CHAPTER 3

Epidemiology of Autism Spectrum Disorders

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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 provide a comprehensive review of the findings and methodological features of published epidemiological surveys concerned with the prevalence of autism spectrum disorders (ASDs[1]) since 1966. This chapter builds upon previous reviews (Elsabbagh et al., 2012; Fombonne, 2003a, 2003b, 2005, 2009a; Fombonne, Quirke, & Hagen, 2011; French, Bertone, Hyde, & Fombonne, 2013; J. G. Williams, Brayne, & Higgins, 2006) and includes the results of pertinent studies since published. The specific questions addressed in this chapter are as follows: (1) What is the range of prevalence estimates for autism and related ASDs? (2) How should the time trends observed in the current prevalence rates of ASDs be interpreted? and (3) What are the correlates of ASDs in epidemiological surveys?

Systematic Review Methodology

Search Strategies

Epidemiological reports included in Tables 3.1 through 3.4 in the current chapter were identified from previous reviews of epidemiological surveys

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

(Elsabbagh et al., 2012; Fombonne, 2003a, 2003b, 2005, 2009a; 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 in the tables is the most detailed and comprehensive account. For example, surveys conducted by the U.S. Centers for Disease Control (CDC) (2007a, 2007b, 2009, 2012) as part of the Autism and Developmental Disabilities Monitoring (ADDM) Network are each included in Table 3.4, although additional accounts for individual states are available elsewhere (e.g., Nicholas et al., 2008; Pinborough-Zimmerman et al., 2012; Rice et al., 2010; Zahorodny et al., 2012).

Inclusion and Exclusion Criteria

The following criteria were set a priori to select epidemiological surveys included in Tables 3.1 through 3.4:

- The full article was published in English. Several studies published in other languages (e.g., from China) are also available for consideration and have been reviewed elsewhere (for a recent review, see Elsabbagh et al., 2012).
- The minimum population was 5,000; studies involving smaller populations were excluded. Emerging evidence from smaller studies around the world is largely consistent with the findings discussed here; the interested reader is encouraged to review studies conducted in Brazil: Paula, Ribeiro, Fombonne, & Mercadante, (2011); in Sweden: Arvidsson et al. (1997), Kadesjo, Gillberg, & Hagberg, (1999), C. Gillberg, Steffenburg, Börjesson, & Andersson (1987), C. Gillberg, Schaumann, & Gillberg, (1995); in the UK: Tebruegge, Nandini, & Ritchie, (2004); and elsewhere for more information.
- The survey included independent validation of caseness by professionals. Studies that relied on questionnaire-based approaches (e.g., Ghanizadeh, 2008) or on parental report (e.g., Blumberg et al., 2013) for ASD diagnosis are not presented in tables, but are referenced in text where relevant. In addition, surveys that imposed further non-ASD criteria were excluded (e.g., presence of additional disability: N. Li, Chen, Song, Du, & Zheng, 2011; singleton births: Grether, Anderson, Croen, Smith, & Windham, 2009; Leonard et al., 2011).
- The following information categories were included or could be ascertained based on information from the survey: the country and area where the survey was conducted, the size of the population base on which the prevalence estimate was ascertained, the age range of the participants, the number of children affected, the diagnostic criteria used in case definition, and the prevalence estimate (number per 10,000). Where available, we also report the proportion of subjects with IQ within the normal range and gender ratios.

Overall, 81 studies published between 1966 and early 2013 met criteria and were selected. Of these, 54 studies provided information on rates specific to autistic disorder (AD), 18 studies on Asperger's disorder (later referred to as Asperger's syndrome; AS), and 13 studies on childhood disintegrative disorder (CDD). A total of 48 studies provided estimates on ASDs combined, of which 24 also provided rates for specific ASD subtypes (14 provided rates for both AD and AS; 10 provided rates for AD but not AS). Surveys were conducted in 23 different countries (including 17 in the United Kingdom, 16 in the United States, and 7 in Japan). The results of over half of the studies ($n = 55$) were published after 2000, with most studies relying on school-aged samples. Finally, a very large variation in the size of the population surveyed was evidenced (range: 5,007 to 4.3 million; median: 50,210; mean: 275,300), with some recent studies conducted by the CDC relying on samples of several hundreds of thousands of individuals.

Study Design and Methodological Issues

Epidemiology is concerned with the study of the repartition of diseases in human population

and of the factors that influence it. Epidemiologists use several measures of disease occurrence. Incidence rate refers to the number of new cases (numerator) of a disease occurring over a specified period in those at risk of developing the disease in the population (denominator, in person × years). Cumulative incidence is the proportion of those who were free of the disease at the beginning of the observation period and developed the disease during that period. Measures of incidence are required to properly estimate several variables such as morbidity due to a disease, possible changes over time, and the risk factors underlying disease status. 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, at that point in time, suffer from the disease. To date, most epidemiological studies of ASDs have been cross-sectional, reflecting the complications involved in conducting a survey when the timing of diagnosis lags behind onset of symptoms and is likely to be influenced by a range of factors potentially unrelated to risk (discussed further in "Correlates of ASDs"). As a result, the most commonly reported measures of ASD population frequency have been prevalence rates (point prevalence or period prevalence), with a few recent exceptions (e.g., Campbell, Reynolds, Cunningham, Minnis, & Gillberg, 2011; Hertz-Picciotto & Delwiche, 2009; Manning et al., 2011; van der Ven et al., 2012). In designing a prevalence study, three major features are critical for the planning and logistics of the study as well as for the interpretation of its results: case definition, case identification (or case ascertainment), and case evaluation methods (Fombonne, 2007).

Case Definition

Over time, the definition of autism has changed, as illustrated by the numerous diagnostic criteria that were used in both epidemiological and clinical settings (see Table 3.1). Starting with the narrowly defined Kanner's autism (1943), definitions progressively broadened in the criteria from that proposed by Rutter (1970), and subsequent International Classification of Diseases, ninth revision (ICD-9; World Health Organization [WHO], 1977); *Diagnostic and Statistical Manual of Mental Disorders*, third edition (*DSM*-III; American Psychiatric Association [APA], 1980); and *DSM-III-R* (APA, 1987), and more recently in the two more major nosographies used worldwide; ICD-10 (WHO, 1992) and *DSM-IV* (APA, 1994). The earliest diagnostic criteria reflected the more qualitatively severe forms of autism's behavioral phenotype, usually associated with severe delays in language and cognitive skills. It was only in the 1980s that 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 (Pervasive Developmental Disorders Not Otherwise Specified [PDD-NOS] or Autism Spectrum Disorders—[ASD]) within a broader class of autism spectrum disorders denominated "pervasive developmental disorders" (PDDs, an equivalent to ASDs) in current nosographies. Although Asperger had described it in the literature as early as 1944, Asperger's disorder only appeared in official nosographies in the 1990s, with unclear validity, particularly with respect to its differentiation from high-functioning autism. Other ASD subtypes that were described in *DSM-III* subsequently disappeared (e.g., Autism–Residual State).

While there is generally high interrater reliability regarding diagnosis of ASDs and commonality of concepts across experts, some differences still persist between nomenclatures about the terminology and operationalized criteria of ASDs. For example, *DSM-IV* (APA, 1994) has a broad category of PDD-NOS, sometimes referred to loosely as "atypical autism," whereas ICD-10 (WHO, 1992) has several corresponding diagnoses for clinical presentations that do not allow an autistic disorder diagnosis and include Atypical Autism (F84.1, a diagnostic category that existed already in ICD-9), Other PDD (F84.8), and PDD—Unspecified (F84.9). As a result, studies that refer to "atypical autism" must be carefully interpreted, and equivalence with the *DSM-IV* concept of PDD-NOS should not be assumed. As no diagnostic criteria are available for these milder forms of the autism

phenotype, the resulting boundaries of the spectrum of ASDs are left uncertain. Whether or not this plays a role in more recent epidemiological studies is difficult to ascertain, but the possibility should be considered in assessing results for subsequent epidemiological surveys.

Case Identification/Ascertainment

When an area or population has been identified for a survey, different strategies have been employed to find individuals matching the case definition retained for the study. Some studies have relied solely on existing service providers databases (Chien, Lin, Chou, & Chou, 2011; Croen, Grether, Hoogstrate, & Selvin, 2002a; Davidovitch, Hemo, Manning-Courtney, & Fombonne, 2012), on special educational databases (Fombonne, Zakarian, Bennett, Meng, & McLean-Heywood, 2006; Gurney et al., 2003; Lazoff, Zhong, Piperni, & Fombonne, 2010; Maenner & Durkin, 2010), or on 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 to the service provider or agencies, rather than sampling from the population at large. As a result, individuals with the disorder who are not in contact with these services are not included as cases, leading to an underestimation of the prevalence proportion. This is a particularly important issue when estimating prevalence using such methods 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, 2012; Idring et al., 2012; Kim et al., 2011). The aim of the first screening stage of these studies is to cast a wide net in order to identify subjects possibly affected with an ASD, with the final diagnostic status being determined at subsequent stages. This process often consists of sending letters or brief screening scales requesting school and health professionals and/or other data sources to identify possible cases of autism. Few of these investigations rely on systematic sampling techniques that would ensure a near complete coverage of the target population. Moreover, such investigations differ in several key aspects with regard to the initial screening stage. First, the thoroughness of the coverage of all relevant data sources vary enormously from one study to another. In addition, the surveyed areas are not comparable in terms of service development, reflecting the specific educational or health care systems of each country and of the period of investigation. Second, the inclusion information sent out to professionals invited to identify children varies from a few clinical descriptors of autism-related symptoms or diagnostic checklists rephrased in nontechnical terms, to more systematic screening strategies based on questionnaires or rating scales of known reliability and validity. Third, uneven participation rates in the first screening stages provide another source of variation in the screening efficiency of surveys, although refusal rates tend, on average, to be very low.

To illustrate the effects of differential participation rates in the first screening stage, two hypothetical scenarios are illustrated in Figure 3.1, 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 a 60% participation rate for ASD and non-ASD cases in the first screening stage, resulting in a total of 90 participating ASD cases that screen positive. With a 70% participation rate for both ASD and non-ASD cases in the final diagnostic stage, we would identify and confirm a total of 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 equally assume an average 60% participation rate, but with a higher 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 average participation rates in the first screening (60%) and the final diagnostic stages (70%), we identify and confirm a total of 84 ASD cases and calculate a biased prevalence estimate of 2% (200/10,000), an estimate that is 0.5% higher

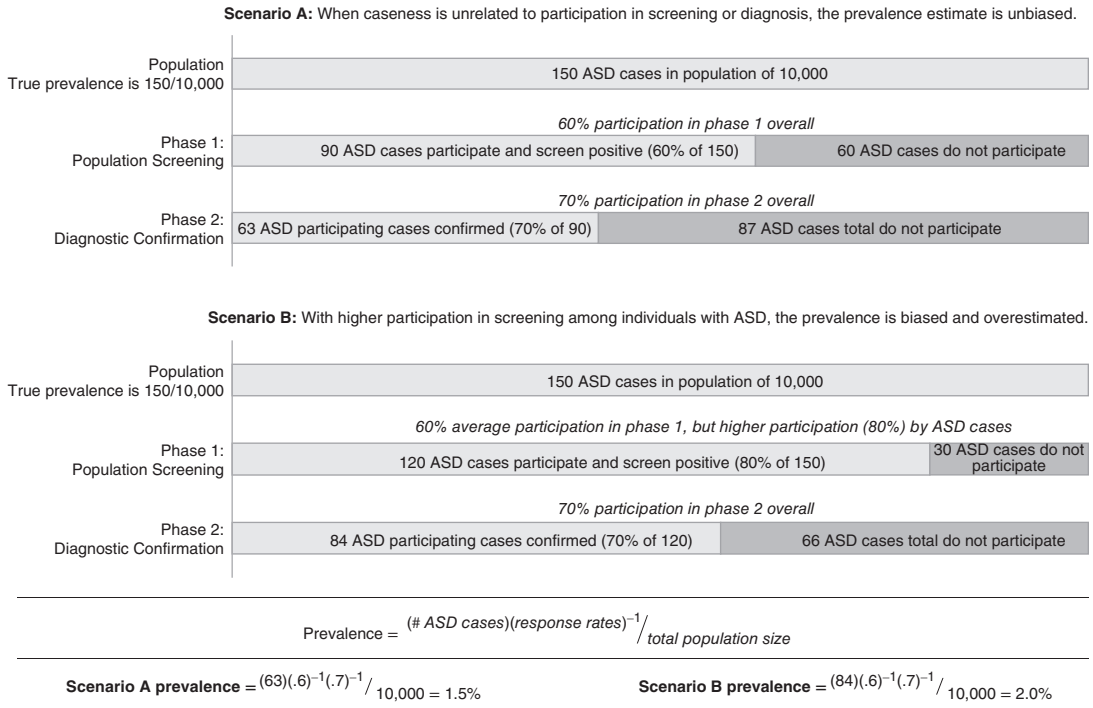

Figure 3.1 Impact of differential participation rates in screening on ASD prevalence estimates: Two hypothetical scenarios.

than the 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 in the estimate.

Another possible scenario (not illustrated) is one in which individuals with ASD are less likely to participate than noncases, leading to underestimation of prevalence. For example, Posserud, Lundervold, Lie, & Gillberg (2010) reported an 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. On the other hand, Webb et al. (2003) reported increased refusal rates among individuals with fewer 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 estimates of prevalence.

The sensitivity of the screening methodology is also difficult to gauge in autism surveys, as the proportion of children truly affected with the disorder but not identified in the screening stage (the 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 screened negative subjects in order to estimate the proportion of false negatives and adjusting the estimate accordingly, has not been used in these surveys. The main reason is that due to the relatively low frequency of the disorder, it would be both imprecise and very costly to undertake such estimations. This may gradually change in view of recent prevalence studies suggesting that autism can no longer be regarded as a rare condition. However, prevalence estimates must be understood as underestimates of

"true" prevalence rates, with the magnitude of this underestimation unknown in each survey.

To provide a concrete illustration of this, the surveys conducted by the CDC in the United States (2007a, 2007b, 2009, 2012) 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.

Case Evaluation

When the screening phase is completed, subjects identified as positive go through a more in-depth diagnostic evaluation to confirm their case status. Similar considerations about the methodological variability across studies apply to these more intensive assessment phases. In the studies reviewed, participation rates in second-stage assessments were generally high (over 80%). The source of information used to determine diagnosis usually involves a combination of data from different informants (parents, teachers, pediatricians, other health professionals, etc.) and data sources (medical records, educational sources), with an in-person assessment of the person with autism being offered in some but not all studies. Obviously, surveys of very large populations, such as those conducted in the United States by the CDC ADDM Network (e.g., 2012) or in national registers (e.g., Idring et al., 2012), did not include a direct diagnostic assessment of all subjects by the research team. However, these investigators could generally confirm the accuracy of their final caseness determination 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 that relies on screening of the population using multiple data sources, a standardized procedure for abstracting records, and a systematic review and scoring system for the data gathered in the screening phase. In the less obvious cases, this information is then 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. Several recent studies have adopted the evaluation approach for population-based autism surveillance developed by the CDC, highlighting the utility of these methods for facilitating multi-source active surveillance in the United States (Windham et al., 2011) and for establishing the validity of registry-based ASD diagnoses in Denmark (94% of registered cases confirmed; Lauritsen et al., 2010).

When subjects were directly examined, the assessments were conducted with 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 (Le Couteur et al., 1989) and/or the Autism Diagnostic Observation Schedule (Lord et al., 2000) have been increasingly used in the most recent surveys (e.g., Isaksen, Diseth, Schjolberg, & Skjeldal, 2012; Kim et al., 2011; Mattila et al., 2011).

Keeping in mind the range and limitations of case definition, identification, and evaluation methods employed in the studies we report, we now turn to the available evidence from epidemiological surveys.

PREVALENCE ESTIMATES

Autistic Disorder

Prevalence estimates for autistic disorder are summarized in Table 3.1. There were 54 studies (including 12 in the United Kingdom, 8 in the United States, and 6 in Japan), with over half of them published since 2001. The sample size varied from 5,007 to 4.95 million, with a median

TABLE 3.1 Prevalence Surveys of Autistic Disorder (AD)

Year	Authors	Country	Area	Population	Age	Number Affected	Diagnostic Criteria	% With Normal IQ	Gender Ratio (M:F)	Prevalence Rate/10,000	95% CI
1966	Lotter	UK	Middlesex	78,000	8–10	32	Rating scale	15.6	2.6 (23:9)	4.1	2.7; 5.5
1970	Brask	Denmark	Aarhus County	46,500	2–14	20	Clinical	—	1.4 (12:7)	4.3	2.4; 6.2
1970	Treffert	United States	Wisconsin	899,750	3–12	69	Kanner	—	3.06 (52:17)	0.7	0.6; 0.9
1976	Wing et al.	UK	Camberwell	25,000	5–14	17[a]	24-item rating scale of Lotter	30	16 (16:1)	4.8[b]	2.1; 7.5
1982	Hoshino et al.	Japan	Fukushima-Ken	609,848	0–18	142	Kanner's criteria	—	9.9 (129:13)	2.33	1.9; 2.7
1983	Bohman et al.	Sweden	County of Västerbotten	69,000	0–20	39	Rutter criteria	20.5	1.6 (24:15)	5.6	3.9; 7.4
1984	McCarthy et al.	Ireland	East	65,000	8–10	28	Kanner	—	1.33 (16:12)	4.3	2.7; 5.9
1986	Steinhausen et al.	Germany	West Berlin	279,616	0–14	52	Rutter	55.8	2.25 (36:16)	1.9	1.4; 2.4
1987	Burd et al.	United States	North Dakota	180,986	2–18	59	*DSM-III*	—	2.7 (43:16)	3.26	2.4; 4.1
1987	Matsuishi et al.	Japan	Kurume City	32,834	4–12	51	*DSM-III*	—	4.7 (42:9)	15.5	11.3; 19.8
1988	Bryson et al.	Canada	Part of Nova Scotia	20,800	6–14	21	New RDC	23.8	2.5 (15:6)	10.1	5.8; 14.4
1988	Tanoue et al.	Japan	Southern Ibaraki	95,394	7	132	*DSM-III*	—	4.07 (106:26)	13.8	11.5; 16.2
1989	Cialdella and Mamelle	France	Rhône	135,180	3–9	61	*DSM-III* like	—	2.3	4.5	3.4; 5.6
1989	Ritvo et al.	United States	Utah	769,620	3–27	241	*DSM-III*	34	3.73 (190:51)	2.47	2.1; 2.8
1989	Sugiyama and Abe	Japan	Nagoya	12,263	3	16	*DSM-III*	—	—	13.0	6.7; 19.4
1991	Gillberg et al.	Sweden	South-West Göteborg, Bohuslän	78,106[c]	4–13	74	*DSM-III-R*	18	2.7 (54:20)	9.5	7.3; 11.6
1992	Fombonne and du Mazaubrun	France	4 régions 14 départements	274,816	9 and 13	154	Clinical/ICD-10 like	13.3	2.1 (105:49)	4.9	4.1; 5.7
1992	Wignyosumarto et al.	Indonesia	Yogyakarita (SE of Jakarta)	5,120	4–7	6	CARS	0	2.0 (4:2)	11.7	2.3; 21.1

(*continued*)

TABLE 3.1 (*Continued*)

Year	Authors	Country	Area	Population	Age	Number Affected	Diagnostic Criteria	% With Normal IQ	Gender Ratio (M:F)	Prevalence Rate/10,000	95% CI
1996	Honda et al.	Japan	Yokohama	8,537	5	18	ICD-10	50.0	2.6 (13.5)	21.08	11.4; 30.8
1997	Fombonne et al.	France	3 départements	325,347	8–16	174	Clinical ICD-10 like	12.1	1.81 (112:62)	5.35	4.6; 6.1
1997	Webb et al.	UK	South Glamorgan, Wales	73,301	3–15	53	*DSM-III-R*	—	6.57 (46:7)	7.2	5.3; 9.3
1998	Sponheim and Skjeldal	Norway	Akershus County	65,688	3–14	25	ICD-10	36.0[d]	2.10 (17:8)	3.8	1.9; 5.1
1999	Taylor et al.	UK	North Thames	490,805[*]	0–16	427	ICD-10	—	—	8.7	7.9; 9.6
2000	Baird et al.	UK	South-East Thames	16,235	7	45[e]	ICD-10	60	14 (42:3)	27.7[e]	20.7; 37.1[*]
2000	Kielinen et al.	Finland	North (Oulu et Lapland)	152,732	3–18	187[*]	ICD-10, *DSM-IV*	50.2	—	12.2	10.6; 14.1[*]
2000	Powell et al.	UK	West Midlands	58,974[*]	1–5	46	Clinical, ICD-10, *DSM-IV*	—	—	7.8	5.8; 10.5
2001	Bertrand et al.	United States	Brick Township, New Jersey	8,896	3–10	36	*DSM-IV*	36.7	2.2 (25:11)	40.5	28.0; 56.0
2001	Chakrabarti and Fombonne	UK (Midlands)	Staffordshire	15,500	2.5–6.5	26	ICD-10, *DSM-IV*	29.2	3.3 (20:6)	16.8	10.3; 23.2
2001	Davidovitch et al.	Israel	Haiffa	26,160	7–11	26	*DSM-III-R*, *DSM-IV*	—	4.2 (21:5)	10.0	6.6; 14.4
2001	Magnússon and Saemundsen	Iceland	Whole Island	43,153	5–14	57	ICD-9, ICD-10	13.5	4.2	13.2	9.8; 16.6
2002	Croen, Grether, Hoogstrate, and Selvin	United States	Northern California (DDS: 1987–1994)	4,950,333	5–12	5, 038	"Full syndrome autism"—CA Dept. of Developmental Services	62.8[f]	4.47 (4116:921)	11.0	10.7; 11.3
2003	Lingam et al.	UK	North East London	186,206	5–14	278	ICD-10	—	5.8 (278:48)	14.9	13.3; 16.8[*]
2005	Barbaresi et al.	United States	Olmstead County, Minnesota	37,726	0–21	112	*DSM-IV*	—	—	29.7	24.6; 35.7[*]
2005	Chakrabarti and Fombonne	UK (Midlands)	Staffordshire	10,903	4–6	24	ICD-10, *DSM-IV*	33.3	3.8 (19:5)	22.0	14.4; 32.2
2005	Honda et al.[g]	Japan	Yokohama	32,791	5	123	ICD-10	25.3	2.5 (70:27)	37.5	31.0; 45.0

2006	Baird et al.	UK	South Thames, London	56,946	9–10	81	ICD-10	47	8.3 (~72:9)	38.9	29.9; 47.8
2006	Fombonne et al.	Canada	Montreal Island, Quebec	27,749	5–17	60	*DSM-IV*	—	5.7 (51:9)	21.6	16.5; 27.8
2006	C. Gillberg et al.	Sweden	Göteborg	32,568	7–12	115	*DSM-III*, *DSM-IV*, Gillberg's criteria for AS	—	3.6 (90:25)	35.3	29.2; 42.2
2007	Croen et al.	United States	Northern California (1995–1999)	132,844	5-10	277	ICD-09-CM	—	4.1	20.9	18.5; 23.5*
2007	Latif and Williams	UK	Wales	39,220	0–17	50	Kanner	—	—	12.7	9.0; 17.0
2007	Oliveira et al.	Portugal	Mainland and Azores	67,795	6–9	115	*DSM-IV*	17	2.9	16.7	14.0; 20.0
2008	Montiel-Nava and Pena	Venezuela	Maracaibo	254,905	3–9	287	*DSM-IV-TR*	—	4.1 (231:56)	11	10; 14
2008	E. Williams et al.	UK	South West (Avon)	14,062	11	30	ICD-10	86.7	5.0 (25:5)	21.6	13.9; 29.3
2009	van Balkom et al.	Netherlands	Aruba (Caribbean)	13,109	0-13	25	*DSM-IV*	36.0	7.3 (22:3)	19.1	12.3; 28.1
2010	Fernell and Gillberg	Sweden	Stockholm	23,566	6	75	*DSM-IV*, *DSM-IV-TR*, ICD-10	11	6.5	32	26; 37
2010	Lazoff et al.	Canada	Montreal	23,635	5–17	60	*DSM-IV*	—	5.8(58:10)	25.4	19.0; 31.8
2011	Kim et al.	S. Korea	Goyang City	55,226	7–12	27	*DSM-IV*	55.6	4.4	94	56; 134
2011	Mattila et al.	Finland	Northern Ostrobothnia County	5,484	8	18	*DSM-IV-TR* including ADOS and ADI	61	2	41	26; 64
2011	Parner et al.[h]	Australia	Western Australia (1994–1999)	152,060	0–10	516	*DSM-IV*, *DSM-IV-TR*	—	4.4	39.3	—
2011	Windham et al.[i]	United States	San Francisco Bay Area (DDS: 1994, 1996)	162,402	0–8	477	"Full syndrome autism"—CA Dept. of Developmental Services	—	6.4 (493:77)	29	26.9; 32.1*
2012	Isaksen et al.	Norway	Oppland and Hedmark	31,015	6–12	42	ICD-10 including ADOS and ADI	—	3.2	14	10; 18

(*continued*)

TABLE 3.1 (*Continued*)

Year	Authors	Country	Area	Population	Age	Number Affected	Diagnostic Criteria	% With Normal IQ	Gender Ratio (M:F)	Prevalence Rate/10,000	95% CI
2012	Kočovská, Biskupsto et al.[j]	Denmark	Faroe Islands	7,128	15–24	15	*DSM-IV*/ICD-10	—	2 (10:5)	21	12; 35
2012	Nygren et al.	Sweden	Göteborg	5,007	2	26	*DSM-IV-TR*	—	5.5	52	35.5; 76.0*
2012	Parner et al.[k]	Denmark	National Register (1980–2003)	1,311,736	6–29	2,446	ICD-8, ICD-9, ICD-10	—	4.4	18.65*	17.9; 19.4*

*Calculated by the authors.

[a]This number corresponds to the sample described in Wing and Gould (1979).

[b]This rate corresponds to the first published paper on this survey and is based on 12 subjects among children aged 5 to 14 years.

[c]For the Göteborg surveys by Gillberg et al. (Gillberg, 1984; C. Gillberg et al., 1991; Steffenburg & Gillberg, 1986), a detailed examination showed that there was overlap between the samples included in the three surveys; consequently only the 1991 survey has been included in this table.

[d]In this study, mild mental retardation was combined with normal IQ (approximately IQ > 50), whereas moderate and severe mental retardation were grouped together (IQ < 50).

[e]This prevalence was calculated by the authors after removing the $n = 5$ cases that also met criteria for Asperger's.

[f]This proportion is likely to be overestimated and to reflect an underreporting of mental retardation to the California Department of Developmental Services.

[g]This figure was calculated by the author and refers to prevalence data (not cumulative incidence) presented in the paper (the M:F ratio is based on a subsample).

[h]Note that this is an updated prevalence estimate: previous estimates have been reported by Nassar et al. (2009; birth years: 1983–1999; prevalence: 20.8/10,000;) and Leonard et al. (2011; birth years: 1984–1999; singletons; prevalence: 21/10,000) using the same register in Western Australia.

[i]Note that there is partial overlap between this sample and that reported by Croen, Grether, Hoogstrate, et al. (2002); because of the minimal overlap between these two sample populations (birth year: 1994), both prevalence estimates are included in this table.

[j]Note that this is an updated prevalence estimate: a previous estimate of 16/10,000 was reported by Ellefsen et al. (2007) based on the same geographical area with the same cohort.

[k]Note that this is an updated prevalence estimate: previous estimates have been reported by Lauritsen et al. (2004; birth years: 1971–2000; prevalence: 11.8/10,000) and by Parner et al. (2011; birth years: 1994–1999; prevalence: 21.8/10,000) using the same national register in Denmark.

of 56,090 (mean: 233,300) subjects in the surveyed populations. Age ranged from 0 to 29 years, with a median age of 8.5 years (mean: 8.4 years). The number of subjects identified with autistic disorder ranged from 6 to 5,038 (median: 55; mean: 234). Males consistently outnumbered females in the 48 studies where gender differences were reported, with a male/female ratio ranging from 1.3:1 to 16:1, leading to an average male/female ratio of 4.3:1.

There was a 134-fold variation in prevalence estimates for autistic disorder, with rates ranging from 0.7 to 94 per 10,000 (median: 13.5; mean: 18). Prevalence rates were negatively correlated with sample size (Spearman's r: -0.55; $p < .0001$), with small-scale studies reporting higher prevalence rates. There was a significant positive correlation between prevalence rate and publication year (Spearman's r: .78; $p < .0001$), with higher rates in more recent surveys. Therefore, a current estimate for the prevalence of autistic disorder must be derived from more recent surveys with an adequate sample size. In 31 studies published since 2000, the mean prevalence was 26.1/10,000 (median: 21.6/10,000). After exclusion of the two studies with the smallest and largest sample sizes, the results were very similar (mean: 25.7/10,000; median: 21.6/10,000). Thus, the best current estimate for the prevalence of autistic disorder is 26/10,000.

Of the 54 studies, 27 reported the proportion of subjects with IQ within the normal range (median: 33.3%; interquartile range: 17.5–50.1%). Over time, there were minor associations between the year of publication of the survey and the sample male/female ratio (Spearman's r: 0.31; $p = .03$) and the proportion of subjects without mental retardation (Spearman's r: 0.32; $p = .1$). Taken in conjunction with the much stronger increase over time in prevalence rates, these results suggest that the increase in prevalence rates is not entirely accounted for by the inclusion of milder forms (i.e., less cognitively impaired) of autistic disorder, albeit this might have contributed to it to some degree.

Asperger's Syndrome

Epidemiological studies of Asperger's syndrome are sparse, due to the fact that it was acknowledged as a separate diagnostic category in both ICD-10 and *DSM-IV* only in the early 1990s. Three epidemiological surveys (not featured in the current analysis due to relatively small population sizes) *specifically* investigated AS prevalence (Ehlers & Gillberg, 1993; Kadesjo et al., 1999, Mattila et al., 2007). However, only a handful of cases were identified in these surveys, with the resulting estimates varying greatly. In addition, it remains unclear if these subjects would have also met criteria for autistic disorder and how prevalence rates would be affected if hierarchical rules were followed to diagnose both disorders. For example, Mattila et al. (2007) reported that 4 out of 10 children previously diagnosed with AS were reassigned to a diagnosis of high-functioning autism following *DSM-IV*/ICD-10 criteria. One survey of high-functioning ASDs in Welsh mainstream primary schools yielded a relatively high (uncorrected) prevalence estimate of 14.5/10,000, but no rate was available specifically for AS (Webb et al., 2003).

Other recent surveys have examined samples with respect to the presence of both autistic disorder and Asperger's syndrome. Eighteen studies published since 1998 provide usable data (Table 3.2). The median population size was 25,690, and the median age 8.3 years. Numbers of children identified with AS varied from 2 to 419 (median: 26; mean: 59). There was a 173-fold variation in estimated prevalence of AS (range: 0.3 to 52/10,000; median: 7.2/10,000; mean: 12.3/10,000). For the majority of studies (15 out the 18 total), the number of children with autistic disorder was higher than that of children with AS. In these studies, the ratio of children with AD to those with AS exceeded 1 (median: 3.0; mean: 5.8), indicating that the rate of AS was consistently *lower* than that for autism (Table 3.2). Unusually high rates of AS relative to autistic disorder were obtained in three studies with

TABLE 3.2 Asperger's Syndrome (AS) in Recent Autism Surveys

				Assessment			Autistic Disorder		Asperger Syndrome		
Year	Authors	Population	Age	Informants	Instruments	Diagnostic Criteria	N	Prevalence Rate/10,000	N	Prevalence Rate/10,000	AD/AS Ratio
1998	Sponheim and Skjeldal	65,688	3–14	Parent Child	Parental Interview + direct observation, CARS, ABC	ICD-10	25	3.8	2	0.3	12.5
1999	Taylor et al.	490,000	0–16	Record	Rating of all data available in child record	ICD-10	427	8.7	71	1.4	6.0
2000	Baird et al.	16,235	7	Parents Child Other data	ADI-R Psychometry	ICD-10 *DSM-IV*	45	27.7	5	3.1	9.0
2001	Chakrabarti and Fombonne	15,500	2.5–6.5	Child Parent Professional	ADI-R, 2 weeks multidisciplinary assessment, Merrill-Palmer, WPPSI	ICD-10, *DSM-IV*	26	16.8	13	8.4	2.0
2003	Lingam et al.	186,206	5–14	Disability Register	Review of medical records	ICD-10	278	14.9	94	5.0	3.0
2004	Lauritsen et al.[a]	643,220*	1–10	National Registry	Available data	ICD-8, ICD-10	759	11.8	419	4.7	1.8
2005	Chakrabarti and Fombonne	10,903	4–6	Child Parent Professional	ADI-R, 2 weeks multidisciplinary assessment, Merrill-Palmer, WPPSI	ICD-10, *DSM-IV*	24	22.0	12	11.0	2.0
2006	Fombonne et al.	27,749	5–17	School registry	Clinical	*DSM-IV*	60	21.6	28	10.1	2.1
2007	Latif and Williams	39,220	0–17	?	Clinical	Kanner, Gillberg AS criteria	50	12.7	139	35.4	0.36
2008	Montiel-Nava and Pena	254,905	3–9	Child Parent Professional	School and/or medical records, CARS, ADOS	*DSM-IV-TR*	287	11.0	39	1.52[b]	7.4

2008	E. Williams et al.	14,062	11	Medical records and educational registry	Clinical	ICD-10	30	21.6	23	16.6	1.3
2009	Nassar et al.[c]	419,917	0–21	Record	Available data (1983–1999)	*DSM-III, DSM-IV, DSM-IV-TR*	700	20.8	40	1.0	17.5
2009	van Balkom et al.	13,109	0–13	Clinic series	Review of medical records	*DSM-IV*	25	19.1	2	1.5	12.5
2010	Fernell and Gillberg	23,566	6	Parent	Clinical	*DSM-IV, DSM-IV-TR*, ICD-10	75	32	14	6	5.4
2010	Lazoff et al.	23,635	5–17	School registry	Review of educational records	*DSM-IV*	60	25.4	23	9.7	2.6
2011	Mattila et al.	5,484	8	Parent Child Professional	Clinical, ADOS-G, ADI-R	*DSM-IV-TR*	18	41	11	25	1.64
2012	Isaksen et al.	31,015	6–12	Parent Child Professional	Clinical, ADOS-G, ADI-R	ICD-10	42	14	89	28	0.47
2012	Kočovská, Biskupsto, et al.[d]	7,128	15–24	Parent Child Professional	DISCO, WISC-R, ASSQ	ICD-10 Gillberg AS criteria	15	21	37	52	0.41

[*]Calculated by the authors.

[a]Note that a subsequent survey by Parner et al. (2012) using an overlapping sample provided updated prevalence estimates for AD and ASD but not for AS; those estimates are included in Tables 3.1 and 3.4 instead of Lauritsen et al. (2004).

[b]The authors note that this is likely an underestimation due to the case ascertainment methods employed.

[c]Note that a subsequent survey by Parner et al. (2011) using an overlapping sample provided updated prevalence estimates for AD and ASD but not for AS; those estimates are included in Tables 3.1 and 3.4 instead of Nassar et al. (2009).

[d]Note that this is an updated prevalence estimate; Ellefsen et al. (2007) initially reported prevalence for AS of 16/10,000 based on a survey of the same geographical area with the same cohort.

a median and mean AD/AS ratio of 0.4 (Isaksen et al., 2012; Kočovská, Biskupsto, et al., 2012a; Latif & Williams, 2007). Isaksen et al. (2012) noted that the increased rates might reflect the catch-all status of AS as a diagnostic category, or that some clinicians may lack the expertise required to differentiate AS from other ASD subtypes. In the Faroe Islands survey, Kočovská, Biskupsto, et al. (2012a) followed up with the same cohort from an earlier 2002 population survey (Ellefsen, Kampmann, Billstedt, Gillberg, & Gillberg, 2007). In addition to identifying 23 newly diagnosed cases, the authors noted diagnostic stability in those previously identified with ASDs overall, yet considerable variability in the stability of diagnostic subtypes (previous AS prevalence estimate: 26/10,000; updated: 52/10,000). In Latif and Williams (2007), AS prevalence estimates appeared to be inflated due to the inclusion of high-functioning autism in the AS definition. The epidemiological data on AS are therefore of dubious quality, reflecting uncertainties around inclusion of AS in recent nosographies as well as the lack of proper measurement strategies that can ensure a reliable difference between AS and autistic disorder.

Childhood Disintegrative Disorder

Thirteen surveys provided data on childhood disintegrative disorder (Table 3.3). In 5 of these, only one case was reported; no case of CDD was identified in 5 other studies. Prevalence estimates ranged from 0 to 9.2/100,000, with a median rate of 1.5/100,000. The pooled estimate, based on 11 identified cases and a surveyed population of about 570,000 children, was 1.9/100,000. Gender was reported in 10 of the 11 studies, and males appear to be overrepresented with a male/female ratio of 9:1. The upper limit of the confidence interval associated to the pooled prevalence estimate (3.5/100,000) indicates that CDD is a rare condition, with about 1 case occurring for every 189 cases of autistic disorder.

Prevalence for Combined ASDs

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, before reviewing the findings of these studies conducted since 2000, we first examine how findings of the first generation of epidemiological surveys of a narrow definition of autism also informed our understanding of the modern concept of autism spectrum disorders.

Unspecified ASDs in Earlier Surveys

In previous reviews, we documented that several studies performed in the 1960s and 1970s had 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, & Kerbeshan, 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,

TABLE 3.3 Prevalence Surveys of Childhood Disintegrative Disorder (CDD)

Year	Authors	Country	Area	Population	Age	Number affected	Diagnostic Assessment	Gender ratio (M:F)	Prevalence Rate/100,000	95% CI
1987	Burd et al.	United States	North Dakota	180,986	2–18	2	Structured parental interview and review of all data available—*DSM-III* criteria	2:—	1.11	0.13; 3.4
1998	Sponheim and Skjeldal	Norway	Akershus County	65,688	3–14	1	Parental interview and direct observation (CARS, ABC)	—	1.52	0.04; 8.5
2001	Chakrabarti and Fombonne	UK (Midlands)	Staffordshire	15,500	2.5–6.5	1	ADI-R, 2 weeks multidisciplinary assessment, Merrill-Palmer, WPPSI–ICD-10/*DSM-IV*	1:—	6.45	0.16; 35.9
2001	Fombonne et al.	UK	England and Wales	10,438	5–15	0	Parental interview and direct observation, *DSM-IV*, ICD-10	—	0	—
2001	Magnússon and Saemundsen	Iceland	Whole Island	85,556	5–14	2	ADI-R, CARS, and psychological tests—mostly ICD-10	2:—	2.34	0.3; 8.4
2005	Chakrabarti and Fombonne	UK (Midlands)	Staffordshire	10,903	4–6	1	ADI-R, 2 weeks multidisciplinary assessment, Merrill-Palmer, WPPSI–ICD-10/*DSM-IV*	1:—	9.17	0; 58.6
2006	Fombonne et al.	Canada	Montreal Island, Quebec	27,749	5–17	1	*DSM-IV*, special needs school survey	1:—	3.6	0; 20
2006	C. Gillberg et al.	Sweden	Göteborg	102,485	7–24	2	*DSM-IV*, review of medical records of local diagnostic center	1:1	2	0.5; 7.1
2007	Ellefsen et al.	Denmark	Faroe Islands	7,689	8–17	0	DISCO, Vineland, WISC-R, ICD-10/*DSM-IV*	—	0	—
2008	Kawamura et al.	Japan	Toyota	12,589	5–8	0	*DSM-IV*, population based screening at 18 and 36 months	—	0	—
2008	E. Williams et al.	UK	South West (Avon)	14,062	11	0	ICD-10, educational and medical record review	—	0	—
2009	van Balkom et al.	Netherlands	Aruba (Caribbean)	13,109	0–13	0	Clinic medical record review	—	0	—
2010	Lazoff et al.	Canada	Montreal	23,635	5–17	1	*DSM-IV*, special needs school survey	1:0	4.23	0.7; 24
	POOLED ESTIMATES			**570,389**		**11**		**9:1**	**1.9**	**1.1; 3.5**

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 (see later discussion) that now use case definitions that incorporate a priori these milder phenotypes.

Newer Surveys of ASDs

The results of surveys that estimated the prevalence of the whole spectrum of ASDs are summarized in Table 3.4. Of the 48 studies listed, 24 also provided separate estimates for autistic disorder and other ASD subtypes; the other 24 studies provided only an estimate for the combined ASD rate. All selected surveys were published since 2000, with the majority (58%) published since 2008. The studies were performed in 17 different countries (including 13 in the United Kingdom and 11 in the United States, of which 4 were conducted by the CDC). Sample sizes ranged from 5,007 to 4.3 million (median: 56,110; mean: 273,200).

Ages of the surveyed populations ranged from 0 to 98 (median: 8; mean: 9). One recent 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 recent surveys specifically focused on toddlers (Nygren et al., 2012) and preschoolers (Nicholas, Carpenter, King, Jenner, & Charles, 2009) provided estimates of approximately 80 per 10,000. In the 45 remaining surveys, the average median and modal age was 8 years (mean: 8.2).

The diagnostic criteria used in 42 studies reflected the reliance on modern diagnostic schemes (10 studies used ICD-10, 24 the *DSM-III*, *DSM-IV*, or *DSM-IV-TR*; both schemes being used simultaneously in 8 studies). Assessments were often performed with standardized diagnostic measures (i.e., Autism Diagnostic Interview—Revised [ADI-R] and Autism Diagnostic Observation Schedule [ADOS]) that match well the more dimensional approach retained for case definition. In 24 studies where IQ data were reported, the proportion of subjects within the normal IQ range varied from 0% to 100% (median: 55.4%; mean: 53.2%), a proportion that is higher than that for autistic disorder and reflects the lesser degree of association, or lack thereof, between intellectual impairment and milder forms of ASDs. Overrepresentation of males was the rule in the 42 studies reporting gender ratios, with male/female ratio ranging from 1.8:1 to 15.7:1 (median: 4.3:1; mean: 4.9:1).

Overall, the number of individuals affected by ASD ranged from 16 to 9,556 across studies (median: 215; mean: 948). There was a 189-fold variation in prevalence that ranged from a low of 1.4/10,000 to a high of 264/10,000 (see Figure 3.2). There was also substantial variation in confidence interval width (the difference between the upper and lower 95% limits of the interval), which indicates variation in sample sizes and in the precision achieved in each study (range: 0.5–146; mean interval width: 23.6). However, some degree of consistency in the ASD prevalence estimates is found in the center of this distribution, with a median rate of 61.6/10,000 and a mean rate of 66/10,000 (interquartile range: 43–80/10,000). Prevalence was negatively correlated with sample size (Spearman's r: $-.40$, $p = .005$), with small-scale studies reporting higher prevalence. There was also a significant positive correlation between ASD prevalence estimates and year of publication (Spearman's r: .29, $p = .04$), indicative of higher rates in more recent surveys.

Of note, five studies since 2000 reported ASD prevalences higher than 100/10,000 (median: 116.1/10,000; mean: 157.8/10,000) (Baird et al., 2006; CDC, 2012; Idring et al., 2012; Kawamura et al., 2008; Kim et al., 2011; see Figure 3.2). 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 (2012), also improve identification of individuals

TABLE 3.4 Prevalence Surveys of ASDs Since 2000

Year	Authors	Country	Area	Population	Age	Number Affected	Diagnostic Criteria	% with Normal IQ	Gender Ratio (M:F)	Prevalence Rate/10,000	95% CI
2000	Baird et al.	UK	South East Thames	16,235	7	94	ICD-10	60	15.7 (83:11)	57.9	46.8; 70.9
2000	Powell et al.	UK	West Midlands	58,654*	1–5	122	*Clinical*, ICD-10, *DSM-IV*	—	—	20.8	17.3; 24.9
2001	Bertrand et al.	United States	New Jersey	8,896	3–10	60	*DSM-IV*	51	2.7 (44:16)	67.4	51.5; 86.7
2001	Chakrabarti and Fombonne	UK	Stafford	15,500	2.5–6.5	96	ICD-10	74.2	3.8 (77:20)	61.9	50.2; 75.6
2001	Fombonne et al.	UK	England and Wales	10,438	5–15	27	*DSM-IV*, ICD-10	55.5	8.0 (24:3)	26.1	16.2; 36.0
2002	Scott et al.	UK	Cambridge	33,598	5–11	196	ICD-10	—	4.0 (—)	58.3*	50.7; 67.1*
2003	Yeargin-Allsopp et al.	United States	Atlanta, GA	289,456	3–10	987	*DSM-IV*	31.8	4.0 (787:197)	34.0	32; 36
2003	Gurney et al.	United States	Minnesota (2001–2002)	787,308*	6–11	4094	Receipt of MN special education services	—	—	52.0[a]	50.4; 53.6*
2003	Lingam et al.	UK	North East London	186,206	5–14	567	ICD-10	—	4.8 (469 :98)	30.5*	27.9; 32.9*
2004	Icasiano et al.	Australia	Barwon	45,153*	2–17	177	*DSM-IV*	53.4	8.3 (158:19)	39.2	33.8; 45.4*
2005	Chakrabarti and Fombonne	UK	Stafford	10,903	4–6	64	ICD-10	70.2	6.1 (55:9)	58.7	45.2; 74.9
2006	Baird et al.	UK	South Thames (1990–1991)	56,946	9–10	158	ICD-10	45	3.3 (121:37)	116.1	90.4; 141.8
2006	Fombonne et al.	Canada	Montreal	27,749	5–17	180	*DSM-IV*	—	4.8 (149:31)	64.9	55.8; 75.0
2006	Harrison et al.	UK	Scotland	134,661	0–15	443[b]	ICD-10, *DSM-IV*	—	7.0 (369:53)	44.2	39.5; 48.9
2006	Gillberg et al.	Sweden	Göteborg	32,568	7–12	262	*DSM-III, DSM-IV*, Gillberg's criteria for AS	—	3.6 (205:57)	80.4	71.3; 90.3
2006	Ouellette-Kuntz et al.	Canada	Manitoba and Prince Edward Island	227,526	1–14	657	*DSM-IV*	—	4.1 (527:130)	28.9*	26.8; 31.2*
2007	Croen et al.	United States	Northern California (1995–1999)	132,844	5–10	593	ICD-9-CM	—	5.5 (501:92)	45	41.2; 48.4*
2007[a]	CDC	United States	6 states	187,761	8	1,252	*DSM-IV-TR*	38 to 60[c]	2.8 to 5.5	67.0	63.1; 70.5*
2007[b]	CDC	United States	14 states	407,578	8	2,685	*DSM-IV-TR*	55.4[d]	3.4 to 6.5	66.0	63; 68

(*continued*)

TABLE g (*Continued*)

Year	Authors	Country	Area	Population	Age	Number Affected	Diagnostic Criteria	% with Normal IQ	Gender Ratio (M:F)	Prevalence Rate/10,000	95% CI
2007	Latif and Williams	UK	South Wales	39,220	0–17	240	ICD-10, *DSM-IV*, Kanner's and Gillberg's criteria	—	6.8 (—)	61.2	53.9; 69.4*
2008	Wong and Hui	China	Hong Kong Registry	4,247,206	0–14	682	*DSM-IV*	30	6.6 (592:90)	16.1	14.9; 17.3*
2008	Montiel-Nava and Pena	Venezuela	Maracaibo	254,905	3–9	430	*DSM-IV-TR*	—	3.3 (329:101)	17	13; 20
2008	Kawamura et al.	Japan	Toyota	12,589	5–8	228	*DSM-IV*	66.4	2.8 (168:60)	181.1	159.2; 205.9*
2008	Williams et al.	UK	Avon	14,062	11	86	ICD-10	85.3	6.8 (75:11)	61.9	48.8; 74.9
2009	Baron-Cohen et al.	UK	Cambridgeshire	8,824	5–9	83	ICD-10	—	—	94[e]	75; 116
2009	Nicholas et al.	United States	South Carolina	8,156	4	65	*DSM-IV-TR*	44.2	4.7	80	61; 99
2009	van Balkom et al.	Netherlands	Aruba	13,109	0–13	69	*DSM-IV*	58.8	6.7 (60:9)	52.6	41.0; 66.6
2009	CDC	United States	11 states	308,038	8	2, 757	*DSM-IV*	59	4.5	90	86; 93
2010	Fernell and Gillberg	Sweden	Stockholm	24,084	6	147	*DSM-IV*, *DSM-IV-TR*, ICD-10	33	5.1 (123:24)	62	52; 72
2010	Lazoff et al.	Canada	Montreal	23,635	5–17	187	*DSM-IV*	—	5.4 (158:29)	79.1	67.8; 90.4
2010	Barnevik-Olsson et al.	Sweden	Stockholm	113,391	6–10	250	*DSM-IV*	0	—	22	19.4; 25.0*
2010	Maenner and Durkin	United States	Wisconsin	428,030	Elementary school–aged	3831	*DSM-IV* like criteria for WI special education services (by school district)	—	—	90	86.7; 92.4*
2010	Posserud et al.	Norway	Bergen	9,430	7–9	16	*DSM-IV*, ICD-10 Included DAWBA and DISCO	—	7 (14:2)	87[f]	—
2011	Al-Farsi et al.	Oman	National Register	528,335	0–14	113	*DSM-IV-TR*	—	2.9 (84:29)	1.4	1.2; 1.7
2011	Brugha et al.	UK	England	7,333	16–98	72	ADOS	100	3.8	98.2	30; 165
2011	Kim et al.	S. Korea	Goyang City	55,266	7–12	201	*DSM-IV*	31.5	3.8	264	191; 337
2011	Mattila et al.	Finland	Northern Ostrobothnia County	5,484	8	37	*DSM-IV-TR* included ADOS-G and ADI-R	65	1.8	84	61; 115
2011	Parner et al.[g]	Australia	Western Australia (1994–1999)	152,060	0–10	678	*DSM-IV, DSM-IV-TR*	—	4.1	51	47; 55.3

2011	Samadi et al.	Iran	National Register	1,320,334	5	826	ADI-R	—	4.3	6.4	5.84; 6.70
2011	Chien et al.	Taiwan	National Health Research Institute	229,457*	0–18	659	ICD-9	—	3.7	28.7	26.6; 31*
2011	Windham et al.[h]	United States	San Francisco Bay Area (1994,1996)	80,249	9	374	"Full syndrome autism"—CA Dept. of Developmental Services, receipt of CA special education services, *or* *DSM-IV*	—	6.2 (324:50)	47	42; 52
2012	CDC	United States	14 states	337,093	8	3, 820	*DSM-IV*	38	4.6	113	110; 117
2012	Davidovitch et al.	Israel	Maccabi HMO Registry	423,524	1–12	2, 034	*DSM-IV*	—	5.2	48	45.9; 50.1
2012	Idring et al.	Sweden	Stockholm County Register	444,154	0–17	5, 100	ICD-09, ICD-10, *DSM-IV*	57.4	2.6	115	112; 118
2012	Isaksen et al.	Norway	Oppland and Hedmark	31,015	6–12	158	ICD-10 included ADOS-G and ADI-R	—	4.27 (128:30)	51	43; 59
2012	Kočovská, Biskuptso, et al.[i]	Denmark	Faroe Islands	7,128	15–24	67	ICD-10, *DSM-IV*, Gillberg's criteria	—	2.7* (49:18)	94	73; 119
2012	Nygren et al.	Sweden	Göteborg	5,007	2	40	*DSM-IV-TR*	63*	4 (32:8)	80	57; 109
2012	Parner et al.[j]	Denmark	National Register (1980–2003)	1,311,736	6–29	9, 556	ICD-8, ICD-9, ICD-10	—	4.1	72.9*	71.4; 74.3*

*Calculated by the authors.

[a]This is the prevalence for children aged 6–11 in the 2001–2002 school year.

[b]Estimated using a capture-recapture analysis, the number of cases used to calculate prevalence was estimated to be 596.

[c]Specific values for % with normal IQ and confidence intervals are available for each state prevalence.

[d]Average across seven states.

[e]Rate based on Special Education Needs register. A figure of 99/10,000 is provided from a parental and diagnostic survey. Other estimates in this study vary from 47 to 165/10,000 deriving from various assumptions made by the authors.

[f]This was the prevalence estimate based on the identified sample; when adjusted for nonresponders, the prevalence was estimated to be even higher (87/10,000).

[g]Note that this is an updated prevalence estimate: previous estimates have been reported by Nassar et al. (2009; birth years: 1983–1999; prevalence: 23.4/10,000) and Leonard et al. (2011; birth years: 1984–1999; singletons; prevalence: 30/10,000) using the same register in Western Australia.

[h]Data for 1996 birth cohort; overall prevalence for both 1994 and 1996 cohorts was 47/10000 although other specific values differed slightly. This study population may overlap to some degree with Croen et al. (2007), where 1995–1999 births only at Kaiser Permanente Northern California (KPNC) were examined; KPNC was one of three types of health-based sources used in Windham et al. (2011).

[i]Note that this is an updated prevalence estimate: a previous estimate of 53.3/10,000 was reported by Ellefsen et al. (2007) based on a survey of the same geographical area with the same cohort.

[j]Note that this is an updated prevalence estimate: a previous estimate was reported by Lauritsen et al. (2004; birth years: 1971–2000; prevalence: 34.4/10,000) and Parner et al. (2011; birth years: 1994–1999; prevalence: 68.5/10,000) using the same national register in Denmark.

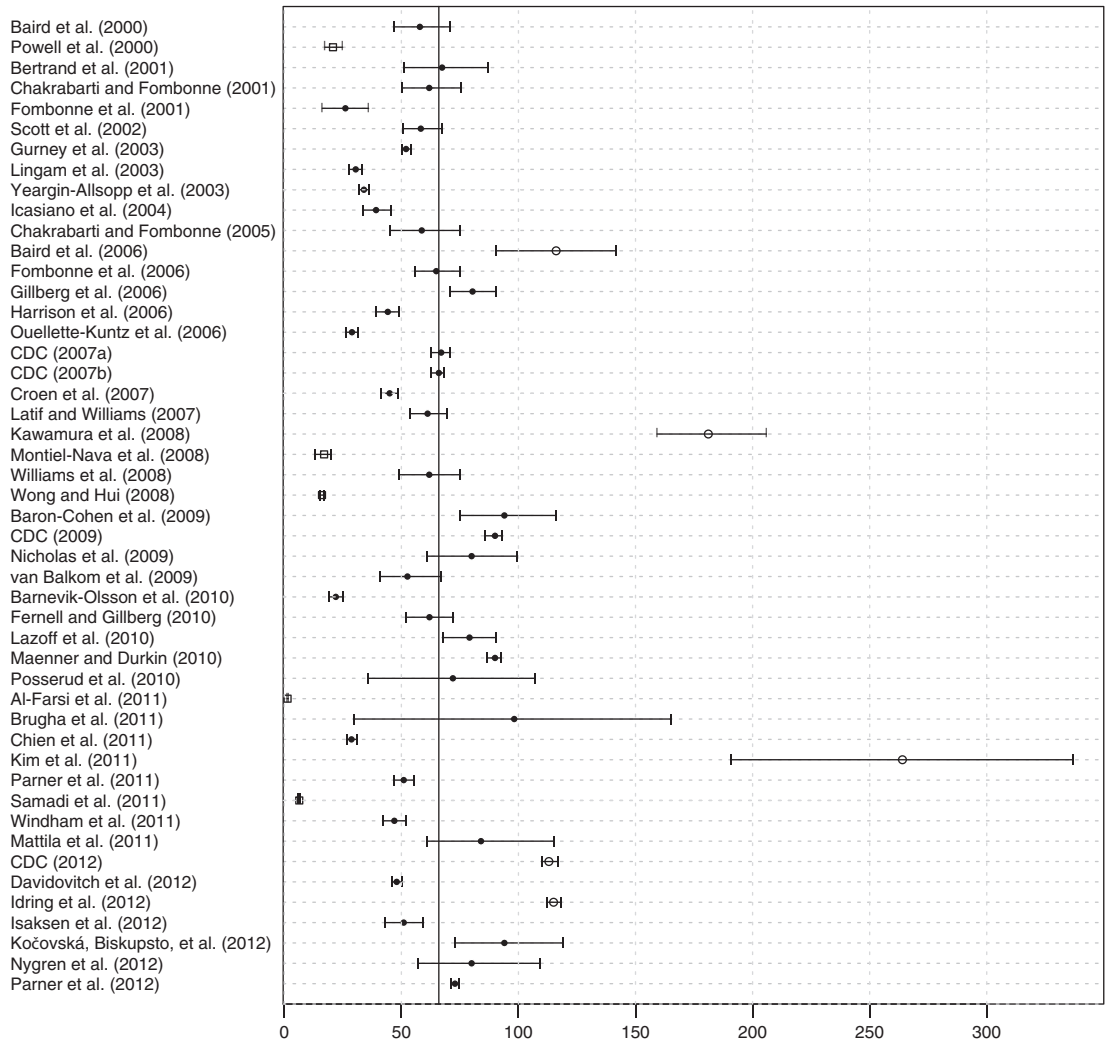

Figure 3.2 Prevalence estimates for ASD from Table 3.4 (rate per 10,000 and 95% confidence interval).

with ASD. The CDC's 2012 prevalence estimate of 113 per 10,000 reflects the highest estimate to date across all of the previous ADDM Network reports (2007a, 2007b, 2009). One factor associated with the prevalence increase in the CDC monitoring survey was improved quality and quantity of information available through records, indicative of greater awareness about ASD among community professionals. As surveillance efforts continue, it is likely that awareness and services will develop in states that were lagging behind, resulting in a predictable increase in the average prevalence for the United States as time elapses. These CDC findings apply to other countries as well, and prevalence estimates from any study should always be regarded in the context of the imperfect sensitivity of case ascertainment that results in downward biases in prevalence proportions in most surveys.

By contrast, the five studies reporting the lowest ASD prevalence estimates (Al-Farsi et al., 2011; Montiel-Nava & Pena, 2008; Powell et al., 2000; Samadi et al., 2011; Wong & Hui, 2008) probably underestimated the true population rates (see Figure 3.2). In three surveys (Al-Farsi et al.,

2011; Samadi et al., 2011; Wong & Hui, 2008), case finding depended on enrollment to a National Registry, a method usually associated with lower sensitivity for case finding. Similarly, both the U.K. (Powell et al., 2000) and Venezuelan surveys (Montiel-Nava & Pena, 2008) relied on review of school and/or medical records for case ascertainment, which are also associated with decreased sensitivity in prevalence surveys. Moreover, both the Omani and Iranian surveys (Al-Farsi et al., 2011; Samadi et al., 2011) attributed the low prevalence estimates to underdiagnosis, limited service access, and cultural factors, all of which likely contributed to underestimation of ASD prevalence in these populations.

Overall, results of recent surveys agree that an average figure of 66/10,000 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 66/10,000 (equivalent to 6.6/1,000 or .66%) translates into 1 child out of 152 with an ASD diagnosis. This estimate is now the best estimate for the prevalence of ASDs currently available. However, this represents an average and conservative figure, and it is important to recognize the substantial variability that exists between studies and within studies, across sites or areas. In the studies reviewed here, 19 of the 48 studies reported ASD prevalence rates higher than 66/10,000, with some recent studies reporting rates even 2 to 4 times higher (Kawamura et al., 2008; Kim et al., 2011).

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 used by investigators, or in the interpretation of their meaning. In particular, it is crucial to differentiate prevalence from incidence. Whereas prevalence is useful to estimate needs and plan services, only incidence rates can be used for causal research. Both prevalence and incidence estimates will increase when case definition is broadened and case ascertainment is improved. Time trends in rates can therefore only be gauged in investigations that hold these parameters under strict control over time. These methodological requirements must be borne in mind while reviewing the 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 (what the term *epidemic* means). However, alternative explanations for the rise in numbers of children diagnosed with ASDs should be ruled out first before supporting this conclusion, and include the following.

Use of Referral Statistics

Increasing numbers of children referred to specialist services or known to special education registers have been taken as evidence for an increased incidence of ASDs. Upward trends in national registries, medical, and educational databases have been seen in many different countries (Gurney et al., 2003; Madsen et al., 2002; 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 many factors such as referral patterns, availability of services, heightened public awareness, decreasing age at diagnosis, and changes over time in diagnostic concepts and practices.

Failure to control for these confounding factors was obvious in previous reports (Fombonne, 2001), such as the widely quoted reports from California Developmental Database Services (CDDS; 1999, 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. However, it is important to note that an increase in referrals does not necessarily mean increased *incidence*. Studies based solely on cases registered for services cannot rule out that the proportion of cases within the general population who registered with services has changed over time.

As an illustration, in Figure 3.3, we contrast 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% within a certain time interval, prevalence would be underestimated at both time points, yet would appear to rise 200% in that time interval while the underlying true incidence and prevalence remained stable. This type of pattern of results was recently reported based on special education data in Wisconsin (Maenner & Durkin, 2010), in which ASD prevalence rates appeared to level off between 2002 and 2008 in school districts with initially high baseline

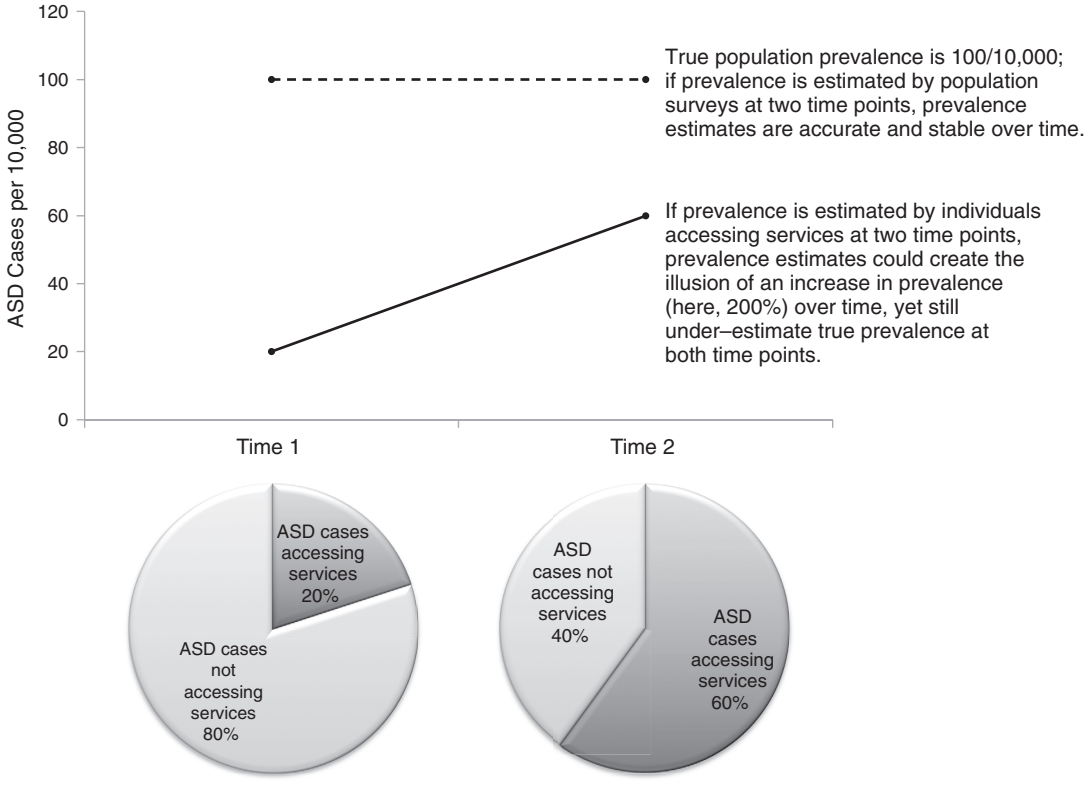

Figure 3.3 Impact on prevalence estimates and trends of two methods for surveying ASD: population sampling, or reliance of service access counts (hypothetical data).

prevalence rates (∼120/10,000), whereas school districts with the lowest baseline rates experienced significant increases in prevalence over the same time period (e.g., in one district rates rose from 5 to 70/10,000; corresponding to a 1300% increase in 6 years). As illustrated in Figure 3.3, in order to accurately estimate prevalence and gauge time trends, data over time are needed both on referred subjects and on nonreferred (or referred to other services) subjects.

The Role of Diagnostic Substitution

One possible explanation for increased numbers of a diagnostic category is that children presenting with the same developmental disability may receive one particular diagnosis at one time and another diagnosis at a subsequent time. 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 switching contributing to the prevalence increase was produced in all U.S. states 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 as having mental retardation were subsequently identified as having an ASD diagnosis. Shattuck showed that the odds of being classified in the autism category increased by 1.21 during 1994–2003. Concurrently, the odds of being classified in the learning disability (LD) (odds ratio: OR = 0.98) and the mental retardation (MR) categories (OR = 0.97) decreased significantly. Shattuck (2006) further demonstrated that the growing prevalence of autism 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 autism became reported in the United States as an independent category in 1993–1994. Finally, Shattuck (2006) showed that, from 1994 to 2003, the mean increase for the combined category of Autism + Other Health Impairments + Trauma Brain Injury + Developmental Delay was 12/1000, whereas the mean decrease for MR and LD was 11/1000 during the same period. One exception to these ratios was California, for which previous authors had debated the presence of diagnostic substitution between MR and autism (Croen, Grether, Hoogstrate, et al., 2002a; Eagle, 2004).

The previous investigations have largely relied on ecological, aggregated data that have known limitations. Using individual level data, a newer study reexamined the hypothesis of diagnostic substitution in the California DDS dataset (King & Bearman, 2009) and showed that 24% of the increase in caseload was attributable to such diagnostic substitution (from the mental retardation to the autism category). It is important to keep in mind that other types of diagnostic substitution are likely to have occurred as well for milder forms of the ASD phenotype, from various psychiatric disorders (including childhood schizoid personality disorders; Wolff & Barlow, 1979) that have not been studied yet (Fombonne, 2009b). For example, children currently diagnosed with Asperger's disorder were 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.

Evidence of diagnostic substitution within the class of developmental disorders has also been provided in U.K.-based studies. Using the General Practitioner Research Database, Jick and Kaye (2003) demonstrated that the incidence of specific developmental disorders (including language disorders) decreased by about the same amount that the incidence of diagnoses of autism increased in boys born from 1990 to 1997. Another U.K. study (Bishop, Whitehouse, Watt, & Line, 2008) showed that up to 66% of adults previously diagnosed as children with developmental language disorders would meet diagnostic criteria for a broad definition of ASD. This change was observed for children diagnosed with specific language impairments, but even more so for those diagnosed with pragmatic language impairments.

Comparison of Cross-Sectional Epidemiological Surveys

Epidemiological surveys of autism each possess unique design features that could account almost entirely for between-studies variation in rates. Therefore, time trends in rates of autism are difficult to gauge from published prevalence rates. The significant aforementioned correlation between prevalence rate and year of publication for autistic disorder could merely reflect increased efficiency over time in case identification methods used in surveys as well as changes in diagnostic concepts and practices (Bishop et al., 2008; Kielinen, Linna, & Moilanen, 2000; Magnússon & Saemundsen, 2001; Shattuck, 2006; Webb, Lobo, Hervas, Scourfield, & Fraser, 1997). In studies using capture-recapture methods, it is apparent that up to a third of prevalent cases may be missed by an ascertainment source, even in recently conducted studies (Harrison et al., 2006). 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 derived from surveys where intensive population-based screening techniques were employed, whereas lower rates were obtained from studies relying on passive administrative methods for case finding. Since no passage of time was involved, the magnitude of these gradients in rates can only be attributed to differences in case identification methods across surveys.

Even more convincing evidence comes from the survey by the CDC on 337,093 U.S. children aged 8 in 2008, where an average prevalence of 113/10,000 was reported across 14 U.S. states (CDC, 2012). One striking finding in this report is the almost 4.5-fold variation in prevalence rates by state (range: 48–212 per 10,000; see Figure 3.4).

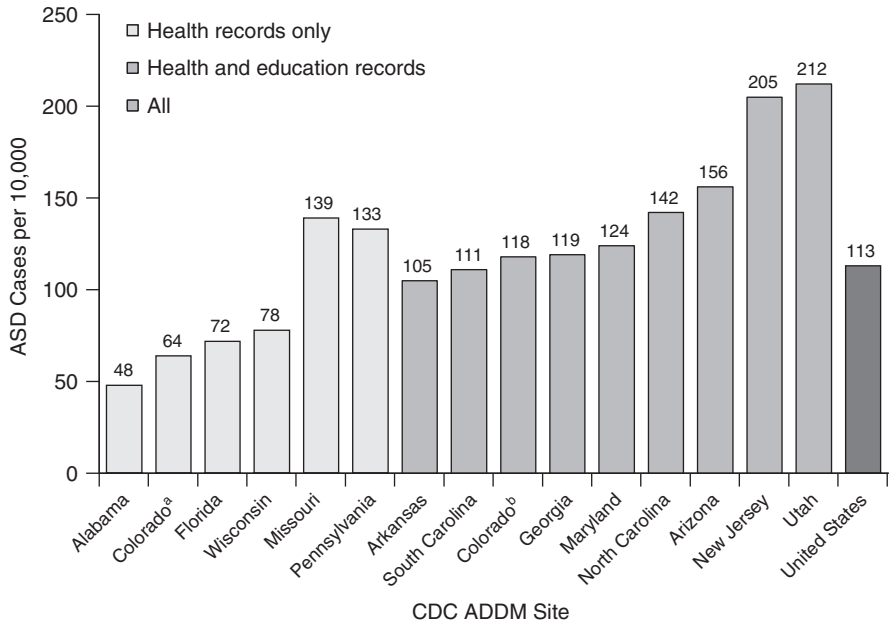

Figure 3.4 Estimated prevalence of ASDs among children aged 8 years in the United States by ADDM site and type of records access (CDC, 2012).

Across individual states, Alabama had the lowest rate of 48/10,000, whereas Utah and New Jersey had the highest rates (212 and 205 per 10,000, respectively; CDC, 2012). It would be surprising if there were truly this much state-to-state variability in the number of children with autism in the United States. These substantial differences most certainly reflected 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.

On average, 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 (89 versus 144 out of 10,000, respectively). This is exemplified by data from Colorado, in which two prevalence estimates were available: one based on six counties with access to health data sources only (64 per 10,000), and the other based on a single county with access to both health and education records (118 per 10,000). Thus, within one state, there was a 1.8-fold variation in prevalence estimates depending on the availability of records (CDC, 2012). Although differences in prevalence estimates across states cannot be attributed solely to records access (e.g., service availability and other state-specific factors are also likely to contribute), this is a factor that is consistently associated with higher prevalence rates in the ADDM Network. The 2012 CDC report also included the highest proportion of identified children with a previously documented ASD classification for any ADDM surveillance year (79%), offering evidence that community providers are increasingly likely to identify and document ASDs. Taken together with the higher proportion of children with ASD diagnosed by 36 months of age and the increased identification among children without intellectual disability, these factors suggest that improved sensitivity in case ascertainment within the ADDM Network has contributed substantially to the increase in prevalence. Thus, no inference on trends in the incidence of ASDs can be derived from a simple comparison of prevalence rates over time, since studies conducted at different periods are likely to differ even more with respect to their methodologies.

Repeat Surveys in Defined Geographical Areas

Repeated surveys, using the same methodology and conducted in the same geographical area at different points in time, can potentially yield useful information on time trends provided that 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) and 9.5/10,000 (1988), the gradient being even steeper if rates for the urban area alone are considered (4.0, 7.5, and 11.6/10,000, respectively) (C. Gillberg et al., 1991). However, comparison of these rates is not straightforward, as different age groups were included in each survey. Second, the increased prevalence in the second survey was explained by improved detection among those with intellectual delays, and that of the third survey by cases born to immigrant parents. That the majority of the latter group was born abroad suggests 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 definition of autism over time that was acknowledged by the authors (C. Gillberg et al., 1991), these findings do not provide evidence for an increased incidence in the rate of autism. Similarly, studies conducted in Japan at different points in time in Toyota (Kawamura, Takahashi, O., & Ishii, 2008) and Yokohama (Honda, Shimizu, Misumi, Niimi, & Ohashi, 1996; Honda, Shimizu, & Rutter, 2005) 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.

Comparisons between successive CDC ADDM surveillance years also shed light on time trends using consistent methodology. In the 2008 surveillance year (prevalence: 113/10,000; CDC, 2012), 11 sites also contributed to the 2006 surveillance year (prevalence: 90/10,000; CDC, 2009). Of those, seven sites identified a higher prevalence in 2008 compared to 2006, whereas three sites were similar across both years, and one site (Alabama) reported a lower prevalence in 2008 compared to 2006, with rates increasing on average 23% during 2006–2008. In comparing the 2008 surveillance year to 2002, 12 out of 13 sites that contributed to both reports identified significantly higher prevalence in 2008 than in 2002 (with the exception of Arkansas), with rates increasing on average 78% during 2002–2008. Nevertheless, CDC researchers concluded that increases in ASD prevalence over successive surveillance years were influenced by a number of factors, including increased awareness and access to services, making it impossible to determine whether any proportion of the observed increase is attributable to a true increase in ASD in the population.

Birth Cohorts

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

An analysis of special educational data from Minnesota showed a 16-fold increase in the number of children identified with an ASD from 1991–1992 to 2001–2002 (Gurney et al., 2003). The increase was not specific to autism since, during the same 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, the prevalence rose from 18/10,000 in those born in 1989, to 29/10,000 in those born in 1991, and to 55/10,000 in those born in 1993, suggestive of birth cohort effects. 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 that expected from a chronic nonfatal condition diagnosed during the first years of life. Their analysis also showed a marked period effect that identified the early 1990s as the period where rates started to increase in all ages and birth cohorts. Gurney et al. (2003) further argued that this phenomenon coincided closely with the inclusion of ASDs in the federal Individuals with Disabilities Educational Act (IDEA) funding and reporting mechanism in the United States. A similar interpretation of upward trends had been put forward by Croen, Grether, Hoogstrate, et al. (2002a) in their analysis of the California DDS data, and by Shattuck (2006) in his well-executed analysis of trends in the Department of Education data in all U.S. states.

Using hypothetical data, increasing prevalence rates across and within birth cohorts are illustrated in Figure 3.5. As reported in several studies (e.g., Gurney et al., 2003; Keyes et al., 2012; Nassar et al., 2009), we portray an increase in the prevalence of ASD by year of birth across three hypothetical successive birth cohorts (a cohort effect; Figure 3.5a). Within each birth cohort, followed longitudinally, prevalence rates also increase as children age

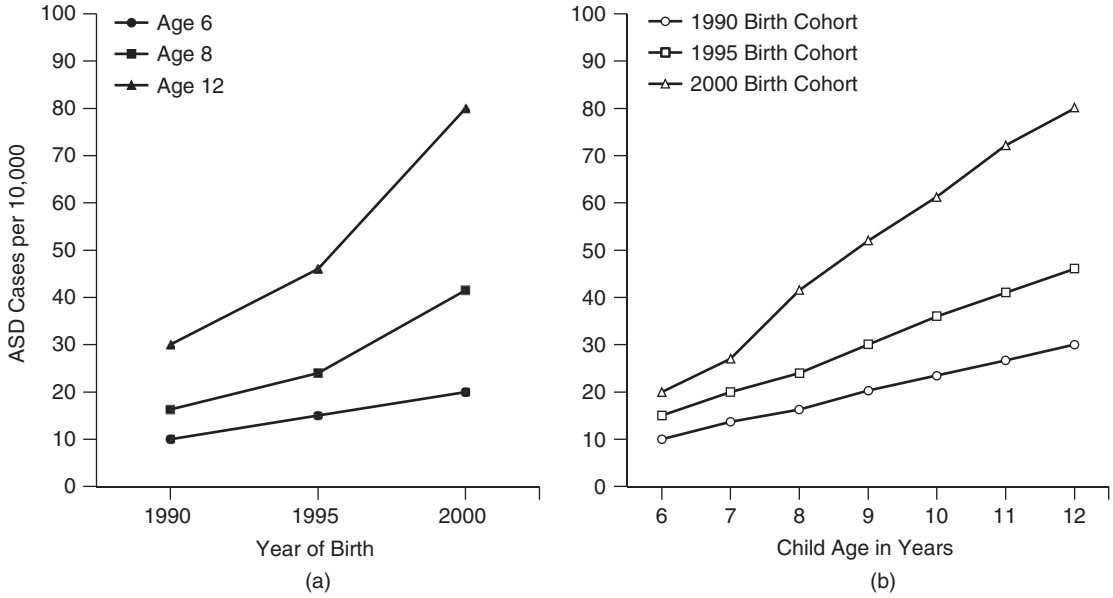

Figure 3.5 Time trends in ASD prevalence rates across and within birth cohorts (hypothetical data).

(Figure 3.5b): 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 cannot 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.

A similar scenario was recently reported in a Faroe Islands survey in which researchers followed up on a 2002 population study (Ellefsen et al., 2007). In the follow-up study, Kočovská, Biskupsto, et al. (2012) observed a substantially increased ASD prevalence of 94/10,000 in individuals aged 15 to 24 years, compared to 53.3/10,000 in almost the exact same sample at ages 8 to 17 (Ellefsen et al., 2007). If treated as two separate cross-sectional studies separated by 7 years, we might interpret this pattern of results as a rise in incidence. However, because the sample was almost exactly the same cohort as in the first study, the researchers suggest the rising prevalence in this study reflects newly identified cases that were simply missed before, and suggest that the apparent rise may be due to lack of awareness for the clinical presentation of ASD in females, which accounted for the majority of missed cases in the follow-up study.

Implications of Upcoming Changes to Diagnostic Criteria

The changes now occurring in the DSM with the new fifth edition (*DSM-5*; APA, 2013) may impact prevalence estimates in the future and, if so, will make it more difficult to compare past and future surveys and interpret time trends. *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 (7 instead of 12) that are combined in two clusters of social communication deficits (three criteria; all must be met) and restricted patterns of behaviors and interests (two of the four criteria must be met). The removal of the loosely defined PDD-NOS from *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. Concerns have been raised that subjects with a previous *DSM-IV* diagnosis of PDD may fail to meet the more stringent diagnostic criteria for ASD in *DSM-5*, thereby losing access to services and support systems that require a diagnosis for eligibility. Nine studies have recently been published that examined the relationship between *DSM-5* and *DSM-IV* or ICD-10 using clinical or research samples of various origins (Frazier et al., 2012; Gibbs, Aldridge, Chandler, Witzlsperger, & Smith, 2012; Huerta, Bishop, Duncan, Hus, & Lord, 2012; Matson, Belva, Horovitz, Kozlowski, & Bamburg, 2012; Matson, Hattier, & Williams, 2012; Mattila et al., 2011; McPartland, Reichow, & Volkmar, 2012; Taheri & Perry, 2012; Worley & Matson, 2012). The proportion of subjects who met criteria for ASD in *DSM-5* ranged from a low 46% (Mattila et al., 2011) to a high 91% (Huerta et al., 2012). It is important to recognize that by design these proportions could only be lower or equal to 100%. Due to the fact that past diagnostic information was collected at a time when *DSM-5* was not available, it is very possible that the new information required in *DSM-5* (e.g., with a new diagnostic emphasis on sensory processing deficits) was simply not available for rescoring *DSM-5*. Equally, the studies were constrained in sampling children with a *DSM-IV* PDD diagnosis and could not therefore estimate which proportion of children who did not meet criteria for *DSM-IV* would have met those for *DSM-5* should data on children failing *DSM-IV* criteria had been available.

The impact of *DSM-5* changes on epidemiological estimates remains to be fully assessed in the context of epidemiological surveys. Only one study has thus far shed light on this issue. Kim et al. (submitted) reanalyzed the survey data of the South Korean study (Kim et al., 2011) and reapplied both *DSM-IV* and *DSM-5* criteria to a population based sample of cases identified in the original survey. The authors used the *DSM-5* ASD category as well as the new diagnostic category of Social Communication Disorder (SCD) that has been added to ASD largely in anticipation of children with few repetitive behaviors and a PDD-NOS diagnosis in *DSM-IV* now meeting criteria for SCD rather than ASD in *DSM-5*. Indeed, the prevalence estimate for *DSM-5* ASD in the South Korean survey was 17% lower (2.20%) than that with *DSM-IV* (2.64%); 99% of subjects with a *DSM-IV* diagnosis and 92% of those with an Asperger Disorder diagnosis met *DSM-5* criteria for ASD, and all others met criteria for SCD. For PDD-NOS, 63% met criteria for ASD and an additional 32% for SCD. When *DSM-5* ASD and SCD were considered together, there was no significant change in the prevalence estimate. 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*.

Conclusion on Time Trends

As it stands now, 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. There is good evidence that changes in diagnostic criteria, diagnostic substitution, changes in the policies for special education, and the increasing availability of services are responsible for the higher prevalence figures. It is also noteworthy that the rise in number of children diagnosed occurred at the same time in many countries (in the early 1990s), when radical shifts occurred in the ideas, diagnostic approaches, and services for children with ASDs. Alternatively, this might, of course, reflect the effect of environmental influences operating simultaneously in different parts of the world. However, there has been no proposed and legitimate environmental risk mechanism to account for this worldwide effect. Moreover, due to the relatively low frequency of autism and ASDs, statistical power is a significant limitation in most investigations, and variations of small magnitude in the incidence of the disorder are very likely to go undetected. Equally, 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 eliminated based on available data. It remains to be seen how changes to diagnostic criteria introduced in the *DSM-5* will impact estimates of ASD prevalence.

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 & Hamhi, 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 (e.g., Powell et al., 2000; Sponheim & Skjedal, 1998).

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, 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; similar results were found in adjusted analysis performed in China (N. Li et al., 2011). In a Swedish case-control study by Rai, Lewis, et al. (2012), 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. (2012) 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 (e.g., 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.

Race/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, Walker, Mandell, Bayles, & Miller, (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. A 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 also suggest an overall lower rate of ASD among non-Hispanic Black (102/10,000) and Hispanic children (79/10,000) compared to White children (120/10,000) in the 14 U.S. states that participated in the study. However, there was considerable variability among the states, with some states reporting higher rates of ASD among Hispanics than among Whites, for example, suggesting that administrative records may have had systematic biases in some states. In addition, when the surveillance data from 2008 was compared to previous waves of data collection, Hispanic and African American populations had greater increases in diagnosis rates (respectively, 29% and 42%; although continued overall rates of underdiagnosis) than non-Hispanic White children (16%; CDC, 2012). 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 may 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. (2012) 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, 2012a, for a recent review on the role of vitamin D in ASD). 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., 1987; C. Gillberg et al., 1991; C. Gillberg et al., 1995).

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.

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; C. Gillberg et al., 1991; C. Gillberg et al., 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 (e.g., Hultman, Sparen, & 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 (e.g., 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 (loss of a child, spouse, parent, or sibling during or up to 1 year prior to pregnancy) was associated with increased risk of ASD (ICD-08/ICD-10 criteria). 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 prenatal exposure to stressful life events, including deaths, serious accidents, and diagnosis of serious illnesses in first-degree relatives, and ASD risk, 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.

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.

CONCLUSION

Epidemiological surveys of autism and ASDs have now been conducted in many countries. Methodological differences regarding case definition and finding procedures make between survey comparisons difficult to perform. However, from recent studies, a best estimate of (66/10,000) (equivalences = 6.6/1,000 or .66% or 1 child in about 152 children) can be confidently derived for the prevalence of ASD. Current evidence does not strongly support the hypothesis of a secular increase in the incidence of autism, but power to detect time trends is seriously limited in existing datasets. While it is clear that prevalence estimates have increased over time, this increase most likely represents changes in the concepts, definitions, service availability, and awareness of autistic-spectrum disorders in both the lay and professional public. 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. The possibility that a true change in the underlying incidence has contributed to higher prevalence figures remains to be adequately tested. Meanwhile, the available prevalence figures carry straightforward implications for current and future needs in services and early educational intervention programs.

CROSS-REFERENCES

Chapter 1 addresses diagnostic issues; Chapter 2 focuses on the broader autism phenotype, Chapter 24 and 25 on screening and assessment instruments, and Chapter 49 on social policy and services planning.

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