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The Epidemiology of Autistic Spectrum Disorders: Is the Prevalence Rising?

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For decades after Kanner's original paper on the subject was published in 1943, autism was generally considered to be a rare condition with a prevalence of around 2–4 per 10,000 children. Then, studies carried out in the late 1990s and the present century reported annual rises in incidence of autism in pre-school children, based on age of diagnosis, and increases in the age-specific prevalence rates in children. Prevalence rates of up to 60 per 10,000 for autism and even more for the whole autistic spectrum were reported. Reasons for these increases are discussed. They include changes in diagnostic criteria, development of the concept of the wide autistic spectrum, different methods used in studies, growing awareness and knowledge among parents and professional workers and the development of specialist services, as well as the possibility of a true increase in numbers. Various environmental causes for a genuine rise in incidence have been suggested, including the triple vaccine for measles, mumps and rubella (MMR). Not one of the possible environmental causes, including MMR, has been confirmed by independent scientific investigation, whereas there is strong evidence that complex genetic factors play a major role in etiology. The evidence suggests that the majority, if not all, of the reported rise in incidence and prevalence is due to changes in diagnostic criteria and increasing awareness and recognition of autistic spectrum disorders. Whether there is also a genuine rise in incidence remains an open question.

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The concept of autism in children was first introduced by Leo Kanner in his seminal paper published in 1943. He described a pattern of behavior he called 'early infantile autism' characterised by severe impairment of social interaction and communication and intense resistance to change. For decades after this publication, childhood autism was considered to be a rare condition. Then, in the late 1980s and the 1990s, this view was challenged. The most recent studies have reported progressively rising annual incidence rates, and prevalence rates considerably higher than in almost all the early studies. In order to provide a background for the discussion of possible reasons for this observed increase, I will briefly outline the history of autism up to the 1960s.

MYTHS, LEGENDS, AND HISTORY

The history of autistic disorders stretches far back into the mists of time. There are ancient myths, common to many parts of the world, of 'changeling children' [Brauner and Brauner, 1986]. These stories concern elfin children left in place of real human babies who have been stolen away by the 'little people.' In some versions of these myths, the description of the beautiful but strange and remote changeling sounds very like a child with autism. The legends concerning the followers of St. Francis of Assisi include stories about Brother Juniper [Frith, 1991]. He was gentle, naïve and very stubborn. He followed the Franciscan precepts with absolute literalness even to the extent of removing all his clothes in public in order to give them to a beggar. The other brothers evidently viewed him with a mixture of fondness and exasperation but thought his odd behavior was due to his saintliness. Nowadays, he might well have been diagnosed as having Asperger's syndrome. There are many examples of characters in fiction who also had marked traits of Asperger's syndrome, including Sherlock Holmes [Frith, 1989] and Mr. Bean in the television comedy series.

Turning to factual history, there are some accounts of individuals who probably did have autism. The most famous of these was Victor, known as the 'Wild Boy of Aveyron.' He was found when he was about 12 years old living wild in the woods of south central France at the end of the 18th century. He was captured and placed in the charge of the physician, J.M.G. Itard, to be educated. Itard wrote long, wonderfully vivid reports on the boy [Lane, 1977]. It is quite clear from these accounts that Victor (as Itard named him) had classic autism with little or no comprehension or use of language or other form of communication.

It seems that no one saw the connections among the individuals with these odd patterns of behavior until Henry Maudsley [1867] discussed what he referred to as 'insanity' in children. Among those he described there were probably some who would now be diagnosed as having autism. Then, in the first half of the 20th century, several workers in the field wrote about forms of 'childhood psychosis.' Attempts were made to define separate 'syndromes' [Wing, 1997]. In the light of current ideas concerning the wide autistic spectrum, it is interesting to recall that James Anthony [1958] noted that there were not enough symptoms to go round to justify all the syndromes that

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were suggested. Of all the early workers in this field, Leo Kanner in the USA and Hans Asperger in Austria are the only ones whose names are now legitimately famous. Kanner was the first to achieve this status. He wrote his classic paper on the behavior pattern he referred to as 'early infantile autism' in 1943. In 1944 Asperger wrote his first paper on a different, but related, behavior pattern he called 'autistic psychopathy' now referred to as Asperger's syndrome. His paper was in German and little known in English speaking countries until the 1980s and 1990s, when an account of his work [Wing, 1981] and a translation of his first paper [Frith, 1991] were published in English.

Although Kanner [1943] at first considered that early infantile autism was genetic in origin, the climate of opinion current at the time, especially in the USA, was heavily influenced by psychoanalytical theory. The result was that parents' attitudes to their children, their personalities, and their child rearing methods were blamed and autism was regarded as an emotional disorder without any neurological basis. The tide began to turn in the 1960s for two main reasons. One was that groups of parents in the USA and the UK, who firmly rejected the idea that they were to blame for their children's problems, came together to form voluntary associations. The other was the development of scientific research into autism. As a result of the ever growing list of studies, autism is now seen as a disorder of the developing brain, mainly genetic in origin and part of a wider spectrum of disorders. This spectrum is characterised by a triad of impairments affecting social interaction, communication and imagination associated with a narrow, repetitive range of activities. Changes in concepts of autism over the decades are reflected in the different editions of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM) and the World Health Organisation's International Classification of Diseases (ICD). These are discussed and references given in the later section on changes in diagnostic criteria.

DEFINITION OF INCIDENCE AND PREVALENCE

As noted in the introduction, increases in rates of autistic spectrum disorders have been found in studies of both incidence and prevalence. There are particular implications for the use of these terms in relation to autistic conditions. Incidence refers to the number of individuals in a specified population in whom the condition being studied *begins* within a specified time period, such as one year. Prevalence refers to the number of individuals in a specified population who have the condition being studied at a specified time, such as one particular day, regardless of when it began. The usefulness of each index varies with the nature of the condition being studied. With a condition such as measles, that has an obvious onset, lasts about two weeks unless there are complications, and has an obvious end point, the annual incidence will be larger and usually of more practical interest than the one day prevalence. Variations on the theme of incidence, such as changes with different seasons, may also be important. The problem with calculating incidence rates for autistic conditions is that the age of onset is very difficult to define and to ascertain. There is strong evidence, to be discussed later in this paper, that genetic factors are of major importance in aetiology. Because autistic conditions are long-lasting, prevalence for age-groups at which all cases should, in theory, be diagnosed, must be larger than the annual incidence and is of particular importance for estimating the services needed. If they could be calculated accurately, incidence rates would be more sensitive indicators than prevalence of changes in etiological factors.

Studies of Incidence

Because of the difficulties of defining onset, there have been only a few studies of the annual incidence of autistic disorders. They include the studies in the UK by Taylor et al. [1999], Powell et al. [2000], who also calculated prevalence, and Kaye et al. [2001]; and in California by Dales et al. [2001]. These covered birth cohorts in the 1980s and 1990s. All showed a steady rise in annual incidence of autistic spectrum disorders but all relied on case records of children diagnosed as autistic and used the year of diagnosis as the year of onset (see discussion later). The children in these studies were not seen by the research workers to confirm the diagnoses.

Studies of Prevalence

Most epidemiological studies of autistic spectrum disorders have examined prevalence. A list of 39 studies of prevalence published to date in the English language or with detailed abstracts in English is given in Table 1. They are referred to by number in the text below. It can be seen that there is a marked tendency over time for an increase in the rates to be found.

POSSIBLE REASONS FOR INCREASE IN INCIDENCE AND PREVALENCE

I shall consider the possible reasons for this observed rise under the following headings:

1. Changes in diagnostic criteria.
2. Differences in methods used in the studies.
3. Increasing awareness among parents, professionals and the general public of the existence of autistic spectrum disorders.
4. Recognition that autistic conditions can be associated with:
   a. severe or profound mental retardation and other developmental or physical disorders;
   b. average or high intellectual ability;
   c. psychiatric disorder of any type.
5. The development of specialist services.
6. Possible causes and relation to age of onset.
7. Possible true increase in numbers.

Changes in Diagnostic Criteria

Evolution of terminology

There are no definitive diagnostic tests for autism. Diagnosis is made from a detailed developmental history and observation of behavior in structured and unstructured situations. This process is fraught with difficulties of definition and standardisation. Over the years since Kanner described the pattern of behavior he called 'early infantile autism,' research in the field has resulted in the development of the concept of a spectrum of autistic disorders [Wing and Gould, 1979], which is considerably wider than Kanner's original group. There have been numerous suggestions for diagnostic criteria, but the discussion in this section will be confined to criteria used in the prevalence studies in Table 1.

Kanner and Eisenberg [1956] published a list of diagnostic criteria for early infantile autism. They emphasised two behavioural features as necessary and sufficient: first, aloofness and indifference to others and, second, intense resistance to change in the child's own repetitive routines, which had to be *elaborate* in form. These features also had to be present by 24 months at the latest. Rutter [1978] published criteria for defining what he

Table 1. Prevalence Studies: Age Specific Rates per 10,000 Children (Age Ranges Vary)

	Authors	Year published	Area studied	Rate of autism*/ other ASD**	Criteria used for autism/other ASD
A	***Studies giving rates of autism (some also give rates of other autistic spectrum disorder)***				
1	Lotter	1966	Middlesex, England	4.5/—	Kanner/—
2	Brask	1970	Aarhus, Denmark	4.3/—#	Kanner/—
3	Treffert	1970	Wisconsin, USA	0.7/2.4	Kanner/DSM-II
4	Wing & Gould***	1979	Camberwell, England	4.6/15.7#	Kanner/Triad
5	Hoshino et al.	1982	Fukushima, Japan	5.0/—	Kanner
6	Bohman et al.	1983	Vasterbotten, Sweden	3.0/2.6	Rutter/Rutter
7	Ishii & Takahashi	1983	Toyota, Japan	16.0/—#	Rutter/—
8	McCarthy et al.	1984	E. Health Bd, Ireland	4.3/—	Kanner/—
9	Gillberg	1984	Gothenburg, Sweden	2.0/1.9	DSM-III/DSM-III
10	Gillberg et al.***	1986	Gothenburg, Sweden	3.3/14.3#	DSMIII/Triad
11	Steffenburg & Gillberg	1986	Gothenburg, Sweden	4.7/2.8	DSM-III/DSM-III
12	Steinhausen et al.	1986	W. Berlin, Germany	1.9/—	Rutter/—
13	Matsuishi et al.	1987	Kurume City, Japan	15.5/—#	DSM-III/—
14	Burd et al.	1987	North Dakota, USA	1.2/2.1	DSM-III/DSM-III
15	Tanoue et al.	1988	Ibaraki, Japan	13.8/—	DSM-III
16	Bryson et al.	1988	Nova Scotia, Canada	10.1/—#	DSM-III-R/—
17	Ritvo et al.	1989	Utah, USA	4.0/—	DSM-III/—
18	Sugiyama & Abe	1989	Nagoya, Japan	13.0/—#	DSM-III/—
19	Cialdella & Mamelle	1989	Rhone, France	5.1/5.2	DSM-III/DSM-III
20	Gillberg et al.	1991	Gothenburg, Sweden	8.4/3.2	DSM-IIIR/DSM-IIIR
21	Fombonne & Mazaubrun	1992	Four regions, France	4.9/—	ICD-10/—
22	Honda et al.	1996	Yokohama, Japan	21.1/—#	ICD-10/—
23	Fombonne & Mazaubrun	1997	Three departments, France	5.4/10.9	ICD-10/ICD-10
24	Arvidsson et al.	1997	Molnlycke, Sweden	31.0/15.0#	ICD-10/ICD-10
25	Webb et al.	1997	S. Glamorgan, Wales	7.2/—	DSM-III-R/—
26	Sponheim & Skjeldae	1998	Akershus, Norway	3.8/1.4	ICD-10/ICD-10
27	Tomita	1999	Tokyo, Japan	32.0/58.0#	ICD-10/ICD-10
28	Kadesjo et al.	1999	Karlstad, Sweden	60.0/60.0#	ICD-10/Gillberg[1]
29	Magnusson & Saemundsen	2000	Iceland	8.6/4.6#	ICD-10/ICD-10
30	Baird et al.	2000	S.E. Thames, England	30.8/27.1#	ICD-10/ICD-10
31	Powell et al.	2000	W. Midlands, England	16.2/17.5#	DSM-IIIR or ICD-10
32	Kielinen et al.	2000	N. Finland	12.2/1.7	DSM-IV/DSM-IV
33	Bertrand et al.	2001	Brick Township, New Jersey, USA	40.0/27.0#	DSM-IV/DSM-IV
34	Croen et al.	2001	California, USA	11.0/—	DSM-IIIR or DSM-IV
35	Chakrabarti & Fombonne	2001	Staffs Co., England	16.8/45.8#	DSM-IV/DSM-IV
B	***Studies giving combined rates for autism and other autistic spectrum disorders***				
36	Fombonne et al.	2001	Great Britain	26.1#	DSM-IV
37	Scott et al.	2002	Cambridge, England	57.0#	DSM-IV
C	***Studies giving combined rates for Asperger's syndrome and high functioning autism***				
38	Ehlers & Gillberg****	1993	Gothenburg, Sweden	36.0 *+35*#	Gillberg[1]
39	Webb et al.	2000	Cardiff, Wales	20.0#	ICD-10

*"Autism" includes Kanner's early infantile autism, childhood autism, autistic disorder, as defined in the relevant sets of criteria.
**"Other ASD" includes subgroups of the autistic spectrum other than "autism." These differ among the studies listed.
***All participants in these studies had IQ below 70.
****The rate in italics is for children with marked social impairment but not the full picture of Asperger's syndrome.
#Population studied < 50,000.
[1]Gillberg's criteria for Asperger syndrome [Ehlers and Gillberg, 1993].

called 'childhood autism.' These were, onset before 30 months, impaired social development, delayed and deviant language development, and insistence on sameness. He described each of these behavioral features in detail.

The term 'autism' as a childhood condition did not appear in the international classification systems until more than 20 years after Kanner's first publication. The first mention was as a subgroup of the schizophrenias, in ICD-8 [World Health Organisation, 1967]. A major change in the concept of childhood autism was evident in DSM-III [American Psychiatric Association, 1980]. This introduced the term 'Pervasive developmental disorders' (PDD) as a general category, thus acknowledging the shift in the concept of autism from a psychiatric to a developmental disorder. Brief diagnostic criteria were given for two subgroups, namely 'infantile autism' with onset before 30 months, and 'childhood onset pervasive developmental disorder' with onset after 30 months but before 12 years.

Another influence in the field was the work of Asperger [1944] which, as noted above, was not well known in English speaking countries until the 1980s. The children he described made inappropriate social approaches. They had good grammar and vocabulary, but used this only to talk about a narrow range, unique to each child, of special interests. They were usually of average or high intelligence but often had specific learning disorders. They were socially and often physically clumsy and inept. Asperger believed his syndrome to be different from Kanner's autism. This is still debated, but most authors now consider it to be part of an autistic spectrum [Frith, 1991]. As a result of studies they carried out in the 1970s, and their interest in Asperger's work, Wing and Gould [1979] developed the concept of the spectrum of autistic disorders, the essential features of which were a triad of impairments of social interaction, communication and imagination, the last being replaced by a narrow range of interests or activities. These are familiar

Table 2. Age Specific Rates per 10,000 by Criteria Used—Means and Ranges (Includes Only Studies Giving Rates for Autism Separately From Other Autistic Spectrum Disorders)

Criteria	No. of studies of autism	Mean rate for autism	Range of rates	No. of studies of other ASD	Range of rates
Kanner	6	3.9	0.7–5.0	—	—
DSM-III	—	—	—	1	2.4
Rutter	3	7.0	1.9–16.0	1	2.6
DSM-III	9	7.0	1.2–15.5	5	1.9–5.2
DSM-III-R	3	8.6	7.2–10.1	1	3.2
DSM-IV/ICD-10	14	21.0	3.8–60.0	10	1.4–58.0
Triad*	—	—	—	2	14.3–15.7
Gillberg**	—	—	—	1	60.0

*Triad of impairments [Wing and Gould, 1979]. The rates are for children with IQ < 70.
**Gillberg's criteria for Asperger syndrome—first version [Ehlers and Gillberg, 1993]. The rate is for children of all levels of IQ.

features appearing in virtually all sets of criteria. The essential point of the spectrum concept was that each of the elements of the triad could occur in widely varying degrees of severity and in many different manifestations. For example, social impairment could be shown as passivity in social interaction or as active but inappropriate and repetitive approaches to others, not just aloofness as in Kanner's syndrome.

The concept of a spectrum began to be seen in the revised version of DSM-III, referred to as DSM-III-R [American Psychiatric Association, 1987]. This kept the general category of PDD but the subgroups were labelled 'autistic disorder' and 'pervasive developmental disorder not otherwise specified.' The definitions of the subgroups differed from those in DSM-III and were given as formal diagnostic criteria covering a range of relevant features of behavior. The age of onset criterion was no longer included. The fourth version, DSM-IV [American Psychiatric Association, 1994], also retained the overall category of PDD but introduced new subgroups. These were 'autistic disorder, Rett's disorder, childhood disintegrative disorder,' and, for the first time, Asperger's syndrome, referred to as Asperger's disorder. There was also a subgroup for 'pervasive developmental disorder not otherwise specified' (PDD NOS). Detailed criteria for the subgroups that could be used in research were given. Those for autistic disorder now included onset before 36 months. The 10th revision of the International Classification of Diseases, ICD-10, had closely similar subgroups and research criteria [World Health Organisation, 1993].

Effects of diagnostic criteria used in prevalence studies

Given the nature of the diagnostic criteria for different subgroups of autistic spectrum disorders, there can be no doubt that there were variations in the ways in which these were interpreted by different research workers. The most careful operational definitions, and training in their use, could not completely remove these differences. Nor can the degree to which they affected the results be measured. It is very possible that the rules dividing autistic disorder, however defined, from other autistic spectrum disorders, were applied differently by different investigators, which may explain some of the variations shown in Table 1. Particular problems arise in relation to autistic disorder and Asperger's syndrome. DSM-IV/ICD-10 rules specify, for Asperger's syndrome, age-appropriate development of language, adaptive skills and curiosity up to three years. If these criteria are applied strictly, they diagnose Asperger's syndrome significantly less and autistic disorder significantly more often than Gillberg's criteria [Gillberg and Gillberg, 2001], which are more closely based on Asperger's own descriptions [Leekam et al., 2000]. This is relevant to Fombonne's [2001] observation that in all the studies he surveyed the rates for Asperger's syndrome were lower than those for autistic disorder. These problems add to the difficulty of interpreting data from the published epidemiological studies. Even if the studies purport to count autistic spectrum disorders, it is not always clear what clinical pictures were included.

Table 2 shows the numbers of studies that purportedly used each of the different criteria for autistic spectrum disorders. The ranges of the rates for each set of criteria are also given. The column labelled 'autism' covers the following: early infantile autism, as defined by Kanner and Eisenberg [1956]; infantile autism as in DSM-III; autistic disorder as in DSM-III-R or DSM-IV; childhood autism as defined by Rutter [1978] or as in ICD-10. (As noted above, the DSM-IV and ICD-10 criteria are virtually identical.) The column labelled 'other ASD' (other autistic spectrum disorders) covers some or all of the other subgroups defined by the different sets of criteria. These differ widely in different studies; for example, some exclude Asperger's or Rett's syndrome. For this reason, only the mean rates for 'autism' (however defined) are given.

As can be seen from Table 2, the mean rates for studies using Kanner and Eisenberg's criteria were the lowest of all. The combination of social aloofness and elaborate repetitive routines is comparatively rare because aloofness is strongly associated with severe or profound mental retardation (1Q below 35). Elaborate rituals and routines, on the other hand, require the cognitive ability to organise aspects of the environment, such as using objects to make complex repetitive patterns, or insisting on lengthy bedtime routines [Wing and Gould, 1979]. Four studies, one early one in Camberwell (4) and three later ones in, respectively, Mölnlycke, (24), Karlstad (28), and Northern Finland (32) applied both Kanner's criteria and ICD-10 childhood autism criteria to the same children. In the Camberwell study, ICD-10 criteria were applied retrospectively to the original data. All children fitting Kanner's criteria also fitted the DSM-IV/ICD-10 criteria but, in each study, the numbers fitting Kanner's syndrome were markedly lower than those fitting DSM-IV/ICD-10 childhood autism. The proportions with Kanner's syndrome ranged from 33 to 45 % of all those diagnosed as having DSM-IV/ICD-10 childhood autism in the four studies. The mean rates for studies using Rutter's criteria or DSM-III were just under double those for Kanner's syndrome. These systems were similar to each other and neither insisted that the repetitive activities had to be elaborate. Both raised the upper limit of age of onset to 30 months.

DSM-III-R was used for only three studies. The mean rate was just over double that for Kanner's syndrome. The highest rates, with a mean over five times that for Kanner's syndrome, were found in studies using DSM-IV/ICD-10. Both DSM-III-R and DSM-IV/ICD-10 allowed for a wide range of types of social and communication impairment and of repetitive activities even within the subgroup of autistic disorder. Unlike Kanner's and Rutter's criteria, DSM-III-R and DSM-IV/ICD-10 did not insist on

language delay or deviance as long as some other type of communication impairment was present, such as poor intonation or inappropriate use of speech in relation to the social context. Volkmar et al. [1992], in a field trial carried out before DSM-IV was published, found that DSM-III-R, in contrast to ICD-10, was significantly over-inclusive when compared with clinicians' diagnoses. The authors suggested that one reason why autistic disorder in ICD-10 was more specific was that ICD-10 included a number of other subgroups in which individuals could be classified, compared with only one, PDD-NOS, in DSM-III-R. It is not likely that the lack of an upper limit for age of onset in DSM-III-R was a significant factor. Volkmar et al. [1985] found that only 5 children out of 129 (4 %) diagnosed as having PDD using DSM-III apparently had an age of onset after 30 months. In the light of these findings, it appears paradoxical that the highest prevalence rates for autism have been found using DSM-IV/ICD-10. Taking this together with the very wide range of rates associated with each set of criteria, it is evident that other factors in addition to the definitions of criteria must have been involved in order to explain the overall rise in rates.

Differences in Methods Used in Studies

Differences among the studies included variations in the sizes and types of target populations and methods of identifying cases [Fombonne et al., 2001].

Size of target populations

Honda et al. [1996] examined 18 prevalence studies published at the time in the English language or with English summaries (all included in Table 1). They found that rates for autism (plus other autistic conditions if reported) of over 10.0 per 10,000 were reported significantly more often in studies covering target populations of less than 50,000. The same analysis for the larger number of studies reported here gave a similar result. Seventeen of the studies using target populations under 50,000 found age specific rates for autistic spectrum disorders (however defined) above 10 per 10,000 and only one reported a rate of under 10. In contrast, only six out of 19 using larger populations found rates over 10 per 10,000 (Chi square = 15.5, df l, $P < 0.001$). The five studies with target populations under 10,000 (22, 24, 27, 28, 33), all using DSM-IV/ICD-10 criteria, gave rates per 10,000 for autistic spectrum disorders ranging from 21 to 120. As noted by Honda et al., studies of large populations give results with smaller confidence intervals, but make it much more difficult to be sure of finding all eligible children. Accurate case-finding is much easier with small target populations.

Proportions of immigrants in the study populations

A significantly higher prevalence rate for autism was reported among children of first generation immigrant parents in the studies in Camberwell (4), Gothenburg (20) and Mölnlycke (24). The study in Ibaraki (15) showed a higher prevalence of autism among children with parents who had moved to the area from other parts of Japan. However, the six other studies in which this issue was considered (17, 25, 29, 31, 33, 34) found no evidence to support the hypothesis of a raised prevalence in children with immigrant parents. All the studies listed in Table 1 were carried out in Europe, North America, or Japan. The published studies do not provide evidence for or against the possibility that particular regions, countries, or areas within countries, differ significantly in their prevalence rates. As noted below, the marked tendency for studies in Japan to find higher rates is probably due to the method of case-finding.

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Methods of case-finding

The authors of almost all of the studies in Table 1 used medical and/or educational agencies to find eligible children. The method of case-finding that generally produced the highest rates was close involvement with the routine developmental checks for pre-school children. Eight of the 15 rates for autism of over 10.0 per 10,000 were based on repeated developmental checks (13, 15, 18, 22, 24, 27, 30, 35) compared with none of the 20 rates below this level (Chi square = 13.83, df 1 $P < 0.001$). Japanese researchers in particular found this approach particularly useful because around 90% of Japanese children attend for these developmental examinations [Honda et al., 1996]. Five of the eight studies using this method (13, 15, 18, 22, 27) were carried out in Japan. Assessments for autistic spectrum conditions were integrated with the routine examinations, and repeated checks carried out over several years. As noted above, the highest rate of all was found from detailed examination of a small target population (28). The lowest rate (0.7) was found in Wisconsin (3). It was one of the earliest studies, published in 1970 before diagnostic criteria for autism appeared in the ICD or DSM classification systems. The rate was based upon computer printouts of clinical and demographic details of children known to agencies throughout the state who had been given a diagnosis of childhood schizophrenia using DSM-II criteria. The author used the records to rediagnose some as having autism based on Kanner's criteria and the absence of any evidence of 'organicity.' None was seen by the author. All these factors combined to give a very low rate for 'classic autism.'

Increasing Awareness Among Parents, Professionals and the General Public of the Existence of Autistic Spectrum Disorders

Until the 1960s there was little general interest in or awareness of autistic spectrum disorders. Then, as noted in the introduction, the development of voluntary associations of parents, later to include interested professionals, began in the USA and the UK early in the 1960s. Their aims were to push for educational and treatment services for their children and to encourage research. They were followed over the ensuing years by associations in other countries throughout the world. These associations were energetic in ensuring publicity concerning the children and their needs through all the media. Professional interest was stimulated by the development, also beginning in the USA and UK in the 1960s, of scientific investigation into the nature of autism [for example, Lotter, 1966; Hermelin and O'Connor, 1970; Rutter, 1970; De Myer, 1975; Schopler and Reichler, 1976; Rutter, 1978]. Awareness of the widening of the criteria for autistic conditions and the concept of Asperger's syndrome has also grown. There has been a profound change since the days when autism was considered to be a rare condition, to be diagnosed only by using Kanner's strict criteria. The appearance of various methods of eliciting the diagnostic criteria for autistic disorders has contributed to the greater will-

ingness of clinicians to diagnose autism and autistic disorders. In the 1990s, two diagnostic interview schedules for obtaining information on developmental history and behavior patterns were published. These were the Autism Diagnostic Interview- Revised (ADI-R) [Lord et al., 1994]; and the Diagnostic Interview for Social and Communication Disorders (DISCO) [Wing, et al., 2001]. For each of these schedules, training courses have been organised for professional workers, thus helping to spread knowledge in the field. The diagnostic process should also include systematic methods of observation and assessment, such as the Autism Observation Diagnostic Schedule (ADOS) [Lord et al., 2000].

From the end of the 1990s public attention has been brought to bear on autistic conditions through media publicity concerning a suggested link between autism and vaccination with mumps, measles and rubella (MMR) vaccine or with other childhood vaccines containing mercury in a preservative. These possibilities have had a powerful influence in making people aware of the existence of autistic conditions although current scientific evidence does not support either hypothesis. This problem will be further discussed below.

Recognition That Autistic Disorders Can Be Associated With Other Conditions

Severe or profound mental retardation and other developmental or physical disorders

Kanner [1943] at first believed that children with his syndrome were of potentially normal intelligence. Scientific studies that measured intellectual ability showed that autism and mental retardation of all levels could and often did co-exist [Lotter, 1966; Rutter, 1970; Wing and Gould, 1979]. It took time for this fact to affect clinical practice but awareness has increased over the years. In 1980 Shah et al. [1982] carried out a study of an institution for adults with mental retardation before it was finally closed. At the time of the study, the years of birth of the residents ranged from 1880 to 1964. The research team assessed the 893 residents and found that 339 (38%) were socially impaired as in autistic spectrum disorders—that is, they were aloof, passive or active but odd in social interaction. Out of all those with social impairment 134 (40%) had classic Kanner's autism [Shah et al., 1982]. Only a few of the youngest residents had previously been diagnosed as having autism. It is not possible to make any calculation of population prevalence rates from these results but they do show that many adults with undiagnosed autistic conditions were to be found in mental retardation institutions in the UK. An unpublished follow-up study has shown that those who are surviving are living in the small residences for adults with mental retardation that have replaced the institutions.

In the UK, until the 1970s, the general tendency was to label as 'mentally retarded' or 'maladjusted' children who were too learning disabled or too behaviorally disturbed to fit into mainstream school. Few of these children were assessed in pediatric clinics or given any other diagnosis. For example, out of the 50 school age children in the Camberwell study (4) diagnosed by the research workers as having autistic spectrum disorders, in 1971 only seven (14%) were known to the educational and school medical services as having autism. Six of the seven fitted Kanner and Eisenberg's [1956] diagnostic criteria. Seven other children were diagnosed by the research team with this type of autism but they were not recorded by the services as having any type of autistic condition. This meant that just over half (57%) of the children aged over 5 years with the most classic form of autism had not been diagnosed. Only one of the remaining 36 school-age children with other spectrum disorders was recorded as having autism [Wing et al., 1976]. The rest of the 50 school-age children were recorded as mildly or severely mentally retarded or, in a few cases, 'maladjusted.' During the 1970s, child development centers began to be set up in the UK. More and more children were referred for detailed developmental assessments and pediatricians began to develop interest and expertise in the field of autistic spectrum disorders. This change was accelerated in the 1980s when autistic conditions were classified in the DSM, and later in the ICD systems, as disorders of development caused by brain dysfunction, instead of being regarded as rare psychiatric conditions.

The paper by Croen et al. [2001] (34) suggests that awareness of the coexistence of autism and mental retardation is still growing. Statistics from the Department of Developmental Services (DDS) of the California Health and Human Services Agency showed a marked increase in the numbers of persons with autism entering the official counting system annually over the previous 11 years. Croen et al., using DDS data, examined the rates of DSM-III-R or DSM-IV autistic disorder (depending when the diagnosis was made) and found an overall rate for the 11 years studied of 11.0 per 10,000. The annual prevalence rates showed a more or less steady increase from 5.78 in 1987 to 14.89 in 1994. However, the annual prevalence rates for the same years for mental retardation of unknown cause showed a more or less steady *decrease* from 28.76 in 1987 to 19.52 in 1994. The rise in absolute numbers of recorded cases of autism has continued since 1994 but their significance cannot be evaluated without further analyses similar to that in the Croen et al. paper. Gilberg et al. [1991] observed that the increase in rates found over the three studies in Gothenburg (9, 11, 20) was due partly to better detection of autism among children with severe mental retardation as well as among those with intelligence in the average or high range.

Another of Kanner's beliefs that probably affected diagnostic practice in the early years was that autism was a unique condition, separate from all other childhood disorders. It is now recognised that autistic spectrum disorders can occur together with any other developmental or physical disability. Epileptic fits are commonly associated [Rutter, 1970]. Language disorders, especially affecting semantics and pragmatics [Brook and Bowler, 1992; Rapin, 1997] and motor co-ordination problems [Smith, 2000] are important aspects of the pattern of disabilities in autistic disorders and can cause diagnostic confusion if the underlying social impairment is not recognised [Gillberg and Billsted, 2000]. Research is now being undertaken on the association of autism or some features of autism with a range of identifiable genetic abnormalities including Fragile X [Turk and Graham, 1997], Turner's syndrome [Creswell and Skuse, 1999], tuberous sclerosis [Hunt and Dennis, 1987], Tourette's syndrome [Kadesjo and Gillberg, 2000] and Down's syndrome [Howlin et al., 1995]. Howlin and her co-authors observed that, because of the stereotyped view of children with Down's syndrome as very sociable and outgoing, the presence of autism, which occurs in around 10% of those with the syndrome, has often not been diagnosed.

Average or high intellectual ability

As noted above, Asperger's work was hardly known in English speaking countries until the 1980s. Most of the children he described had average or high intellectual ability, although a minority had mental retardation. Spreading knowledge of Asperger's syndrome heightened awareness that autistic conditions, even classic Kanner's autism, could be found in chil-

dren and adults of high ability. This shift of emphasis allowed the inclusion in the spectrum of children with the more subtle as well as those with the most obvious features of autism [Denckla, 1986]. Wolff [1995] described a group of adults whom she had followed up from childhood when they had subtle signs of autistic spectrum disorder. She referred to them as having 'schizoid personality disorder of childhood' but, in her book written in 1995, she acknowledged that their pattern of skills, disabilities and behavior fitted into the most able end of the whole autistic spectrum. This work added yet another factor in the widening of the concept of the spectrum. Wolff found that the future outlook for this group was, on the whole, good, most becoming independent as adults and some being high achievers in their work. A minority had a history of psychiatric conditions, alcohol or drug problems or delinquency.

Psychiatric disorders of any type

Before and for years after Kanner's first paper on his syndrome, autistic conditions were often diagnosed as childhood schizophrenia. The studies of Kolvin and his colleagues [Kolvin, 1971] were important in clarifying the differences between these diagnoses. It is likely that, in the past, while some adults with autism were diagnosed as having mental retardation and placed in the relevant institutions, others had a life-long label of schizophrenia and lived in institutions for the mentally ill. No studies of the prevalence of autistic conditions among residents in such institutions before they were closed had been published. Ryan [1992] suggested that some individuals diagnosed as having 'treatment resistant' mental illnesses such as schizophrenia, bipolar disorder or obsessive-compulsive disorder may have had Asperger's syndrome. Wing and Shah [2000] found that 17% of people referred to a specialist center and diagnosed as having an autistic spectrum disorder when aged 15 or over had marked catatonic features. Some had previously been diagnosed as having catatonic schizophrenia because the possibility of autism had not been considered. Tantam [1988] studied 60 adults who had been referred to a psychiatrist because of lifelong social isolation and conspicuous eccentricity. They had had a variety of psychiatric diagnoses, including both neuroses and psychoses. When Tantam examined their histories in detail he found that 46 of the 60 fitted the criteria for autistic disorder or Asperger's syndrome. Nylander and Gillberg [2001] found that 16 out of 499 adults (3.2%) attending a treatment center for psychiatric disorders had an autistic spectrum disorder, almost all not previously diagnosed. The findings were similar to those of studies of special hospitals for mentally ill offenders in England [Scragg and Shah, 1994; Hare et al., 1999]. Bejerot et al. [2001] found that 20% of 64 individuals diagnosed with obsessive-compulsive disorder had marked autistic traits. It is not possible to calculate the prevalence of psychiatric disorders among those with autistic spectrum disorders from these findings nor, conversely to calculate the proportion of those with autistic spectrum disorders among all those with psychiatric conditions. However, it is clear that, in the past, some individuals with autistic disorders have been misdiagnosed as having a psychiatric illness and that this mistake still occurs, though it is to be hoped that it is becoming less common. It would be interesting to know the prevalence of autistic spectrum disorders among adults living in all kinds of accommodation for the unemployed and the homeless, including those living on the streets, but no studies of this kind have been published.

Development of Specialist Services

Two different trends in service provision in the UK have affected patterns of referrals and the numbers of children and adults with autistic spectrum disorders seen by different professionals. On the one hand, the numbers of large institutions for people with mental retardation and special schools for children with mild mental retardation, specific learning disabilities and/or disturbed behavior have gradually diminished since the late 1970s. As discussed above, in the past, children and adults, including some with autistic spectrum disorders, would have been admitted to these services because of their learning or behavior problems without having any specialised diagnostic assessment. On the other hand, all kinds of specialist provision for children and adults with autistic spectrum disorders have been developed since the 1970s. They include diagnostic services for autistic spectrum disorders and related conditions, family support services, special schools, and special classes or individual support within mainstream schools, residential homes for adults, occupational services, specialised leisure opportunities, and social training groups for more able people with autistic conditions [Wing, 2001]. Although there is still a long way to go before provision is anywhere near adequate for everyone involved, progress has been made and is continuing. The improvement in and increasing availability of services over time has made parents more willing to think of the possibility of an autistic spectrum disorder if they are worried about their child's development and more willing to accept such a diagnosis. Professionals are also more likely to be willing to make a diagnosis of an autistic condition if they know that it will lead to appropriate help for the child or adult and the family concerned.

Administrative decisions can have an effect on the level of awareness of particular conditions. In the USA, in 1991, autism was included for the first time in the Individuals with Disabilities Education Act (IDEA). This was around the time that the numbers of children diagnosed as having autism began to rise, so it seems likely that this Act contributed to the increase. However, the same dramatic rise in rates of diagnosis has been seen in the UK where, with the exception of Scotland, there is no requirement for education authorities to record the numbers of children with autistic spectrum disorders.

Possible Causes and Relation to Age of Onset

The causes of autism are relevant to the problem of age of onset and possible changes in incidence and prevalence. There is strong evidence from twin studies that genetic factors are of major importance in the etiology of over 90 % of cases of autistic disorder diagnosed according to DSM-IV [Rutter, 2000]. It appears that several genes are likely to be involved. Studies of families of children with autistic disorder suggest that genetic factors are also important in relation to the wider autistic spectrum [Bolton et al., 1994]. Asperger [1944; 1991] observed that traits related to his syndrome were often seen in the parents of the children concerned. In their review, Gillberg and Colman [1996] found that estimates made by different workers of the proportions of children with autistic disorder who have medical conditions that possibly have caused the autism vary from around 11 to 37 %. The proportions were related to the intensity of the medical investigations. (Although epilepsy is common in people with autistic spectrum disorders, affecting one quarter or more of those with typical autistic disorder [Rutter, 1970], it was not included as a possible causal condition. It was considered to be an additional effect of whatever brain dysfunction has led to the autism.) The percentages with diagnosable medical conditions were higher for those with severe or profound mental retardation. The proportions for other autistic spectrum disorders varied from 12 to 53 %. However, the great majority of the medical conditions listed were also genetic, such as tuberous sclerosis, or pre-

natal in origin, such as maternal rubella. There are only a few reported examples of typical autistic behavior beginning at some time after birth following, for example, herpes simplex encephalitis [Gillberg, 1986]. These findings suggest that, in most cases, the basic pathology underlying autistic spectrum disorders is present from before birth.

There is a delay of varying length before parents become aware that their child is not developing as expected. Studies of home videos taken in the first year of life of children later diagnosed as autistic showed subtle symptoms of autism that could be reliably identified by the researchers [Baranek, 1999; Werner et al., 2000]. Baranek noted that caregivers used strategies to compensate for their children's unresponsiveness before they reported any autistic symptoms. Volkmar et al. [1985] found that only 4% of 129 children and adults diagnosed as having pervasive developmental disorders were reported to have had an onset after 30 months and they were behaviorally indistinguishable from the rest of the group. Some parents of children with autistic spectrum disorders reported that their children had begun to develop some limited speech (sometimes mainly echolalia) and then stopped speaking during their second or third year.

Although this picture has long been known, it has recently been referred to as 'regressive autism' (see below in connection with MMR vaccine). In a comparative study, Fombonne and Chakrabarti [2001] found that children with this history had no other developmental or clinical characteristics that distinguished them from other children with autistic disorder. It is certainly possible that many of those with 'regressive autism' had had the basic pathology underlying autism from birth. 'Regressive autism' is not the same as the condition known in DSM-IV/ICD-10 as 'childhood disintegrative disorder,' in which there is good evidence of completely normal development for at least two years and then a catastrophic loss of self care and other skills. This has a prevalence of less than 1 per 10,000 and there is no evidence of any increase in this condition [Fombonne and Chakrabarti, 2001]. As noted previously, all the studies of incidence have shown a steady rise year on year but these have used the year in which the children were *diagnosed* as the year of onset. Howlin and Asgharian [1999] found that, although parents had begun to be worried much earlier, the average age when a diagnosis was confirmed by professionals was 5.5 years for autism and 11.0 years for Asperger's syndrome. Volkmar et al. [1985] pointed out that the age of diagnosis is more appropriately called the age of recognition and should not be assumed to be the real age of onset. Increasing awareness and changes in diagnostic practice leading to a continuing trend for more and earlier diagnoses would affect the results of incidence studies that use age of diagnosis as age of onset, making it appear that incidence was rising.

Genetic factors alone are very unlikely to account for a real rise in rates that appears to have occurred so rapidly and continuously year on year. If there is a real rise that is continuing, environmental factors must be involved. Many suggestions have been made concerning possible causes, including constituents of the diet, environmental pollutants, antibiotics, allergies, vaccines, and traces of neurotoxins such as mercury present in preservatives used for some vaccines (though not in the MMR vaccine now used in the UK), but none has as yet been scientifically validated. The current public concern that the combined measles, mumps and rubella (MMR) vaccine is responsible for the observed increase in autistic conditions arises from the work of one particular group of researchers [Wakefield et al., 1998; Wakefield and Montgomery, 2000]. These workers examined children with autistic spectrum disorders referred to a pediatric gastroenterologist because of gastrointestinal symptoms such as chronic constipation. They described what they considered to be a particular form of inflammatory bowel disease. They put forward the hypothesis that this was due to MMR vaccination, which was causing a new variant of 'regressive' autism and that the observed rise in rates of autistic disorders was related to this new condition. But, as noted above, there is no evidence that 'regressive autism' differs from other forms of autistic disorder. Wakefield et al. [1998] did not examine children with autistic spectrum disorders who did not have gastrointestinal problems, nor did they examine children with severe chronic constipation without any form of autism. The four incidence studies discussed above [Taylor et al., 1999; Powell et al., 2000; Kaye et al., 2001; Dales et al., 2001] were designed to examine whether the introduction of MMR affected the annual incidence of autistic disorder and other autistic spectrum conditions. As noted previously, all found a steady rise year by year but the slopes of the graphs were unaffected by the introduction of MMR. This was considered to be strong evidence that the triple vaccine was not causing the observed rise in the incidence of autistic conditions. The studies did not reveal the reasons for the annual rises but the authors suggested that increasing awareness of autism among parents and professionals was an important factor. Chen and De Stefano [1998] pointed out the lack of evidence from large databases of any significant link between MMR and chronic bowel or behavior problems. Fombonne and Chakrabarti [2001] examined epidemiological data concerning children with autistic spectrum disorders, some diagnosed before MMR vaccination and some after. They found no evidence to support a distinct syndrome of MMR-induced autism or of 'autistic enterocolitis.'

There is strong evidence from twin studies that genetic factors are of major importance in the etiology of over 90 % of cases of autistic disorder diagnosed according to DSM-IV.

It remains a possibility that MMR vaccination precipitates autism in a small number of children who are vulnerable, perhaps because of genetic loading that would otherwise be insufficient to produce overt autistic disorder. The numbers would be too small to affect the rates found in the incidence studies. All types of immunisation carry a very small risk of adverse effects but this has to be balanced against the very much higher risk of death or severe disability from the illnesses against which the vaccines provide protection. Some parents are demanding that their children are given each of the MMR vaccines separately but this policy could allow infections to occur during the interval between each vaccination. In any case, there is no published evidence for or against the safety of this procedure as compared with giving the combined MMR.

Possible True Increase in Numbers

Even if MMR vaccination is not responsible for the increase in numbers, it is still possible that there is a real and

continuing rise. From the evidence presented, all the factors discussed above have contributed to the observed rise in rates of autistic spectrum disorders but there is no certainty as to how much of the rise they explain.

Three small but intensive studies, one in Camberwell and two in Gothenburg, are of particular interest when considering the possibility of a rise in rates from whatever cause. In the Camberwell study (4), because of the methodology used, only 3 children among those identified as having autistic spectrum disorders had an overall IQ of 70 or above. The rest, a total of 71 children aged under 15 years, had IQs under 70. The age specific prevalence for all autistic spectrum disorders based on these 71 children was 20 per 10,000 children aged under 15 years. A study in Gothenburg (10), also counting all autistic spectrum disorders in those with IQ under 70 using comparable diagnostic criteria, found 18 per 10,000, very close to the Camberwell finding. A later study in Gothenburg (38) examined children aged 13–17 with IQ of 70 and above in order to identify all with high-functioning autistic spectrum disorders. The age specific rates found were 36 per 10,000 for Asperger's syndrome diagnosed according to Gillberg's criteria [Ehlers and Gillberg, 1993] and 35 per 10,000 for children with social impairment but not the full criteria for Asperger's syndrome. Adding all the findings from the two Gothenburg studies gave a rate of 89 per 10,000 for all IQ levels and all autistic spectrum disorders.

The important point about these studies is that the Camberwell children were born in the years 1956–1970, the Gothenburg children with IQ under 70 were born 1966–1970 and those with IQ of 70 and above were born 1975–1983. They were born well before the time when rising numbers began to be reported, but the rate found for the whole IQ range and the whole spectrum was higher than all but two of the later studies—one in Tokyo (27) giving almost the same rate (90 per 10,000) and the other a very small scale study in Karlstad, Sweden (28) reporting a rate of 120 per 10,000.

Taken at face value, these findings suggest that, if the case finding had been as thorough and the same criteria for the whole autistic spectrum had been applied in all the early epidemiological studies, the rates found would have been higher even than most of those found recently. Furthermore, the annual rise in incidence found in the studies quoted above would be likely to continue until all children with autistic spectrum disorders were diagnosed in their pre-school years.

However, the findings have to be viewed with caution because the Camberwell and Gothenburg studies of children with IQ under 70 had total populations of children in the age range studied of approximately 35,000 and 24,000 respectively, and the Gothenburg study of the more able children had a total population in the age range of only 1519 children. Nevertheless, it should be noted that the studies were particularly intensive and all the children who were suspected of having an autistic spectrum disorder were examined in detail by the research workers.

The evidence discussed in this paper suggests that most, if not all, of the reported rise in incidence and prevalence is due to changes in diagnostic criteria and greater awareness among both parents and professionals. It remains an open question whether there has also been a genuine rise in the numbers of children with autistic spectrum disorders and, if so, how large it is and whether it is still continuing.

If the numbers of children with autistic spectrum disorders have always been so large, it is legitimate to ask where are all the adults with these conditions? Torben et al. [1999] followed up 341 children with autistic spectrum disorders for an average of 24 years, until they were aged 14–48 years and found a crude mortality rate of 3.5 %, almost double the expected rate for the general population of the same age.

The findings reported above concerning adults diagnosed as mentally retarded or mentally ill suggest that there is an unknown but possibly large number of adults with undiagnosed autistic spectrum disorders. Follow-up studies into adult life show the wide variation in outcome, from total dependence to full independence in adult life [Rutter, 1970; Gillberg, 1991; Larsen and Mouridsen, 1997; Howlin, 2000], but no studies of prevalence of autistic spectrum disorders in adults of any age have been published.

SUGGESTIONS FOR FUTURE RESEARCH

The evidence discussed in this paper suggests that most, if not all, of the reported rise in incidence and prevalence is due to changes in diagnostic criteria and greater awareness among both parents and professionals. It remains an open question whether there has also been a genuine rise in the numbers of children with autistic spectrum disorders and, if so, how large it is and whether it is still continuing. It is not possible to return to the past in order to apply current diagnostic criteria to all the early studies. The question of whether there are really more children with autistic spectrum disorders now than in the past cannot be answered definitively. Some studies can be done to examine aspects of the problems of incidence and prevalence. For example, the very strict criteria for Kanner's syndrome used in Lotter's study (1) could be applied to any new prevalence study in addition to the much wider criteria for autistic spectrum disorders now in use. This might indicate whether the rate for this particular subgroup has changed.

Research on MMR vaccination is important because parental concern has led to a drop in the numbers of children being vaccinated, with the consequent danger of epidemics of measles, mumps or rubella, all of which can cause long term disability or death in a small but significant number of children. A study in which children were assessed regularly for any features of autistic spectrum disorder from birth until five years of age, when the diagnosis should be clear for most participants, would be of interest. It would not be ethical to demand that some were given MMR in one dose, some as separate injections and some not vaccinated at all, but the parents' own decisions are likely to vary. It would be appropriate to ensure the inclusion of some siblings of children with autistic spectrum disorders because they are known to have a higher risk of developing such conditions. At least one large population cohort of children exists who were screened for communication impairment at 18 months [Baird et al., 2000]. They have been followed up and those with autism, developmental disorders without autism and those with typical development have been iden-

tified. A cohort of this kind would provide the basis for a detailed study of a potential causal role for MMR vaccination and abnormalities of the immune system. Examination of the hypotheses concerning MMR vaccine, autism and abnormalities in the bowel would be helpful but performing ileocolonoscopy in children for research purposes presents major ethical problems.

Establishing the age of onset for each child with an autistic spectrum disorder is crucial for studies of incidence and for examining the significance of any environmental factors operating after birth. It is also important for investigation of the so-called 'regressive autism.' In order to develop infant assessment methods, more work is needed on the behavior and abilities of babies who are later found to have an autistic spectrum disorder. Again, the siblings of children with autism would be appropriate participants but children who possibly have other types of developmental disorders and those likely to have typical development should also be included for comparison.

The problem underlying all research into the causes of autistic spectrum disorders is that the human and financial resources required to carry out any studies that can answer the questions considered in this paper would be immense. The numbers of participating infants and children required in order to be sure of finding enough with autistic spectrum disorders to give statistically meaningful results are daunting. This will continue to be the case unless and until reliable physical or psychological methods of identifying autistic conditions are found. The effort and cost of large-scale studies would be worthwhile if clear-cut results were obtained. However, regardless of the reasons, the prevalence of autistic spectrum disorders is much higher than the first epidemiological studies suggested and this has major implications for all the individuals, their families and the helping agencies involved.

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