additional objective is to explore potential risk factors for ASD in view of the increasing occurrence in the population. For this purpose, a register-based total population study was set up and ASD case ascertainment validated as a means and research model for achieving the overall objective.

All studies were based on the Stockholm Youth Cohort (SYC), a longitudinal total population study of 0-17 year olds resident in Stockholm County at any time since 2001. Prospectively compiled data for this population were merged from regional and national registers. In study I, we found that 96.0% of clinical case notes from randomly sampled ASD cases in the SYC were consistent with a diagnosis of ASD. Furthermore, we confirmed ASD in 82.5% of affected twins in the SYC by means of cross-validation against a twin study. In study II, we reported that ASD prevalence at the end of 2011 was 1.5% among 0-27 year olds (N=735,096), of whom 25.9% had a registered diagnosis of ID. The ASD prevalence was highest among teenagers at 2.4%. The male: female prevalence ratio for ASD decreased with age (from 3.3:1 among 0-12 year olds, to 1.9:1 among 18-27 year olds), particularly for ASD without ID. Between 2001 and 2011, the prevalence of ASD increased almost 3.5 fold among 2-17 year olds, mainly due to an eightfold increase of ASD without ID. In contrast, the prevalence of ASD with ID increased only slightly during this period.

The recent increase in ASD prevalence has attracted research interest toward risk factors for ASD that have increased in a parallel manner, such as parental age and weight. In study III, we found that higher parental age increased the risk of offspring ASD as well as stronger parental age effects for ASD with, than without, ID. We found the risk of ASD to be greater for offspring of older mothers than for those of older fathers. Furthermore, the paternal age effect on ASD risk was only evident among offspring to mothers aged 35 years or younger, while maternal age increased the risk of ASD regardless of paternal age. In the population-based analysis of study IV, we found that maternal overweight increased the risk of ASD, while no such effect was evident in the sibling analysis. In addition to the finding that too much weight gain during pregnancy increases the risk of offspring ASD, this study was the first to report that too little weight gain also constitutes a risk.

In conclusion, the prevalence of identified ASD without comorbid ID has increased substantially between 2001 and 2011 in Stockholm, and ASD currently affects more than 2% of teenagers, with important implications for the planning of health and educational services. Changes in diagnostic practice and awareness are likely to be the main drivers of the rise, but an actual true increase in ASD incidence cannot be ruled out. Collectively, these studies confirm the relevance of categorizing ASD according to ID. Finally, the SYC, with its extensive register-based data as well as a valid and thorough ASD case ascertainment constitutes an important resource for ASD research.