

ORIGINAL ARTICLE

Prevalence of autism and parentally reported triggers in a north east London population

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Background: The recorded prevalence of autistic spectrum disorders has risen over recent decades. Measles, mumps and rubella (MMR) vaccine has been blamed, by causing a “new variant” form of “regressive autism” associated with “autistic enterocolitis”.

Aims: To estimate the prevalence of autism and to assess any changes in parental perception regarding the onset or causes of autism.

Methods and Results: A total of 567 children with autistic spectrum disorder in five districts in north east London were identified, born 1979–98. Reported autism, excluding the 94 cases of Asperger’s syndrome, increased by year of birth until 1992, since when prevalence has plateaued. This flattening off persisted after allowing for expected delay in diagnosis in more recent birth cohorts. The age at diagnosis of autistic spectrum disorder was estimated to have decreased per five year period since 1983, by 8.7% for childhood autism and by 11.0% for atypical autism. There was some evidence that MMR was more likely to be mentioned as a trigger after August 1997 than before.

Conclusions: The prevalence of autism, which was apparently rising from 1979 to 1992, reached a plateau from 1992 to 1996 at a rate of some 2.6 per 1000 live births. This levelling off, together with the reducing age at diagnosis, suggests that the earlier recorded rise in prevalence was not a real increase but was likely due to factors such as increased recognition, a greater willingness on the part of educationalists and families to accept the diagnostic label, and better recording systems. The proportion of parents attributing their child’s autism to MMR appears to have increased since August 1997.

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In a previous study in north east London,¹ which showed no epidemiological association between measles, mumps, and rubella (MMR) vaccination and autism, we described an apparent rise in the prevalence of autism by year of birth. Similar rises in reported rates of autism have been described elsewhere. Although much of this apparent rise is considered to relate to changes in diagnostic criteria, wider recognition of the disorders, a greater willingness on the part of educationalists and families to accept the label, and other factors,² there has been widespread concern, fuelled by media speculation, that MMR vaccine might be responsible. MMR vaccination has been incriminated by allegedly causing a “new variant” form of autism associated with bowel problems and regression.³ The existence of this postulated condition has been refuted.^{4,5}

We repeated our earlier study¹ after an interval of two and a half years, based on the same population and using the same methodology. One aim was to investigate the relation between MMR vaccination and bowel problems and regression (published elsewhere⁶). Other epidemiological aims, related to the continuing public concern about a possible role for MMR vaccine in the initiation of autism, were to determine whether the rise in reported prevalence by year of birth was continuing, to assess possible changes over time in the age at which the diagnosis of autism was made, and to document triggers for the onset of autism as reported by the parents before and after medical journal and media speculation about a possible link between MMR and “regressive autism”.³

METHODS

Five of the eight districts from the original survey were included. Three were country town/semi-rural districts, one was suburban, and the other inner-city. The other three districts (two country town/semi-rural, one suburban) had

undergone changes in personnel, management arrangements, and/or computer systems, which made them unsuitable for repeat study. The original eight districts were chosen for study as users of the Regional Interactive Child Health computing System (RICHS) special needs module. The remaining eight RICHS users in North East Thames did not use this module. The original eight districts and the five of those eight used for the present study provide a general population sample with a broad range of social and cultural characteristics.

Case identification and ascertainment

Records were identified during the year 2000 for all children in the study districts with a diagnosis of autistic spectrum disorder (ASD) born since 1979. An initial list of children was compiled from computer based disability registers (RICHS, or TotalCare). Relevant ICD-9 and ICD-10 codes were identified. This computer generated list was augmented by approaching consultant community paediatricians, special needs schools, and local child psychiatry teams for any additional cases. Some 90% of cases (range 88–96% for the five districts) were obtained from the computerised disability register (special needs module).

The diagnosis accepted was that of the principal clinician who had seen the child. Diagnostic validity was checked by matching symptoms recorded in the notes against ICD-10 criteria. Each record (usually comprising hospital, child development team, school, and health visitor notes) was systematically reviewed by one of two paediatric registrars. There was no direct patient, general practitioner, or other clinician contact.

Abbreviations: ASD, autistic spectrum disorder; MMR, measles, mumps, and rubella

Table 1 Description of the 567 children with autistic spectrum disorders

Variable	Childhood (n=278)	Atypical (n=195)	Asperger's (n=94)
Number overall (prevalence per 10000)*	278 (14.9)	195 (10.5)	94 (5.0)
Number male (%)	230 (83%)	158 (81%)	81 (86%)
Number with regression (%)†	75 (27%)	43 (23%)	2 (2%)
Number with bowel symptoms (%)	49 (18%)	32 (16%)	9 (10%)
Median age in months at regression (IQR)	18 (14–20)	20 (18–26)	40 (2 cases, at 15, 65)
Numbers	74	43	2
Median age in months at parental concern (IQR)	18 (14–24)	22 (16–30)	28 (20–42)
Numbers	262	179	85
Median age in months at provisional diagnosis (IQR)	35 (30–41)	44 (34–64)	74 (56–115)
Numbers	273	191	87
Median age in months at final diagnosis (IQR)	40 (34–48)	51 (40–78)	97 (70–125)
Numbers	266	171	89

*Prevalence was calculated using the subset of 429 children aged 5–14 at the end of 2000 and using 1996 mid-census denominator data for 5–14 year olds in the study area (186206).

†Regression status not available for four atypical cases and two Asperger's cases.

IQR, interquartile range.

Information recorded

The dates of provisional diagnosis, definitive diagnosis, age at first parental concern about the child's development, and age at developmental regression where a feature, were noted. Provisional diagnosis was defined as the first mention of the terms "autism" or "autistic spectrum" in any clinical record or letter. Definitive diagnosis was defined as when the principal clinician documented autism or Asperger's syndrome as a diagnosis.

Regression reflected mention in the notes of the word or a description of a loss of skills,^{5 6} even if transitory. Additional information was extracted from the case notes of children with possible regression to determine which skills were lost, at what age this had occurred, and possible triggers identified by the family, as well as when regression was first documented in the clinical notes and whether there had been any change in the history of regression.

General health concerns, including bowel problems persisting at least three months, were recorded. It is likely that any significant health problem would be documented as children with ASDs are often seen many times for diagnosis and then have regular review, usually at least annually.

Immunisation details were obtained from the local child health computer system (RICHS or TotalCare). This information was recorded independently of the clinical record; researchers assessing the clinical records were blind as to the vaccination dates.

To ensure consistency of data collection between researchers, 40 cases were assessed independently by both recorders or an independent observer; when the data recorded were compared on key variables, including age at first parental concern, age at regression, and age at provisional and final diagnosis, over 98% showed an exact match. Computerised data were double entered and validated, then anonymised. Ethical approval for the study was obtained from the relevant committees.

Analysis

Modelling of trends in autism by year of birth and age at diagnosis

These analyses used data from all cases of childhood and atypical autism, born between 1983 and 1999, who had a provisional diagnosis by the age of 10 years and before the end of December 1999. These restrictions reflected the likely under-ascertainment of cases born prior to 1983 and of cases diagnosed in the year 2000, as well as the large potential effect

of a few outliers diagnosed after the age of 10 years. Recorded age at provisional diagnosis was used in these analyses to provide the earliest age at which cases were identified as autism.

In order to obtain estimates of expected total cases (that is, including those not yet diagnosed for more recent years) the data were cross-classified by year of birth and age in years at diagnosis and analysed using Poisson regression with Excel and S-plus. The estimated number of cases in each cell (ϵ) was modelled by a function of the form $\epsilon(t,s) = \lambda(t) f(s|t)$, where $\lambda(t)$ represents the estimated number of births in year t diagnosed as autistic and $f(s|t)$ is the probability that a case has a provisional diagnosis at age s given that the case was born in year t . The age distribution was modelled using a gamma distribution with mean $\mu \exp(-\rho t)$ where ρ is the parameter which allows for an exponential change in the mean age at provisional diagnosis. The gamma distribution was then truncated to restrict age at diagnosis to be between 1 and 10 years. The gamma distribution was chosen because it provided a good fit to the positively skewed distribution of age at diagnosis. The function $\lambda(t)$ was modelled using linear, quadratic, and cubic polynomials. The linear model fits an exponential increase over time with $\lambda(t) = \exp(b_0 + b_1 t)$, the quadratic model with $\lambda(t) = \exp(b_0 + b_1 t + b_2 t^2)$ allows for a departure from an exponential increase, such as a flattening off and decline, the cubic model with $\lambda(t) = \exp(b_0 + b_1 t + b_2 t^2 + b_3 t^3)$ allows for a further change such as a decline, then a rise, then a decline.

Separate models as appropriate for the data, were fitted for the childhood and atypical cases and expected numbers by birth year were calculated allowing for the estimated number of cases yet to be diagnosed. The overall expected numbers were then calculated by adding up the expected numbers of childhood and atypical cases.

RESULTS

Descriptive

Table 1 shows details of the study population. A total of 567 children with ASD were identified, born between 1979 and 1998. Of these, 278 (49%) had childhood autism, 195 (34%) atypical autism, and 94 (17%) Asperger's syndrome.

A diagnosis of childhood autism could be ICD-10 confirmed from symptoms detailed in the clinical notes in 248 of 278 children (89%). For the other 30 there was insufficient information recorded, especially regarding social interaction; 17 of these cases had less than two social interaction symptoms documented. Four others presented after 3 years of age with

no symptoms recorded in the notes regarding their condition before the third birthday.

All but one child with atypical autism met the criteria for diagnosis, and 79 (84%) of the 94 children with Asperger's syndrome met either ICD-10 or Gilberg and Gilberg's 1989 criteria.⁷ The proportion of cases with the different types of autism was similar across the five study districts ($p = 0.32$).

Overall 83% of the cases were male, with no significant difference in this percentage by type of autism ($p = 0.56$). This sex difference is comparable with other published data.⁸

Some 188 children were documented as attending a special school; 180 were at a special unit and 140 were integrated into mainstream education. The remaining 59 children were preschool, had left school, or no school was mentioned in the notes. The percentage of children who were documented as having seen different health professionals varied; 466 of 473 (99%) of children with childhood or atypical autism were documented as having seen a developmental paediatrician, and 460 (97%) a speech and language therapist. Some 436 (92%) were recorded as having been seen by an educational psychologist and 200 (42%) had attended a specialist centre for assessment of autism. However, only 18 (6%) childhood cases, eight (4%) atypical cases, and one (1%) child with Asperger's syndrome were documented as having seen a specialist gastroenterologist. Bowel problems were reported in 17% of cases overall, with no significant difference by type of autism ($p = 0.18$). A detailed description of the bowel problems in this study population and the lack of a relation with MMR vaccination have been reported elsewhere.⁴

Using denominator data for children aged 5–14 in the five districts, prevalence rates were calculated for those cases aged 5–14 at the end of 2000 (table 1). These rates give an overall estimate for the prevalence of atypical and childhood autism of 19.3 per 10 000 children.

Median age at regression, parental concern, and provisional and final diagnosis were much higher in cases of Asperger's syndrome and slightly higher in those with atypical compared with childhood autism (table 1). Regression was reported in only two cases with Asperger's syndrome (2%), compared to 23% with atypical autism and 27% with childhood autism. Because of these, and other differences from childhood and atypical autism, Asperger cases were not included in subsequent analyses. There were some missing data regarding age at regression, first parental concern, and provisional and final diagnosis for all forms of autism. This reflected the difficulties of assigning accurate dates from sometimes limited clinical records. The missing data are unlikely to have had any effect on the number of cases of childhood and atypical autism identified before 2000 as we identified cases until the end of 2000 and the median gap between provisional and final diagnosis for childhood and atypical autism was about six months, and less in recent years.

The median age at provisional and final diagnosis was more than 15 months after first parental concern or regression onset. There was a tendency to preferential reporting to the nearest 6 months, at 12, 18, 24, 30, and 36 months. The median ages at provisional and final diagnosis are likely to underestimate the true average age at diagnosis because children born in recent years could only be included if they were diagnosed young. However, the modelling of trends in autism by year of birth and age at diagnosis allowed corrected estimates of age at diagnosis (see below).

Regression

Regression was reported in 118 cases (25%) with atypical and childhood autism. The age when regression began was available in 117. In 12 of these children developmental regression was not noted in the early record but only retrospectively, years later, as part of the parental statement for the child's special educational needs. More detailed information on skills loss was obtained for 106 of these 117

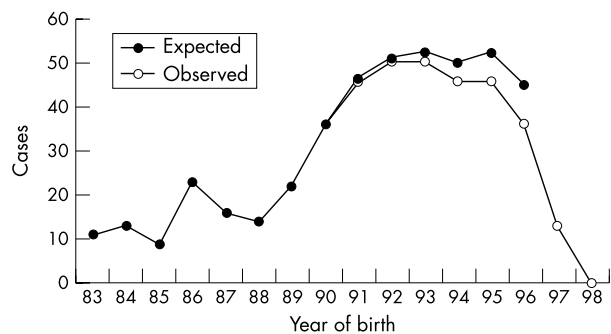


Figure 1 Observed and expected number of childhood or atypical autism cases diagnosed by the age of 10 years, by year of birth. (Expected number allows for the model estimate of the number of cases still to be diagnosed from 2000 onwards.)

children. Only one child regressed after 50 months—at 88 months. Reported skill losses with the regression included: 59 (56%) regression or loss of speech as the only symptom; eight (8%) behaviour alone; 32 (30%) both behaviour and language; and two (2%) motor regression. The type of regression was not specified for the other five children.

In 44 (42%) of the 106 children with detailed information on regression, a specific trigger was mentioned as a possible cause. The most common (13 children) was a household or social change such as the birth of a sibling, then vaccination (12 cases). Other triggers mentioned were: viral and bacterial infections ($n = 7$), seizures ($n = 7$), postsurgery ($n = 2$), and other causes ($n = 3$). The MMR vaccine was mentioned specifically in eight of the 12 cases where a vaccine was suspected. Although families would not have been directly asked about this possibility, this finding suggests that very few parents (less than 2% in this cohort) considered that MMR vaccine might have triggered their child's autism.

Widespread public concern about the possible relation between autism and MMR began in August 1997, with the pre-publication release of information about the Wakefield study,¹⁰ which attracted considerable and ongoing media attention. The date at which onset of developmental regression was first recorded in the notes was obtained for the 106 cases. After excluding unvaccinated cases and those vaccinated when aged over 24 months (of whom all but one were children vaccinated in the 1988–89 catch-up campaign), we found MMR was reported as the trigger in 6/30 (20.0%) post-August 1997 compared to 2/46 (4.3%) before August 1997 ($p = 0.052$).

From August 1997 the reported presence or timing of regression changed in 13 cases. For six of these, regression was mentioned for the first time after August 1997, even though many health professionals had seen these children before this date. In seven cases the recorded timing of onset of regression changed in relation to MMR: six closer, one further away.

Modelling of trends in autism by year of birth and age at diagnosis

Figure 1 shows the observed and expected number of cases by year of birth for childhood and atypical autism combined. This clearly illustrates that the recorded rise in cases by year of birth which occurred in children with autism born up to 1992 did not continue beyond that year. Figure 2 shows the separate data for childhood and atypical autism. The levelling after 1991 (with ≈ 50 new cases total per year) gives a prevalence of autism since then, for children age 5–14 years in this population, of some 2.6/1000 live births (childhood 1.5; atypical 1.1), using as the denominator Office of National Statistics mid-census population estimates. Confirming our previous finding⁴ there was no evidence of a step up in prevalence in the cohorts eligible to receive MMR vaccine in the second year of

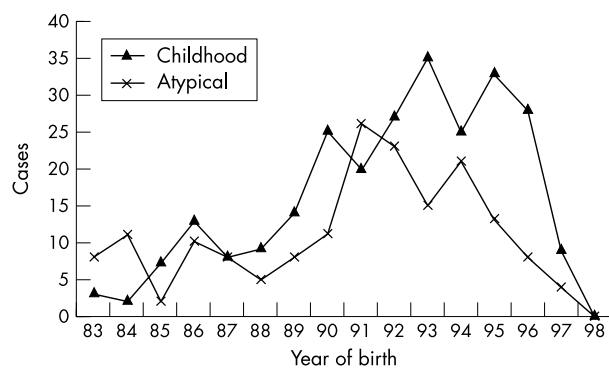


Figure 2 Observed numbers of children with childhood and with atypical autism diagnosed by the age of 10 years, by year of birth.

life—that is, those born from 1987. A step would be expected if MMR caused autism because, although some children born before 1987 did receive MMR, this was in a catch-up campaign and these children would have been vaccinated with MMR older than the usual age when autism becomes manifest. The catch-up campaign was not relevant in our population.¹⁰ Although data up until the end of 1999 were used in the model, expected results are only given up until the end of 1996. This is because the diagnosis was not usually made until after the age of 3 years for childhood and 4 years for atypical autism, which means there was limited data for birth years 1997–99 and extrapolation using polynomials is not usually accurate outside the range of the data.

For childhood autism, the best fitting model for the trend by birth year was a quadratic polynomial, which is due to the fact that the exponential apparent increase stopped after 1990. There was evidence of a reduction in the mean age at diagnosis over time, with an average 8.7% decrease every five years ($p = 0.021$). The overall average age at provisional diagnosis was 3.2 years. However, the average age was estimated to have reduced from 3.5 years in 1985 to 3.0 by 1995.

For atypical autism the best fitting model for the trend over time was a cubic polynomial, which is due to an initial apparent low prevalence, followed by an increase, then a levelling off and slight decrease. There was evidence of a reduction in the mean age at diagnosis over time, with an average 11.0% decrease every five years ($p = 0.04$). The overall average age at provisional diagnosis for atypical autism was 5.2 years; however, the average age was estimated to have fallen from 6.0 in 1985 to 4.9 by 1995.

DISCUSSION

We have shown a levelling off since the early 1990s in the previously rising recorded prevalence of autism. In our earlier study we showed an exponential increase in the number of reported cases by year of birth.¹ The additional two and a half years data in the present study suggests that the rise has stopped and that prevalence has reached a plateau. Prevalence estimates for combined childhood and atypical autism from 1992 to 1996 were steady at about 45–50 cases per birth year in this study population. We have also shown that age at diagnosis has decreased over time. This suggests that diagnostic practice has changed. Such change is likely to reflect, at least in part, a greater willingness by educationalists and parents to accept the diagnostic label, greater awareness of autism by health professionals, especially the considerable recent expansion of specialist paediatricians trained to recognise the conditions, changes in diagnostic criteria for autism, as well as better record systems.

We have also documented changes in the likelihood that parents would incriminate MMR as the trigger for their child's autistic state, particularly for any associated regression, since

1997, the year when publicity about a possible link between MMR vaccination and autism became widespread. In some cases, we found that the history of regression had changed since 1997 from that recorded earlier. These findings are likely to affect systematically the validity of the self controlled case series analysis linking vaccination to age at regression in children with autism. We investigated this possibility and found such evidence of parental recall bias when reporting the onset of regression in relation to MMR vaccination since 1997.¹¹

Our prevalence rate for childhood autism of 14.9 per 10 000 for children age 5–14 years is comparable with the 16.8 reported by Chakrabarti and Fombonne¹² for Staffordshire children. Both figures are rather lower than the 30.8 (which included some cases of Asperger's syndrome) in south east England, reported by Baird and colleagues.¹³ Our rates (10.5 per 10 000) for atypical autism and (5.0) for Asperger's syndrome are likely to be an underestimate, as children with autistic spectrum disorder known to the health services are likely to be at the more severe end of the spectrum and more likely to present in the preschool period. It is likely that some of our atypical cases might actually be childhood autism; limitations in the available health records, especially regarding social interaction, made allocation difficult in some instances. Clinical notes were not always complete, but diagnoses could be verified in the majority of cases. The comparable results for childhood autism between our study and that in Stafford shows the potential effectiveness of health service disability registers as an epidemiological tool.

Developmental regression in autism is not a new finding. Lotter in 1966 described developmental setbacks, including speech loss, in 10 of 32 autistic children.⁶ Fombonne and Chakrabarti⁵ document reported rates of regression varying between 22% and 50%, in earlier published studies, with no evidence of a rising proportion in more recent case series. The proportion of children with autism who have regression is unclear.¹⁴ Children with autism often fail to develop normal social speech between the age of 12 and 18 months. Parents and even health workers may over-interpret as regression, the loss of infantile babbling which occurs in normal children as well as in children with autism during the same age span. True regression may occur in rather less than 20% of cases.⁵

In our study, a sizable minority of parents had an explanation for their child's "regressive loss of skills". Whether, as stated in many cases, these children were completely "normal" before the "regression", or were in fact subtly different, is a matter of conjecture. We found that the trigger for regression in some cases had changed after the publicity and media attention associated with, and preceding,⁹ the 1998 Wakefield *et al* paper.³ Before August 1997, parents incriminated trigger factors such as domestic stress, seizures, or viral illness. Post-1997, parents were more likely to attribute regression to vaccination, especially the MMR vaccine. Parents are usually very reliable historians regarding their child's early life. Sometimes, however, there is the possibility of recall bias, in some instances reflecting changing beliefs about causal relationships. An expert group convened by the Medicines Control Agency reviewed the records of 92 children with autism whose parents thought that MMR had caused or triggered their child's condition. In 36 (39%) there was evidence in the medical record that there had been concerns about the child's behaviour before the MMR vaccination. However, in only one (1%) of these cases did the parent recall this early concern.¹⁵

Davidovitch *et al* examined maternal perceptions in autism.¹⁶ They found that where children with autism had developmental regression, almost all mothers attributed specific causes for their child's autism, compared to only two thirds of mothers where children did not show regression.

They stated: "it is possible that regression is associated with a different perception of the condition by parents".

Our large population study, using robust methodology and analytical methods, has helped define the current epidemiology of the condition, confirming and extending earlier findings. The prevalence of autism appears to have stabilised. There is no evidence for a new phenotype of autism ("new variant", or "regressive autism").^{4,5} The claims that MMR vaccine is involved in the initiation of autism, and/or with regression, and/or with bowel problems associated with autism, are not supported by any credible scientific evidence, while there is compelling and increasing evidence showing no association. Further research is needed into the causes and best treatment of autism and related conditions. Our finding of changing histories with recall bias, will affect the interpretation of future epidemiological research in autism.

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