Chapter 12: Epidemiology of Autism Spectrum Disorders

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Abstract In this chapter, we review existing prevalence estimates for ASDs since 2000 and discuss methodological factors impacting the estimation of prevalence and the interpretation of changes in prevalence estimates over time. Possible explanations for an increase in the prevalence of ASD within and across populations are considered. Increases in ASD diagnostic rates cannot currently be attributed to a true increase in the incidence of ASD due to multiple confounding factors. It remains to be seen how changes to diagnostic criteria introduced in the DSM-5 will impact estimates of ASD prevalence going forward.

1. Introduction

Epidemiological surveys of autism were first initiated in the mid-1960s in England (Lotter, 1966; 1967) and have since been conducted in over 20 countries. In this chapter, we

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provide a comprehensive review of the findings and methodological features of published epidemiological surveys about the prevalence of autism spectrum disorders (ASDs[1]). This chapter builds upon previous reviews (Elsabbagh et al., 2012; Fombonne, 2003a; 2005; Fombonne, Quirke, & Hagen, 2011; French, Bertone, Hyde, & Fombonne, 2013; Hill, Zuckerman, & Fombonne, 2014; J. G. Williams, Higgins, & Brayne, 2006) and includes the results of pertinent studies since published. The specific questions addressed are: (1) What is the range of prevalence estimates for ASDs?; and (2) How should the time trends observed in the current prevalence rates of ASDs be interpreted?

1.1. Study Design and Methodological Issues

Epidemiologists use several measures of disease occurrence including incidence, cumulative incidence, and prevalence. Prevalence is a measure used in cross-sectional surveys (in which there is no passage of time) and reflects the proportion of subjects in a given population who suffer from the disease at that point in time. Most epidemiological studies of ASDs have assessed prevalence (point prevalence or period prevalence) as a cross-sectional approach is more appropriate for disorders where timing of diagnosis lags behind onset of symptoms and is likely to be influenced by a range of factors unrelated to risk. In designing a prevalence study, three elements are critical: case definition, case identification (or case ascertainment), and case evaluation methods (Fombonne, 2007).

1.1.1. Case Definition

The definition and diagnostic criteria of autism has changed over time. Starting with Kanner's definition of autism (1943), case definitions have progressively broadened to include

[1] Autism spectrum disorder (ASD) is the modern term that replaces the former pervasive developmental delay (PDD).

criteria proposed by Rutter (1970), and subsequently the International Classification of Diseases, ninth revision (ICD-9; World Health Organization, 1977); the *Diagnostic and Statistical Manual of Mental Disorders*, third edition (*DSM-III*; American Psychiatric Association [APA], 1980), until two recent nosographies were adopted worldwide; ICD-10 (World Health Organization, 1992) and the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition and text revision (*DSM-IV* and *DSM-IV-TR*, respectively; APA, 1994; 2000).

Early diagnostic criteria reflected the more qualitatively severe behavioral phenotypes, usually associated with severe delays in language and cognitive skills. In the 1980s less severe forms of autism were recognized, either as a qualifier for autism occurring without intellectual disability (i.e., high-functioning autism), or as separate diagnostic categories (e.g. Pervasive Developmental Disorders Not Otherwise Specified [PDD-NOS] or Autism Spectrum Disorders [ASD]). Asperger's disorder appeared in the 1990s, with unclear validity, particularly with respect to its differentiation from high-functioning autism. Some ASD subtypes that were described in *DSM-III* subsequently disappeared (e.g., Autism-Residual State); however, other nomenclatures have since added new diagnostic categories, such as "atypical autism" and "PDD unspecified" (ICD-10).

The changes now occurring with the introduction of *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (*DSM-5*; APA, 2013), may impact prevalence estimates in the future. *DSM-5* proposes a single new category of Autism Spectrum Disorders, conceptually equivalent to the previous diagnostic class of PDDs. However, fewer diagnostic criteria have been retained that are combined in two clusters of social communication deficits and restricted patterns of behaviors and interests. The removal of the loosely defined PDD-NOS that was in *DSM-IV-TR* (APA, 2000) will likely increase the specificity of the ASD diagnostic category, and

the removal of Asperger Disorder as a separate category is consistent with research that has generally failed to provide evidence for the discriminant validity of this diagnostic concept vis-à-vis forms of autistic disorder that are not associated with severe language impairments or intellectual deficits.

The impact of *DSM-5* changes remains to be fully assessed in the context of epidemiological surveys. Two recent population-based surveys have addressed this issue. Maenner and colleagues (2014) retrospectively applied the new diagnostic criteria to a previously obtained population-based sample from the Centers for Disease Control and Prevention (CDC) 2006 and 2008 surveillance years. They found that 81.2% of children classified as having ASD according to *DSM-IV-TR* (APA, 2000) also met *DSM-5* criteria (APA, 2013), resulting in a *DSM-5* based prevalence of 100/10,000 – an estimate lower than the 2006 and 2008 estimates. In addition, 304 children met *DSM-5* but not *DSM-IV-TR* criteria. In a similar study, Kim and colleagues (2014) reported that 92% of children with ASD according to *DSM-IV-TR* also met *DSM-5* criteria. However, when *DSM-5* ASD and Social Communication Disorder (SCD; a new diagnostic category in *DSM-5*) were considered together, there was no significant change in the prevalence estimate (Kim et al., 2014). It is important to note that new diagnostic information required in *DSM-5* (e.g., emphasis on sensory processing deficits) is generally not available in prior studies, leading to potentially biased estimates. Additionally, previous studies are often constrained in sampling children with a *DSM-IV* PDD diagnosis and cannot therefore accurately estimate the proportion of children who did not meet criteria for *DSM-IV* yet would have met those for *DSM-5*.

While there is currently high interrater reliability overall regarding diagnosis of ASDs and commonality of concepts across experts, differences still persist between nomenclatures

about the terminology and operationalized criteria of ASDs. It is unclear to what extent the changing nomenclature of ASDs plays a role in prevalence estimates described in epidemiological studies. More studies are on their way that will provide further examination of the impact on prevalence estimates of narrowing the ASD definition in *DSM-5*.

1.1.2. Case Identification/Ascertainment

When a population is identified for a survey, different strategies are employed to find individuals matching the study's case definition. Some studies rely solely on service provider databases (Chien, Lin, Chou, & Chou, 2011; Croen, Grether, Hoogstrate, & Selvin, 2002b; Davidovitch, Hemo, Manning-Courtney, & Fombonne, 2013), special education databases (Fombonne, Zakarian, Bennett, Meng, & McLean-Heywood, 2006; Gurney et al., 2003; Lazoff, Zhong, Piperni, & Fombonne, 2010; Maenner & Durkin, 2010), or national registers (Al-Farsi et al., 2011; Parner et al., 2012; Samadi, Mahmoodizadeh, & McConkey, 2011) for case identification. These studies have the common limitation of relying on a population group that was readily accessible, rather than sampling from the population at large. As a result, individuals with the disorder who are not in contact with services are not included as cases, leading to an underestimation of prevalence. This limitation is particularly problematic in communities with recognized limitations in available services.

Other investigations have relied on a multistage approach to identify cases in underlying populations (e.g., CDC, 2014; Idring et al., 2012; Kim et al., 2011). In these studies' first screening stage, a wide net is cast to identify subjects possibly affected with ASD, with the final diagnostic status being determined at subsequent stages. This process often consists of sending letters or screeners to school and health professionals, searching for possible cases of autism. Few such investigations rely on systematic sampling techniques that would ensure a near

complete coverage of the target population, and screening often varies substantially in ascertainment of all relevant data sources. Additionally, surveyed areas often differ in terms of specific educational or health care systems available, and inclusion information sent often varies in reliability and validity. Finally, uneven participation rates in the screening stage can lead to variation in the screening efficiency of surveys.

To illustrate how differential participation in the screening stage affect prevalence estimates, two hypothetical scenarios are illustrated in Figure 1, both of which are based on a true ASD prevalence of 150/10,000 and a sensitivity of 100% for the screening process and total accuracy in the diagnostic confirmation. In Scenario A, we assume 60% participation for ASD and non-ASD cases in the first screening stage, resulting in 90 participating ASD cases that screen positive. With 70% participation for both ASD and non-ASD cases in the diagnostic stage, we would identify and confirm 63 ASD cases in the second phase. Weighting back phase 2 data, we would obtain an unbiased prevalence estimate of 1.5% (or 150/10,000) in this scenario. In Scenario B, we also assume 60% overall participation, but with a 80% participation rate for ASD cases, reflecting a scenario in which individuals with ASD are more likely to participate in the first screening stage than non-ASD cases. Thus, with the same participation rates in the first screening (60%) and final diagnostic stages (70%), we identify 84 ASD cases and calculate a biased prevalence estimate of 2% (200/10,000), an estimate that is 0.5% higher than true prevalence. The bias arises for two reasons: (1) participation in screening is associated with case status (here, with ASD cases more likely to participate than non-cases); and (2) as investigators typically have no such information, weights used for prevalence estimation were not adjusted correspondingly, resulting in the upward bias.

[INSERT FIGURE 1 HERE]

It is also possible that individuals with ASD participate less than non-cases, which would result in underestimates of prevalence. For example, Posserud and colleagues (2010) reported ASD prevalence of 72/10,000 in their identified sample and estimated a prevalence of 128/10,000 in nonresponders (based on teacher ratings during the screening phase), indicating increased refusal rates among those with more ASD symptoms. Unfortunately, few studies have been able to estimate the extent to which willingness or refusal to participate is associated with final caseness, so it is not known what effect differential participation rates at different phases in population surveys may have on prevalence estimates

The sensitivity of the screening methodology is difficult to gauge in autism surveys, as the proportion of children truly affected with the disorder but not identified in the screening stage (false negatives) remains generally unmeasured. Few studies provided an estimate of the reliability of the screening procedure. The usual approach, which consists of randomly sampling screen-negative subjects to adjust estimates, has not been generally used, mainly due to the relatively low frequency of ASD, which makes such a strategy both imprecise and costly.

As an example, the surveys conducted by the CDC (2007a; 2007b; 2009; 2012; 2014) rely, for case ascertainment, on scrutinizing educational and medical records. Children not accessing such services cannot be identified. Although some recent surveys that systematically screen the normal school population might detect a large pool of unidentified cases (Kim et al., 2011), it remains to be seen if this applies to most populations and requires change in sampling approaches for surveying autism. Of note, the CDC methodology identifies ASD cases without prior official ASD diagnosis (21% of identified cases in 2008; CDC, 2012), suggesting that underidentification is a widespread phenomenon.

Since more recent prevalence studies suggest that autism can no longer be regarded as rare, screening for false negatives may become a more common strategy. Currently, however, prevalence estimates must be understood as underestimates of "true" prevalence rates, with the magnitude of this underestimation unknown in each survey.

1.1.3. Case Evaluation

When the screening phase is completed, subjects identified as positive go through a more in-depth diagnostic evaluation to confirm case status. Similar considerations about methodological variability across studies apply in more intensive assessment phases. The information used to determine diagnosis usually involves a combination of data from informants (parents, teachers, pediatricians, other health professionals, etc.) and data sources (medical records, educational sources), with a direct assessment of the person with autism being offered in some but not all studies. When subjects are directly examined, assessments typically use various diagnostic instruments, ranging from a typical unstructured examination by a clinical expert (but without demonstrated psychometric properties) to the use of batteries of standardized measures by trained research staff. The Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter, & Couteur, 1994) and/or the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000) have been increasingly used in the most recent surveys (Table 1).

Obviously, surveys of large populations, such as those conducted in the United States' CDC ADDM Network (2007a; 2007b; 2009; 2012; 2014) or in national registers (Idring et al., 2012), cannot include direct diagnostic assessment of all subjects by researchers. However, investigators generally improve the accuracy of caseness determinations by undertaking, on a randomly selected subsample, a more complete diagnostic workup (Rice et al., 2007). The CDC surveys have established a methodology for surveys of large populations based on screening of

the population using multiple data sources, standardized records abstraction, and systematic review and scoring of the data gathered in the screening phase. In the less obvious cases, this information is combined with input from experienced clinicians with known reliability and validity. This methodology is adequate for large samples, and is likely to be used in the future for surveillance efforts.

2. Systematic Review of Prevalence Estimates

2.1. Unspecified ASDs in Earlier Surveys

A new objective of more recent epidemiological surveys has been to estimate the prevalence of all disorders falling onto the autism spectrum, thereby prompting important changes in the conceptualization and design of surveys. However, in previous reviews, we documented that several studies performed in the 1960s and 1970s provided useful information on rates of syndromes similar to autism but not meeting the strict diagnostic criteria for autistic disorder then in use (Fombonne, 2003a; 2003b; 2005). At the time, different labels were used by authors to characterize these clinical pictures, such as the *triad of impairments* involving deficits in reciprocal social interaction, communication, and imagination (Wing & Gould, 1979), autistic mental retardation (Hoshino, Kumashiro, Yashima, Tachibana, & Watanabe, 1982), borderline childhood psychoses (Brask, 1970), or autistic-like syndromes (Burd, Fisher, & Kerbeshian, 1987). These syndromes would fall within our currently defined autistic spectrum, probably with diagnostic labels such as atypical autism and/or PDD-NOS. In 8 of 12 surveys providing separate estimates of the prevalence of these developmental disorders, higher rates for the atypical forms were actually found compared to those for more narrowly defined autistic disorder (see Fombonne, 2003a). However, this atypical group received little attention in previous epidemiological studies; these subjects were not defined as “cases” and were not included in the

numerators of prevalence calculations, thereby underestimating systematically the prevalence of what would be defined today as the spectrum of autistic disorders.

For example, in the first survey by Lotter (1966), the prevalence would rise from 4.1 to 7.8/10,000 if these atypical forms of autism had been included in the case definition. Similarly, in Wing, Yeates, Brierly, & Gould's study (1976), the prevalence was 4.9/10,000 for autistic disorder, but the prevalence for the whole ASD spectrum was in fact 21.1/10,000 after the figure of 16.3/10,000 (Wing & Gould, 1979), corresponding to the triad of impairments, was added. The progressive recognition of the importance and relevance of these less typical clinical presentations has led to changes in the design of more recent epidemiological surveys that use case definitions that incorporate a priori these milder phenotypes, which we now turn to.

2.2. Search Strategies

Keeping in mind the range and limitations of case definition, identification, and evaluation methods employed in epidemiological surveys, we present the results of epidemiological reports conducted since 2000 in Table 1. These reports were identified from previous reviews of epidemiological surveys (Elsabbagh et al., 2012; Fombonne, 2003a; 2003b; 2005; 2009b; Fombonne et al., 2011; French et al., 2013; J. G. Williams et al., 2006) and through systematic searches using major scientific literature databases (Medline, PsycINFO, Embase, PubMed). Where multiple surveys based on the same or overlapping populations were evident, the publication listed is the most detailed and comprehensive account. For example, surveys conducted by the U.S. CDC (2007a; 2007b; 2009; 2012; 2014) as part of the Autism and Developmental Disabilities Monitoring (ADDM) Network are each included in the table, although additional accounts for individual states are available elsewhere (Nicholas et al., 2008; Pinborough-Zimmerman et al., 2012; Rice et al., 2010; Zahorodny et al., 2014).

2.3. Inclusion and Exclusion Criteria

The following criteria were set to select epidemiological surveys included in Table 1:

- The full article was published in English.
- The minimum population was 5,000.
- The survey included independent validation of caseness by professionals. In addition, surveys that imposed further non-ASD criteria were excluded.
- The following information categories were included or could be ascertained based on information from the survey: country and area where the survey was conducted, size of the population for which the prevalence estimate was ascertained, age range of participants, number of children affected, diagnostic criteria used in case definition, and prevalence estimate (number per 10,000). Where available, we also report the proportion of subjects with IQ within the normal range and gender ratios.

2.4. Prevalence Estimates for Combined ASDs since 2000

The results of the 53 surveys that estimated the prevalence of the whole spectrum of ASDs are summarized in Table 1. All selected surveys were published since 2000, with the majority (55%) published in 2009 or later. The studies were performed in 18 different countries (including 14 in the United Kingdom and 12 in the United States, of which 5 were conducted by the CDC). Sample sizes ranged from 5,007 to 4.5 million (median: 58,654; mean: 346,776). Ages of the surveyed populations ranged from 0 to 98 (median: 8; mean: 9). One study was specifically conducted on adults and provided the only estimate (98.2/10,000) thus far available for adults (Brugha et al., 2011). Two surveys focusing on toddlers (Nygren et al., 2012) and preschoolers (Nicholas, Carpenter, King, Jenner, & Charles, 2009) provided estimates of

approximately 80 per 10,000. In the 50 remaining surveys, the average median age was 8.23 years (*SD* = 2.8).

[INSERT TABLE 1 HERE]

The diagnostic criteria used in 53 studies reflected the reliance on modern diagnostic schemes (11 studies used ICD-10, 25 the *DSM-III*, *DSM-IV*, or *DSM-IV-TR*; both schemes being used simultaneously in 9 studies). Assessments were often performed with standardized diagnostic measures (i.e., ADI-R and ADOS). In 26 studies where IQ measures were reported, the proportion of subjects within the normal IQ range varied from 0% to 100% (median: 55.4%; mean: 53.9%), a proportion that reflects the lesser association, or lack thereof, between intellectual impairment and milder forms of ASDs. Overrepresentation of males was seen in the 47 studies reporting gender ratios, with male/female ratio ranging from 1.8:1 to 15.7:1 (median: 4.5:1; mean: 4.9:1).

There was a 189-fold variation in ASD prevalence, ranging from 1.4/10,000 to 264/10,000 (see Figure 2). There was also substantial variation in confidence interval width, reflecting variation in sample sizes and consequently in each study's precision (range: 0.5–146; mean interval width: 22.4). However, some consistency in ASD prevalence is found in the center of this distribution, with a median rate of 61.9/10,000 and a mean rate of 68.9/10,000 (interquartile range: 44.2–84.0/10,000). Prevalence was negatively associated with sample size (Kendall's tau: -.23, $p = .01$), with small-scale studies reporting higher prevalence.

There was also a significant positive correlation between ASD prevalence estimates and publication year (Kendall's tau: .26, $p = .007$), with higher rates in more recent surveys. Eight studies since 2000 reported ASD prevalence estimates higher than 100/10,000 (Baird et al., 2006; CDC, 2012; CDC, 2014; Idring et al., 2012; Kawamura, Takahashi, & Ishii, 2008; Kim et

al., 2011; Ouellette-Kuntz et al., 2006b; Saemundsen, Magnusson, Georgsdóttir, Egilsson, & Rafnsson, 2013). Baird et al. (2006) and Kim et al. (2011) both employed proactive case finding techniques, relying on multiple and repeated screening phases, involving both different informants at each phase and surveying the same cohorts at different ages, which certainly enhanced the sensitivity of case identification. Multisource active surveillance techniques, as employed in the Stockholm Youth Cohort (Idring et al., 2012) and by the CDC's ADDM Network (2007a; 2007b; 2009; 2012; 2014), also improve identification of individuals with ASD. The most recent CDC prevalence estimate of 147 per 10,000 reflects the highest estimate to date across all of the previous ADDM Network reports (CDC, 2014).

[INSERT FIGURE 2 HERE]

Overall, results of recent surveys agree that an average figure of 69/10,000 can be used as the current estimate for the spectrum of ASDs. The convergence of estimates around 60 to 90 per 10,000 for all ASDs combined, conducted in different regions and countries by different teams, is striking especially when derived from studies with improved methodology. The prevalence figure of 69/10,000 (equivalent to 6.9/1,000 or .69%) translates into 1 child out of 145 with an ASD diagnosis. This estimate is now the best current estimate for the ASD prevalence. However, it represents an average and conservative figure, and substantial variability exists between studies and within studies, across sites or areas.

3. Time Trends in Prevalence and Their Interpretation

The debate on the hypothesis of a secular increase in rates of autism has been obscured by a lack of clarity in the measures of disease occurrence. As noted previously, it is crucial to differentiate prevalence from incidence, since only incidence rates can be used for causal research, and prevalence and incidence will increase when case definition is broadened or case

ascertainment is improved. Moreover, epidemiological surveys of ASDs possess unique design features that could account almost entirely for between-study variation in prevalence estimates, making time trends even more difficult to gauge. Time trends in prevalence estimates can therefore only be evaluated in investigations that hold methodological parameters under strict control over time. Such requirements must be considered when reviewing evidence for a secular increase in rates of ASDs, or testing for the "epidemic" hypothesis.

The epidemic hypothesis emerged in the 1990s when, in most countries, increasing numbers were diagnosed with ASDs leading to an upward trend in children registered in service providers' databases that was paralleled by higher prevalence rates in epidemiological surveys. These trends were interpreted as evidence that the actual population incidence of ASDs was increasing. However, because methodological factors contribute to variability in prevalence estimates, these must be considered before concluding that there is a true rise in the number of children diagnosed with ASDs and include the following:

3.1. Use of Referral Statistics

Increasing numbers of children referred to specialist services or known to special education registers have been taken as evidence for increased ASD incidence. Such upward trends have been seen in many different countries (Gurney et al., 2003; Lotter, 1966; Shattuck, 2006; Taylor et al., 1999), all occurring in the late 1980s and early 1990s. However, trends over time in *referred* samples are confounded by referral patterns, availability of services, heightened public awareness, decreasing age at diagnosis, and changes over time in diagnostic concepts and practices.

[INSERT FIGURE 3 HERE]

As an illustration, Figure 3 contrasts two methods for surveying ASD using hypothetical data: one based on sampling from the total population, and the other relying solely on service access counts. Here, assuming a constant incidence and prevalence of 100/10,000 between Time 1 and Time 2 (meaning there is no epidemic), population surveys at two time points result in prevalence estimates that are not only accurate but also stable over time, showing no prevalence change in the target population. However, if prevalence is estimated based only on service access counts where the number of ASD individuals accessing services increases from 20% to 60% over time, prevalence would be underestimated at both time points, yet would appear to rise 200% while the underlying true incidence and prevalence remained stable. Such a pattern of results was recently reported based on special education data in Wisconsin (Maenner & Durkin, 2010), in which ASD prevalence rates were stable between 2002 and 2008 in school districts with initially high baseline prevalence rates (≈120/10,000), whereas school districts with low baseline rates experienced significant increases in prevalence (e.g., in one district rates rose from 5 to 70/10,000; corresponding to a 1300% increase in 6 years). Failure to control for these confounding factors was obvious in previous reports (Fombonne, 2001), including widely quoted reports from California Developmental Database Services (CDDS, 2003).

Additionally, the decreasing age at diagnosis results in itself to increasing numbers of young children being identified in official statistics (Wazana, Bresnahan, & Kline, 2007) or referred to specialist medical and educational services. Earlier identification of children from the prevalence pool may therefore result in increased service activity that may lead to a misperception by professionals of an epidemic.

3.2. Diagnostic Substitution

Another possible explanation for increased prevalence in a diagnostic category is that children presenting with the same developmental disability may receive one particular diagnosis initially and another diagnosis subsequently. Such diagnostic substitution (or switching) may occur when diagnostic categories become increasingly familiar to health professionals and/or when access to better services is ensured by using a new diagnostic category.

The strongest evidence of diagnostic substitution contributing to ASD prevalence increase was shown in a complex analysis of Department of Education Data in 50 U.S. states (Shattuck, 2006), indicating that a relatively high proportion of children previously diagnosed with mental retardation were subsequently identified as having ASD. Shattuck showed that the odds of having ASD increased by 1.21 during 1994–2003 while the odds of having learning disability (LD) (odds ratio [OR] = 0.98) and mental retardation (MR) (OR = 0.97) decreased. Shattuck (2006) further demonstrated that the growing ASD prevalence was directly associated with decreasing prevalence of LD and MR within states, and that a significant downward deflection in the historical trajectories of LD and MR occurred when ASD became reported in the United States as an independent category in 1993–1994.

Using individual level data, a newer study reexamined the hypothesis of diagnostic substitution in the California DDS dataset (M. King & Bearman, 2009) and showed that 24% of the increase in caseload was attributable to diagnostic substitution (from MR to ASD). It is important to keep in mind that other types of diagnostic substitution are likely to have occurred as well for milder forms of ASD. For example, children currently diagnosed with Asperger's disorder may be previously diagnosed with other psychiatric diagnoses (i.e., obsessive-

compulsive disorder, school phobia, social anxiety, etc.) in clinical settings before the developmental nature of their condition was fully recognized (Fombonne, 2009a).

3.3. Cross-Sectional Variability in Epidemiological Surveys

Evidence that method factors could account for most of the variability in published prevalence estimates comes from a direct comparison of eight recent surveys conducted in the United Kingdom and the United States (Fombonne, 2005). In each country, four surveys were conducted around the same year and with similar age groups. As there is no reason to expect large variations in between-area differences in rates, prevalence estimates should therefore be comparable within each country. However, there was a 6-fold variation in rates for U.K. surveys, and a 14-fold variation in U.S. rates. In each set of studies, high rates were found when intensive population-based screening techniques were employed, whereas lower rates were found in studies relying on passive administrative methods for case finding. Since no passage of time was involved, the magnitude of these gradients in rates is likely to reflect methodological differences.

Even more convincing evidence comes from the most recent survey by the CDC on 363,749 children aged 8 in 2010, where an average prevalence of 147/10,000 was reported across 11 U.S. states (CDC, 2014). One striking finding in this report is the almost four-fold variation in prevalence rates by state (range: 57–219 per 10,000; see Figure 4). Across individual states, Alabama had the lowest rate of 57/10,000, whereas New Jersey had the highest rate of 219/10,000 (CDC, 2014). Estimated ASD prevalence was significantly lower in states that had access to health data sources only compared to that of states where educational data was also available (97.7 versus 149 out of 10,000, respectively), a factor that is consistently associated with higher prevalence rates in the ADDM Network. It would be surprising if there were truly this much inherent state-to-state variability in the number of children with autism in the United

States. Thus, these differences likely reflect ascertainment variability across sites in a study that was otherwise performed with the same methods, at the same time, on children of the same age, and within the same country.

[INSERT FIGURE 4 HERE]

3.4. Repeated Surveys in Defined Geographical Areas

Repeated surveys, using the same methodology and conducted in the same geographical area at different time-points, can potentially yield useful information on time trends if methods are kept relatively constant. The Göteborg studies (C. Gillberg, 1984; C. Gillberg, Steffenburg, & Schaumann, 1991) provided three prevalence estimates that increased over a short period of time from 4.0 (1980) to 6.6 (1984) to 9.5/10,000 (1988), the gradient being even steeper in urban areas only (C. Gillberg et al., 1991). However, comparison of these rates is not straightforward, as different age groups were included in each survey. Furthermore, increased prevalence was associated with improved detection among those with intellectual delays in the second survey, and with improved detection of cases born to immigrant parents in the third survey, suggesting that migration into the area could be a key explanation. Taken in conjunction with a change in local services and a progressive broadening of the autism definition over time (C. Gillberg et al., 1991), findings provide weak evidence for increased autism incidence. Similarly, studies conducted in Japan at different points in time in Toyota (Kawamura et al., 2008) and Yokohama (Honda, Shimizu, & Rutter, 2005; Honda, Shimizu, Misumi, Niimi, & Ohashi, 1996) showed rises in prevalence rates that their authors interpreted as reflecting the effect of both improved population screening of preschoolers and a broadening of diagnostic concepts and criteria.

Two separate surveys of children born between 1992 and 1995 and between 1996 and 1998 in Staffordshire, United Kingdom (Chakrabarti & Fombonne, 2001; 2005), were performed

with rigorously identical methods for case definition and case identification. The prevalence for combined ASDs was comparable and not statistically different in the two surveys (Chakrabarti & Fombonne, 2005), suggesting no upward trend in overall rates of ASDs, at least during the short time interval between studies.

3.5. Birth Cohorts

In large surveys encompassing wide age ranges, increasing prevalence among most recent birth cohorts could be interpreted as indicating a secular increase in ASD incidence, provided that alternative explanations can be confidently eliminated. This analysis was used in two large French surveys (Fombonne & Mazaubrun, 1992; Fombonne, Mazaubrun, Cans, & Grandjean, 1997). The surveys included birth cohorts from 1972 to 1985 (735,000 children, 389 of whom had autism). When pooling the data of both surveys, age-specific rates showed no upward trend (Fombonne et al., 1997).

However, data assessing birth cohorts can be problematic, as illustrated in Figure 5, which shows an increase in the prevalence of ASD by year of birth across three hypothetical successive birth cohorts (a cohort effect; Figure 5a). Within each birth cohort, followed longitudinally, prevalence increases as children age (Figure 5b): for children in the 2000 birth cohort, based on previous ASD prevalence estimates, age 6 prevalence is 20/10,000, whereas at age 12, we may expect prevalence of 80/10,000 for the same birth cohort. Increasing prevalence rates with age within birth cohorts is unlikely to reflect the onset of ASD in later childhood and early adolescence. It is more likely that observed increases in prevalence reflect underdiagnosis in the preschool years as well as changes in public awareness, service availability, and diagnostic concepts and practices.

[INSERT FIGURE 5 HERE]

As an example, an analysis of special educational data from Minnesota showed a 16-fold increase in children identified with ASD from 1991–1992 to 2001–2002 (Gurney et al., 2003). However, during the same time period, an increase of 50% was observed for all disability categories (except severe intellectual deficiency), especially for the category including attention-deficit/hyperactivity disorder (ADHD). The large sample size allowed the authors to assess age, period, and cohort effects. Prevalence increased regularly in successive birth cohorts; for example, among 7-year-olds, prevalence rose from 18/10,000 among those born in 1989, to 29/10,000 among those born in 1991, to 55/10,000 in those born in 1993. Within the *same* birth cohorts, age effects were also apparent since for children born in 1989 the prevalence rose with age from 13/10,000 at age 6, to 21/10,000 at age 9, and 33/10,000 at age 11. As argued by Gurney et al. (2003), this pattern is not consistent with the natural etiology of ASD, which first manifests in early childhood. Gurney et al's analysis also showed a marked period effect, where rates started to increase in all ages and birth cohorts in the 1990s. The authors noted that this phenomenon coincided closely with the inclusion of ASDs in the federal Individuals with Disabilities Educational Act in the United States. A similar interpretation of upward trends had been put forward by Croen and colleagues (2002b) in their analysis of the California DDS data, and by Shattuck (2006) in his analysis of trends in U.S. Department of Education data.

4. Correlates of ASDs in Epidemiological Surveys

Studies of associations between ASDs and socioeconomic status (SES), race/ethnicity, and immigrant status have shown variable results and face numerous technical challenges. In general, studies that base diagnosis rates on developmental service utilization may undercount minority and low SES children. Underprivileged children have less health services access overall (Shi & Stevens, 2005) and particularly low mental health services access (Kataoka, Zhang, &

Wells, 2002), which can lead to underidentification of ASD. In contrast, children with more educated, wealthier, or more health-literate parents may have resources to make their way to ASD diagnostic services and, therefore, an ASD diagnosis (Tsai, Stewart, Faust, & Shook, 1982). Cross-sectional studies based on parent report of ASD are problematic for the same reason, as parent report of ASD is more likely among families who have adequate access to ASD-related services. Undercounting of minorities may additionally occur in the context of multistage, population-based research. Minority and low SES families may participate in such research studies at disproportionately low rates, due to higher rates of distrust of scientific researchers (Rajakumar, Thomas, Musa, Almario, & Garza, 2009) or less access to research opportunities. They also may be excluded from studies or incorrectly assessed if forms are not available in appropriate languages or if a language-congruent assessor is not available (Laing & Kamhi, 2003). Finally, because ASD is a relatively rare event, population-based studies of ASD prevalence may have relatively small numbers of low SES, minority, or immigrant children meeting case criteria, making data difficult to interpret (Powell et al., 2000; Sponheim & Skjeldal, 1998).

4.1. Socioeconomic Status

Socioeconomic status can be defined variously, the most common methods being parental education, income, parental occupation, or some combination of these factors. Over 20 studies have investigated associations between these factors and ASD prevalence.

Many recent U.S.-based studies suggest an association between higher SES (as assessed by one of these factors) and higher ASD prevalence. Several recent studies have used CDC ADDM data combined with imputed sociodemographic data from U.S. Census tracts to show a link between parental income/education and ASD diagnosis. Using 2007 data from New Jersey,

(P. Thomas et al., 2012) showed that the ASD prevalence ratio between the highest income tract (>$90,000 USD) and the lowest income tract (< $30,000 USD) was 2.2. In addition, children in the higher income tracts were more likely to have a higher number of professional evaluations and a lower age of diagnosis, suggesting a referral bias or an under-diagnosis of children at the lower end of the SES spectrum. Using CDC ADDM data from all 14 participating states, Durkin et al. (2010) developed a composite SES indicator that took into account both parental education and household income. This study found a dose-response relationship between SES and ASD prevalence, regardless of gender and data source. SES-based differences in prevalence were significantly weaker when children with a previous ASD diagnosis (as opposed to a new diagnosis in context of the study) were excluded, a finding that suggests that prior access to ASD diagnostic services may explain some of the difference. Both of these studies benefit from a population-based data collection framework; however, they are limited in that no individual level SES data was available.

Similarly, Bhasin and Schendel (2007) conducted a population-based case-control study, directly measuring maternal education and imputing household income from census tract data in Atlanta, Georgia. Higher median family income was significantly associated with autism overall. Both markers of higher SES (higher maternal education and higher median family income) were significantly associated with autism without intellectual disability (ID) but not autism with ID, suggesting that, in addition to biases based on service access, diagnostic substitution may be occurring more frequently among children with higher SES. Leonard et al. (2011) observed a similar finding in Western Australian children born from 1984 to 1999. The prevalence of ASD without ID was significantly increased among children whose mothers had more economic resources.

One criticism of these recent studies, particularly the studies based in the United States, is that SES has been confounded by inequitable health services access, and that in a setting where health services access is more equitable, the effects of SES might be lessened or even reversed. In a Denmark population-based case-control study, Larsson et al. (2005) found that the risk of ASD was actually higher among children with less parental wealth in bivariate analyses, but that after adjusting for other demographic factors, there was no association of either parental education or wealth with ASD. In a Swedish case-control study by Rai and colleagues (2012b), children in families with lower income and whose parents had manual occupations were at higher risk for ASD diagnosis after multivariate adjustment. In England, which also has national health insurance, Brugha et al. (2011) found that ASD adults with higher educational attainment had lower rates of autism after multivariate adjustment; however, it is likely that an ASD diagnosis may have reduced the subjects' educational attainment. In contrast, in an Israeli study, where access to and coverage of ASD-related services was reported to be excellent, Davidovitch et al. (2013) found lower prevalence of ASD in children who lived in low-income versus higher-income communities, or whose families did not purchase supplemental private insurance.

Overall, many recent large-scale studies have shown an association between ASD prevalence and SES, although it appears that these differences were due to decreased access to diagnostic services among children with lower SES, or diagnostic substitution between ID and ASD among children with higher SES. In settings where health care is more accessible, these effects seem to lessen or even reverse. To date, no plausible biological mechanism has been proposed or supported that might explain SES-related differences in ASD prevalence. The fact that older studies either did not show SES associations (C. Gillberg & Schaumann, 1982; Ritvo et al., 1989; Tsai et al., 1982) or showed variability based on referral source (Wing, 1980) or

autism subtype (Sanua, 1987) also support the fact that SES differences are due to differences in ASD ascertainment as opposed to an underlying biological or psychosocial mechanism.

4.2. Race and Ethnicity

Many studies of racial/ethnic minorities show lower rates of ASD compared to White or European populations, although these differences appear to be narrowing in more current studies. The evidence is strongest for African American and Hispanic populations in the United States. Several recent studies are highlighted here, although other recent studies show similar findings (Liptak et al., 2008; Mandell et al., 2009). Since minority race and ethnic status often correlates with lower SES and worse health care access, studies attempting to assess the effects of race/ethnicity on ASD diagnosis should control for SES and health care accessibility factors in their analyses.

Using administrative data from Texas school districts, Palmer and colleagues (2010) showed that the number of autism diagnoses in a school district was inversely proportional to the number of Hispanic school children in that district, after adjusting for number of pediatricians, child psychologists, and neurologists by county, as well as county median household income. One strength of this approach is that it did attempt to adjust for SES as well as differential services availability, as well as comorbid ID and learning disabilities on a population level. Interestingly, these factors better explained variability in ASD diagnoses among White non-Hispanic children than Hispanic children, suggesting that SES and access factors alone do not explain lower diagnosis rates in Hispanics, at least on a population level. However, this ecological study did not measure individual-level access factors (e.g., insurance adequacy) or factors such as provider bias that may also impact ASD diagnostic rates.

The most recent CDC ADDM data (CDC, 2014) also suggest an overall lower rate of ASD among non-Hispanic black (123/10,000) and Hispanic children (108/10,000) compared to White children (158/10,000) in the US. Although there was considerable variability among the states, all 11 sites reported higher rates of ASD among Whites than among black and Hispanic children. However, the prevalence of ASD without intellectual disability among white children was nearly double the prevalence among either black or Hispanic children (odds ratio = 1.8, $p <$ .01), indicating that underdiagnosis of ASD in minority populations in the US may be magnified in those children without comorbid intellectual disability. Pedersen et al. (2012) examined racial/ethnic differences more thoroughly using several waves of ADDM data in Arizona, which has a large Hispanic population. That study also found a lower rate of ASD in Hispanic children compared to non-Hispanic White children. ASD prevalence increased in both populations over the study years, and the gap in prevalence between racial/ethnic groups decreased. The authors speculated that much of this difference might be attributable to underutilization and lack of access to ASD services by Hispanic families. They also speculated that these differences might reflect the "Hispanic paradox" or "healthy immigrant" effect, in which Hispanic immigrants to the United States have lower rates of multiple adverse health outcomes despite multiple SES and health-care access risk factors (Franzini, Ribble, & Keddie, 2001). However, the fact that differences in diagnostic rates are narrowing rather rapidly suggests that changes in awareness and utilization of services may be more likely than inherent genetic or developmental differences by race/ethnicity.

Windham et al. (2011) used a large administrative sample from multiple sources in Northern California, to show a lower prevalence of ASD among children of Hispanic and Black mothers compared to children of White non-Hispanic mothers, after adjusting for maternal

education and age, with similar decreases in racial differences over the study years. However, the observed racial variation was attenuated by adjustment for SES and varied significantly by data source, suggesting that variable health services utilization may have affected ASD rates.

Finally, in a U.S. population-based study using parent report of ASD diagnosis, Kogan et al. (2009) found lower rates of ASD diagnosis in non-Hispanic Black and multiracial children when compared to White children, after adjusting for parental education and income. This study also noted a disproportionately high number of Black children whose parents reported a past diagnosis of ASD that subsequently resolved, which runs contrary to most epidemiologic data about ASD lifetime trajectories. This finding suggests that low rates of ASD among Black children may be due to racial differences in parent health beliefs about ASD. This study found no significant difference in ASD diagnoses by Hispanic versus non-Hispanic ethnicity; however, follow-up analysis of the same dataset by Schieve et al. (2012) showed that there were significantly lower rates of ASD among Hispanic children with foreign-born parents compared to White children. Schieve et al. concluded that by failing to take into account the heterogeneity of Hispanic children with ASD, previous studies that grouped all Hispanics together might have been biased toward a null result. The authors felt that the findings were likely related to differences in parental awareness and access to care stemming from a lower level of acculturation for this subgroup. They also speculated that the findings might reflect the healthy immigrant effect.

In studies outside of the United States, reports about racial/ethnic differences in ASD prevalence have been more mixed, and most studies are not adjusted for SES, which makes it difficult to assess the unique effect of race/ethnicity from other confounders. In addition these studies are difficult to interpret since what constitutes a minority race or ethnicity is quite

variable by country. In Israel, Davidovitch et al. (2013) found a lower prevalence of ASD among Arab Israelis in rural settlements and in ultra-Orthodox Jews than in the general Israeli population, although prevalence was not adjusted for SES differences. Findings from a 1999–2003 census report in Stockholm, Sweden (Barnevik-Olsson, Gillberg, & Fernell, 2010) revealed that the prevalence rate of autism (autism and PDD-NOS/autistic-like condition) with learning disability was higher in Somali- versus non-Somali Swedish children. The study did not adjust for SES differences between these mothers and other Swedish mothers. The authors hypothesized that lower levels of vitamin D in immigrant Somali mothers may have affected fetal brain development and possibly led to autism and other concerning behavioral characteristics; however, the study did not measure vitamin D in any of the participants (see Kočovská, Fernell, Billstedt, Minnis, & Gillberg, 2012b). Several older, unadjusted studies also suggest a higher prevalence of ASD among recent Swedish immigrants, although these immigrants' countries of origins were so mixed that it is difficult to interpret this information in terms of ethnic or racial differences (C. Gillberg et al., 1991; C. Gillberg, Schaumann, & Gillberg, 1995; C. Gillberg, Steffenburg, Börjesson, & Andersson, 1987).

Overall, most recent studies about racial/ethnic differences in ASD diagnosis do suggest that race/ethnicity affects diagnostic rates above and beyond SES alone, at least in U.S.-based populations. However, given that the racial/ethnic effects are present in several traditionally underserved racial/ethnic groups, are quite variable by data source and study type, and have narrowed over time, they are most likely explained by differential health services utilization, parental health beliefs, and acculturation. Little high-quality data is available about the effects of race/ethnicity in non-U.S. settings.

4.3. Migration and Prenatal Exposure to Stressful Events

Migration has historically been implicated as a possible risk factor for autism, based on observed higher rates of autism among immigrant populations in some epidemiological surveys (Barnevik-Olsson et al., 2010; C. Gillberg et al., 1987; 1991; 1995; Wing, 1980). However, evidence for an association between migration and ASD has been inconsistent, with some recent studies reporting increased ASD risk among immigrant populations (Hultman, Sparén, & Cnattingius, 2002; Keen, Reid, & Arnone, 2010; Lauritsen, Pedersen, & Mortensen, 2005) and others reporting equivalent and even decreased ASD risk in some populations (Croen, Grether, & Selvin, 2002a; C. Gillberg et al., 1987; Hultman et al., 2002; Lauritsen et al., 2005). Most of the early claims about migration as a possible correlate of autism derived from post hoc observations of very small samples and were not subjected to rigorous statistical testing. However, recent studies have attempted to reexamine the association between migration and ASDs. For example, in a recent study using a population-based Swedish cohort, Magnusson et al. (2012) found that children of migrant parents were at increased risk for ASD with intellectual disability compared to children of Swedish-born parents. However, the reverse was true for ASD without intellectual disability: Children of Swedish-born parents were at significantly higher risk than children of migrant parents, particularly those from countries with low human development indices. The authors suggest that the most plausible explanation for this pattern of findings is the underdiagnosis of ASD in migrant children with high cognitive abilities; for these children, the more subtle social deficits associated with ASD may be overlooked or misattributed to language or cultural differences. In addition, because case ascertainment was based on service use, migrant families may have been less aware of or less likely to seek services in the community in the absence of clear developmental or cognitive delays. However, the researchers also suggest that

we cannot dismiss the possibility of environmental factors associated with migration and acting in utero that may contribute to ASD.

One environmental factor associated with migration that has been posited to contribute to ASD risk is prenatal exposure to stressful life events, due to the fact that migration itself is likely to be a stressful event as it may occur when families flee armed conflict or other extreme conditions in their home country (C. Magnusson et al., 2012). Using a population-based cohort of approximately 1.5 million singleton children in Denmark, J. Li et al. (2009) examined whether prenatal exposure to maternal bereavement, defined by the loss of a child, spouse, parent, or sibling during or up to 1 year prior to pregnancy, was associated with increased risk of ASD. J. Li et al. (2009) found no evidence of an effect of maternal bereavement on autism risk, even after accounting for the timing, nature, and severity of the exposure, although maternal bereavement was rare even in the total population (experienced by 2.5%). Similarly, in a recent study utilizing population-based cohorts in Sweden and England, Rai, Golding, et al. (2012a) also found no evidence for an association between ASD risk and prenatal exposure to stressful life events such as deaths, serious accidents, and diagnosis of serious illnesses in first-degree relatives, although again these events were extremely rare (experienced by 1% of the population). Thus, the hypothesis of an association between migration, as well as exposure to other prenatal stressful events, with ASD remains largely unsupported by the empirical results. However, it should be noted that even with large-scale population-based cohorts, these events were extremely rare.

4.4. Implications and Unmet Research Needs

Overall, the research findings related to low SES, minority, and immigrant populations primarily point to problems of underdiagnosis due to problems in access to health care services

and health literacy. Evidence for a biological difference based on SES, race/ethnicity, or immigration is weak, as is the case for multiple other chronic health conditions among children and adults (Pearce, Foliaki, Sporle, & Cunningham, 2004). In order to obtain an accurate depiction of ASD prevalence in underserved populations, investigators will need to specifically reach out to these populations to ensure equal participation, and also oversample these groups so that sample sizes are adequate. In addition there is a need for validated screening and diagnostic tools in multiple languages to ensure that diagnoses, when they occur, are accurate. Finally, key variables in these analyses such as parental education, income, and race/ethnicity need to be directly measured as opposed to imputed from census tract data.

5. Conclusions

Epidemiological surveys of ASDs pose substantial challenges to researchers seeking to measure rates of ASD, particularly given the range of case definition, case identification, and case evaluation methods employed across surveys. However, from recent studies, a best estimate of (69/10,000) (equivalences = 6.9/1,000 or .69% or 1 child in about 145 children) can be derived for the prevalence of ASD. Currently, the recent upward trend in rates of *prevalence* cannot be directly attributed to an increase in the *incidence* of the disorder, or to an epidemic of autism. Although power to detect time trends is seriously limited in existing datasets, there is good evidence that changes in diagnostic criteria and practices, policies for special education, service availability, and awareness of ASDs in both the lay and professional public may be responsible for increasing prevalence over time. It is also noteworthy that the rise in number of children diagnosed occurred concurrently in many countries in the 1990s, when services for children with ASD also expanded significantly. Statistical power may also be a significant

limitation in most investigations; thus, variations of small magnitude in ASD incidence may be undetected or should be interpreted with caution.

Nonetheless, the possibility that a true increase in the incidence of ASDs has also partially contributed to the upward trend in prevalence rates cannot, and should not, be completely eliminated based on available data. To assess whether the incidence has increased, methodological factors that account for an important proportion of the variability in rates must be stringently controlled for. New survey methods have been developed for use in multinational comparisons; ongoing surveillance programs are currently underway and will soon provide more meaningful data to evaluate this hypothesis. Additionally, it remains to be seen how changes to diagnostic criteria introduced in the *DSM-5* will impact ASD prevalence estimates going forward. Meanwhile, the available prevalence figures carry straightforward implications for current and future needs in services and early educational intervention programs.

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