Autism spectrum conditions (ASC). From this, their prevalence estimate was 44.2 per 10 000, or 0.44%. This corresponds to 1 in 225. These estimates are clearly much higher than was the case in the past, where prevalence was traditionally estimated to be 4 in 10 000.

BEYOND COUNTING AND PREVALENCE ESTIMATES

Now we know that ASC are common. How should we understand their causes? Harrison and colleagues7 find that the 13–15 year old age group who would have received their MMR during the data collection phase were actually less numerous than the 4–10 year old age group, suggesting this high rate cannot be due to the MMR vaccine (since both age groups were exposed to the MMR). Instead they argue that these data suggest better recognised, better recording of cases, and growth of services.

In terms of causes, the consensus is that ASC have a genetic aetiology,7 which leads to altered brain development,5–11 affecting social and communication development and leading to the presence of unusual narrow interests and extreme repetitive behaviour. The model we can use involves multiple levels (see fig 1). In what follows I will elaborate on the two new ideas, shown in bold in the model.

HYPER-SYSTEMISING

A universal feature in the environment that the brain has to react to is change. There are two types of structured change:

- **Agentive change**: If an object change is perceived to be self-propelled, the brain interprets the object as an agent, with a goal. Such change cannot easily be predicted in any other way. To interpret agentive change, humans have specialised neurocognitive mechanisms, collectively referred to as the “empathising system”. The neural circuitry of empathising is now quite well mapped. Key brain areas involved in empathising include the amygdala, the orbito and medial frontal cortex, and the superior temporal sulcus.

- **Non-agentive change**: Any structured change that is not self-propelled is interpreted by the brain as a non-agentive change. “Structured” means non-random, for example that there is a precipitating event, or some other pattern. The brain does not deploy the empathising mechanisms to predict such change. Instead, the human brain engages in “systemising”, that is, it searches for structure (patterns, rules, regularities, periodicity) in data, to test if the changing data are part of a system. Systemising involves observation of input–operation–output relationships, leading to the identification of laws to predict that event x will occur with probability p. Some systems are 100% lawful (for example, an electrical light switch, or a mathematical formula) (see table 1).

![Figure 1](http://adc.bmj.com/)

<table>
<thead>
<tr>
<th>Assortative mating of two high systemisers (SM set at level 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific genotype</td>
</tr>
<tr>
<td>Altered brain structure and function</td>
</tr>
<tr>
<td>Hyper-systemising</td>
</tr>
<tr>
<td>Hypo-empathising</td>
</tr>
<tr>
<td>'Mindblindness' and social communication difficulties</td>
</tr>
<tr>
<td>Increased sensory sensitivity</td>
</tr>
<tr>
<td>Preference for 'simple' systems</td>
</tr>
<tr>
<td>Need for sameness (change-resistance)</td>
</tr>
<tr>
<td>Repetitive behaviour</td>
</tr>
<tr>
<td>Avoidance of 'complex' systems</td>
</tr>
<tr>
<td>Narrow interests and obsessions with systems</td>
</tr>
<tr>
<td>Reduced generalisation</td>
</tr>
<tr>
<td>Attempts to systemise the social world</td>
</tr>
<tr>
<td>Increased language delay and learning difficulties</td>
</tr>
</tbody>
</table>

PERSPECTIVES

1 High functioning autism (HFA) can be thought of as within one standard deviation of population mean IQ (that is, IQ of 85 or above); medium functioning autism (MFA) can be thought of as between one and three standard deviations below the population mean (that is, IQ of 55–84). Low functioning autism (LFA) can be thought of below this (that is, IQ of 54 or below).
The systemising mechanism (SM)

The hyper-systemising theory of ASC posits that human brains have a systemising mechanism (SM), and this is set at different levels in different individuals. In people with ASC, the SM is set too high. The SM is like a volume control. In people with ASC, the SM is set too high. The SM is set at the maximum setting (Level 8). Thus, people with HFA try to socialise or empathise by “hacking” (that is, systemising), and on the picture sequencing task, they perform above average on sequences that contain temporal or physical-causal information. People with MFA perform above average on the false photograph task. In LFA, their obsessions cluster in the domain of systems, such as watching electrical fans go round; and given a set of coloured counters, they show extreme “pattern imposition.”

Experimental evidence for hyper-systemising in AS includes the following: (i) people with AS score higher than average on the systemising quotient (SQ); (ii) people with AS perform a normal or high level on tests of intuitive physics or geometric analysis; (iii) people with AS can achieve extremely high levels in domains such as mathematics, physics, or computer science; and (iv) people with AS have an “exact mind” when it comes to art and show superior attention to detail. The higher the level of the SM, the less generalisation, since systemising involves identifying laws that might only apply to the current system under observation. At Level 7, one would expect some language delay, but this might only be a moderate (since someone whose SM is set at Level 7 can tolerate a little more or less language, or a higher or lower IQ, or differing degrees of mind blindness.)


---

### Table 1: An example of 100% lawful systems: (a) An electricity switch, and (b) a mathematical rule

<table>
<thead>
<tr>
<th>Input/Operation</th>
<th>Output</th>
<th>Operation and output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Input = switch position</td>
<td>Operation = switch change</td>
<td></td>
</tr>
<tr>
<td>Up</td>
<td>On</td>
<td></td>
</tr>
<tr>
<td>Down</td>
<td>Off</td>
<td></td>
</tr>
<tr>
<td>Input = number</td>
<td>Operation = add 2</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2: An example of systemising hydrangea colour

<table>
<thead>
<tr>
<th>Input (type of hydrangea)</th>
<th>Output (colour of hydrangea)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acidic</td>
<td>Neutral</td>
</tr>
<tr>
<td>Annabelle</td>
<td>White</td>
</tr>
<tr>
<td>Blauer prinz</td>
<td>White</td>
</tr>
<tr>
<td>Bouquet rose</td>
<td>Blue</td>
</tr>
<tr>
<td>Deutschland</td>
<td>Purple</td>
</tr>
<tr>
<td>Enziandom</td>
<td>Blue</td>
</tr>
</tbody>
</table>


Systemising involves recording input and output and deriving the rules how an operation changes the output.
variance in the way language is spoken and still see meaningful patterns). And the child’s mindblindness would be less than total. If the SM is set at Level 6, such an individual would be able to deal with systems that were slightly less lawful. This would therefore be expressed as only mild language delay, mild obsessions, mild delay in theory of mind, and stilted social behaviour, such as attempts at systemising social behaviour.

THE ASSORTATIVE MATING OF TWO HIGH SYSTEMISERS

The evidence for systemising being part of the phenotype for ASC includes the following: fathers and grandfathers of children with ASC are twice as likely to work in the occupation of engineering (a clear example of a systemising occupation), compared to men in the general population. The implication is that these fathers and grandfathers have their SM set higher than average (Level 4). Students in the natural sciences (engineering, mathematics, physics) have a higher number of relatives with autism than do students in the humanities. Mathematicians have a higher rate of AS compared to the general population, and so do their siblings.

The evidence that autism could be the genetic result of having too high systemisers as parents (assortative mating) includes the following: (a) both mothers and fathers of children with AS have been found to be strong in systemising on the Embedded Figures Test; (b) both mothers and fathers of children with autism or AS have increased rates of systemising occupations among their fathers; and (c) both mothers and fathers of children with autism show hyper-masculinised patterns of brain activity during a systemising task. Whether the current high rates of ASC simply reflect better recognition, growth of services, and widening of diagnostic categories to include AS, or also reflect the increased likelihood of two high systemisers having children, is a question for future research.

CONCLUSIONS

The core of autism spectrum conditions (ASC) is both a social deficit and what Kanner astutely observed and aptly named “need for sameness”.

Key behaviours that follow from extreme systemising include:

- Highly repetitive behaviour (e.g. producing a sequence of actions, sounds, or set phrases, or bouncing on a trampoline)
- Self-stimulation (e.g. a sequence of repetitive body-rocking, finger-flapping in a highly stereotyped manner, spinning oneself round and round)
- Repetitive events (e.g. spinning objects round and round, watching the cycles of the washing machine; spinning the wheels of a toy car)
- Preoccupation with fixed patterns or structure (e.g. lining things up in a strict sequence, electrical light switches being in either an ON or OFF position throughout the house)
- Prolonged fascination with systemisable change (e.g. sand falling through one’s fingers, light reflecting off a glass surface, playing the same video over and over again)
- Tantrums at change: as a means to return to predictable, systemisable input
- Need for sameness: to impose lack of change onto their world, to turn their world into a totally predictable environment, to make it systemisable
- Social withdrawal: since the social world is largely unsystemisable
- Narrow interests: in systems (e.g. types of planes)
- Mind blindness: since the social world is largely unsystemisable
- Attention to detail: the SM records each data point in case it is a relevant variable in a system
- Reduced generalisation: hyper-systemising means a reluctance to formulate a law until there has been sufficient data collection. This could also reduce IQ and breadth of knowledge
- Language delay: since other people’s spoken language varies every time it is heard, so it is hard to systemise
- Islets of ability: channelling attention into the minute detail of one lawful system (e.g. the script of a video, or prime numbers)

According to the hyper-systemising theory, ASC is the result of a normative systemising mechanism (SM)—the function of which is to serve as a change predicting mechanism—being set too high. This theory explains why people with autism prefer either no change, or appear “change resistant”. It also explains their preference for systems that change in highly lawful or predictable ways (such as mathematics, repetition, objects that spin, routine, music, machines, collections). Finally, it also explains why they become disabled when faced with systems characterised by “complex” or less lawful change (such as social behaviour, conversation, people’s emotions, or fiction), since these cannot be easily systemised.

While ASCs are disabling in the social world, hyper-systemising can lead to talent in areas that are systemisable. For many people with ASC, their hyper-systemising never moves beyond phase 1 (massive collection of facts and observations—lists of trains and their departure times, watching the spin-cycle of a washing machine), or phases 2 and 3 (massive repetition of behaviour—spinning a plate or the wheels of a toy car). But for those who go beyond phase 3 to identify a law or a pattern in the data (phases 4 and 5), this can constitute original insight. In this sense, it is likely that the genes for increased systemising have made remarkable contributions to human history.

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38 Mind blindness in this model (see fig 1) is seen as arising from twin abnormalities: the SM being set too high, such that complex systems such as the social world are hard to predict via systemising; and atypical development of empathising mechanisms that in the normal case make it possible to make sense of the social world via an non-SM route.
Influenza related hospital admissions in children: evidence about the burden keeps growing but the route to policy change remains uncertain

J S Nguyen-Van-Tam

Commentary on the paper by Beard et al (see page 20)

Influenza has long been recognised as a disease which affects children; however, it is only fairly recently that the literature on this subject has switched focus from community settings towards the burden of hospitalisations. This issue carries an article by Frank Beard and colleagues which draws attention to the issue in Sydney, Australia and addresses the issue in a quantitative as well as a qualitative way.1 It follows on from, and replicates the methodologies employed by similar pivotal studies in the USA and Hong Kong.2 3

Most experienced commentators would agree that the foundations of our understanding of the burden of influenza in children, are based on data generated by a series of prospective community studies which took place in the 1960s and 1970s in the USA, all of which combined clinical surveillance with attempts at virus isolation and serological studies, to a greater or lesser extent. They are probably too large and too expensive to ever contemplate repeating in the present era. In Tecumseh, Michigan, between 100 and 300 families with at least one child were studied continuously for six years from 1966 to 1971—a period which included the emergence of influenza A/H3N2, the last pandemic virus, in 1968.4 In Seattle, Washington, a similar study took place between 1965 and 1969 and again from 1975 to 1979, involving over 215 families with young children.5 In Houston, Texas, similar observations were made over the period 1976 to

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Infection

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illnesses (not just acute respiratory hospitalised for a broad variety of acute illnesses, or preschoolers. From the above consensus on whether the peak rate occurs in teenagers, primary age children, or preschoolers. From the above data that children act as introducers and spreaders of influenza infection in communities and individual households (presumably because they mix more often and shed viruses in higher titre than adults, and for longer). Glezen and Couch showed an upwards shift in the age distribution of influenza positive patient specimens (from children aged 5–19 years towards adults aged 20–44 years) as two influenza A epidemics progressed over time; similarly, in the same setting, school absenteeism preceded industrial absenteeism by about two weeks and paediatric admissions for pneumonia preceded those in adults by a similar period. Another notable finding from these studies was the disparity between serological and clinical attack rates. Fox et al showed that among teenagers and children with serologically proven infection, between 83% and 69% suffered a clinical illness; other studies have estimated that only 58% of infections in teenagers are accompanied by symptoms. What seems clear is that asymptomatic infections in children are not uncommon.

The relative importance of influenza compared with other childhood respiratory viruses, notably RSV, has also been well described in hospital based studies. In Kawasaki, Japan, a study of paediatric admissions over seven winter seasons from 1991 to 1998 revealed that 14% of admissions were attributable to serologically confirmed influenza, compared with 17.5% due to RSV; other respiratory viruses accounted for a much lower proportion of the total burden. In Kiel, Germany, a similar study of acute respiratory admissions over four winter seasons using PCR testing of nasopharyngeal specimens, revealed that 8.3% of such admissions were attributable to influenza infection, compared with 12.7% due to RSV; while RSV was the dominant pathogen in children aged 0–3 months, from the age of 2 years upwards, influenza predominated. Similar contemporary data are also available from Spain and Italy. In Leicester, UK a similar prospective study was carried out over one winter season in children <6 months, hospitalised for a broad variety of acute illnesses (not just acute respiratory illness), and again based on PCR testing of nasopharyngeal specimens. This revealed that 5.4% overall had an influenza infection at the time of admission, but more surprisingly, the proportion of influenza infected children among those presenting with acute respiratory illness (5.0%) was similar to the proportion among children presenting with non-respiratory illness (6.0%). These data suggest that the burden of hospitalisation due to influenza is underestimated by considering only acute respiratory admissions. Further evidence from Lyon, France suggests that during defined influenza epidemic periods the rate of virus isolation from small children who are unwell is probably many times higher.

Attempts have been made to quantify hospital admissions in children due to influenza, since the 1980s. Mullooly and Barker attempted this in 1982, by estimating the excess hospitalisation rate seen in epidemic compared with non-epidemic years in Oregon, using retrospective data. These researchers found an overall excess rate of 9/104 admissions with influenza related diagnoses, but this rose to 32/104 in children with at-risk conditions. Broadly comparable results also emerged from a similar study in Harris County, Texas. However neither of these studies used methods which adequately adjusted for the potential influence of RSV infections. In contrast, the cluster of recent studies, including the current one by Beard et al, have paid much closer attention to the possible confounding effects of RSV infection.

The featured study in this issue illustrates that the estimation of excess hospitalisation in children due to influenza is highly sensitive, not only to the precise mathematical method used, but also to the selection of either a summer baseline or one comprised of winter periods when influenza was not circulating (“periseasonal baseline”). Nevertheless, the study findings illustrate a consistent trend towards the highest rates of influenza related hospitalisation in children <12 months old. The excess hospitalisation rates observed in Sydney were also far higher than those recorded in recent US studies, but distinctly lower than those calculated by Chiu et al in Hong Kong. All three US studies focused on healthy children only, and might therefore be expected to have produced somewhat lower excess rates than in Sydney; but, on its own, this is unlikely to account for such dramatic differences. It is equally as unclear why the rates of excess hospitalisation in Hong Kong are so much higher than in Sydney. Aside from confounding, other possible explanations include true differences in the incidence of influenza in children in different countries, and genuine differences in thresholds for hospital admission of children with acute respiratory illness in different health systems.

Notwithstanding the uncertainties in the data regarding the true burden of influenza related hospital admissions in children, two consistent messages appear to be emerging. First, that the magnitude of hospital admission due to influenza in children is far from trivial and, at times, on a par with excess admission rates in adults at-risk. Second, that the burden of influenza related hospitalisation in children is concentrated among those <5 years, and especially those <12 months. However, in relation to the second point, the same is also true for RSV.

There have been many calls for individual countries to emulate the US policy for routine annual vaccination of young children against influenza. One of the critical factors in deciding on the cost effectiveness of such a policy is being able to establish reliable country specific data on the burden of hospitalisation. The current study and others like it will assist national governments in this task. Nevertheless even if the likelihood of cost effectiveness seems high, there are other equally important considerations which have to be factored in before scientific evidence becomes official policy. Not least of these are the availability of a suitable vaccine; space within the national childhood immunisation schedule to accommodate delivery of an annual seasonal programme (first time vaccines may require two doses); and finally, perceptual acceptability.


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Competing interests: none declared

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 Psychopathology before and after temporal lobectomy for epilepsy

Children with temporal lobe epilepsy (TLE) commonly exhibit psychopathology but children with severe TLE who are candidates for temporal lobectomy are even more likely to have a psychiatric disorder and many have more than one psychiatric diagnosis. A report from the Great Ormond Street Hospital for Children, London (A McElcan and colleagues. Developmental Medicine and Child Neurology 2005;47:666–72) includes 60 children who underwent temporal lobectomy between 1992 and 1998. Forty-three children (72%) had at least one psychiatric diagnosis before operation and 27 (45%) had more than one. The main psychiatric diagnoses preoperatively were disruptive behaviour disorder, not otherwise specified (DBD (NOS)), which affected 25 of 60 children; pervasive developmental disorder (PDD), 23 children; attention deficit hyperactivity disorder (ADHD), 14; and oppositional defiant disorder/conduct disorder (ODD/CD), 14. Five children had emotional disorders before operation. Of 57 children followed up after operation 25 had DBD (NOS) (five lost that diagnosis and five gained it), 21 had PDD, 13 ADHD, and 13 ODD/CD. Twelve had emotional disorder, ten for the first time. Overall, 72% had a psychiatric diagnosis before operation and 72% of those followed up had a psychiatric diagnosis after operation. Except for PDD, there was no significant relationship between psychiatric disorders and brain pathology, sex, seizure frequency, or postoperative seizure outcome. PDD was significantly associated with younger age of seizure onset, right sided temporal lobe lesions, and cognitive difficulties, and was non-significantly associated with male sex. Thirty-seven children had no or rare seizures after operation, nine had much fewer seizures, and 11 had an unchanged or worse seizure frequency. Psychiatric outcome after temporal lobectomy is uncertain, unrelated to seizure outcome, and needs to be discussed with parents during the preoperative work-up.
Two new theories of autism: hyper-systemising and assortative mating

S Baron-Cohen

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