

leads to a chronic onset of autistic symptoms.

Like you, I wish to see a full scientific description of O'Leary and colleagues' study as soon as possible. But, we must realise that the current epidemiological evidence does not refute MMR immunisation possibly triggering the onset of autism. In everyone's interest, we must keep an open mind.

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- 1 Editorial. Measles, MMR, and autism: the confusion continues. *Lancet* 2000; 355: 1379.
- 2 Taylor B, Miller E, Farrington CP, Cetropoulos MP, Favout-Mayaud JL, Waight P. Autism and measles, mumps, and rubella vaccine: no epidemiological evidence for a causal association. *Lancet* 1999; 353: 2026–29.
- 3 Farrington CP, Nash J, Miller E. Case series analysis of adverse reactions to vaccines: a comparative evaluation. *Am J Epidemiol* 1996; 143: 1165–73.
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Sir—The title of your editorial¹ is misconceived as there is no evidence linking MMR vaccine to autism. There is abundant evidence of the safety of MMR. Yet in England many autistic children have been granted legal aid to pursue claims alleging that their condition was caused by MMR. A case must satisfy the statutory requirement of reasonable grounds to receive legal aid. Last September, the High Court stated that “no positive link has ever been established”.

This litigation raises serious questions about the Legal Aid Board, which usually grants legal aid on the advice of the applicant's lawyer. Such advice cannot be independent because the lawyer has a direct financial interest in advancing the case irrespective of its merit. There is a clear conflict of interest. Not surprisingly, the success rate of legally aided pharmaceutical litigation in UK is negligible despite the expenditure of vast sums of public money in legal aid fees.

This high profile litigation provides popular support for the unsubstantiated health scare linking MMR to autism. The Board thus endangers the health of children by necessarily confusing a mere hypothesis concerning a possible role of MMR in the aetiology of autism as proper scientific evidence of causation.

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- 1 Editorial. Measles, MMR, and autism: the confusion continues. *Lancet* 2000; 355: 1379.

Sir—Your editorial,¹ to use your own words, raises far more questions than it answers. Perhaps I can add a few more questions, plus some hard and disturbing facts, to the debate. I should first mention that I am the parent of a child who became autistic after monovalent measles and MMR vaccinations.

Do children such as my son have typical autism? I believe not. My son, and others like him, do not possess the isolationist-behavioural or self-injurious features. They also have acute multiple food allergies and hyperactivity, which they also acquired shortly after vaccination. So is this autism at all, or is it brain damage that only partially resembles autism? And is the damage to the brain directly linked to gut permeability, as seems to be the case in children such as my son and the children studied by Wakefield and colleagues?² And what was it that damaged the gut in the first instance?

If there was a link with MMR, one might expect new-variant autism to start showing in official statistics. Why has autism among boys aged 3 years in part of east Surrey, UK, been running at one in 69 for the past 3 years? (C Clarke, Learning Disability Services, East Surrey Health Authority, personal communication, 1999).

Why did the education authority of Wakefield, West Yorkshire, UK, only have five autistic pupils in 1992, but had 111 by 1999? Can a 22-fold increase in just 7 years be credibly explained simply by better diagnosis of the condition?

Why is every autistic child in the Shetlands and the Western Isles of Scotland aged 12 years or under, with not a single case among children aged 13–19? Is this significant, given that MMR was introduced in 1988 (G Garson, Shetland Health Board, personal communication, 2000; and M Plant, Gilbert Brain Hospital, personal communication, 1999)?

Why is the USA also recording sharp increases in autism—eg, increases of 876% in 8 years (New Jersey), 627% in 6 years (Illinois), 13-fold in 6 years (Colorado), almost 1200% in 10 years (Broward, Miami), and so on (US state education data)?

Many accounts given by parents are consistent. I watched my son descend from a perfectly normal toddler of 14 months to a mentally-handicapped toddler of 16 months, after measles vaccination. It was a powerful experience to observe this at very close hand. At 14 months he could post square bricks through square slots, today at 13 years, he still cannot.

In evidence presented to the UK Bovine Spongiform Encephalitis (BSE)

Inquiry the then Medicines Division was reported to be in a disordered and very seriously understaffed state in 1988, the very year that MMR was licensed. Is this an irrelevant coincidence, or does it provide an illuminating insight into the background to MMR's introduction into the UK?

MMR is said to be safe because there have been few reported adverse reactions. Yet degeneration into autism is not a recognised adverse reaction. The onset of autism after MMR has therefore not been monitored. Is it a new syndrome that has not been recognised, and then been missed, by most of the medical community?

All these pieces of the MMR-autism jigsaw fit together extremely well. They also fit well with the findings of Wakefield, O'Leary, and others. And there are no features among the children of parents like myself—not one—that do not fit into the Wakefield–O'Leary explanation.

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- 2 Wakefield AJ, Murch SH, Anthony A, et al. Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *Lancet* 1998; 351: 637–41.

Sir—As father of a boy with autism aged 15 years, and as President of the Autism Autoimmunity Project, USA, I was interested by your editorial.¹ I was at the autism-vaccine hearings on April 6, 2000, in Washington, DC, USA, chaired by US Congressman Dan Burton. These hearings were vital for those of us who have autistic children and want more immunology research to be done, such as that of Wakefield,² O'Leary (unpublished), and Singh,³ and their colleagues.

Existing research that denies an association between autism and the MMR vaccine is either biased—because it originates from public-health authorities—or uses flawed epidemiology, such as in the study by Taylor and colleagues.⁴ When asked at the hearings whether he could provide for independent scrutiny the data that backed up his study, Taylor said he would have to check with his superiors. Wakefield, Singh, and O'Leary agreed to provide their data and had no need to check with their superiors. Quite a difference in response.

In April, 1999, I attended an autism biomedical conference in Atlantic City

(NJ, USA), and heard Jacqueline Bertrand of the Centers for Disease Control (CDC) talk about the increase of occurrence of autism in Brick Township, New Jersey. I later asked her whether any of the children were not vaccinated. She said no—Brick Township was a highly vaccinated population. I asked her whether CDC had done any immune panel blood tests or planned to do so. Again, she said no. The CDC said they did not know what caused the increase in occurrence of autism in Brick, but that the MMR vaccine was not a factor. Where is the logic and where is the science to support this assertion?

My son, Eric, was tested with an immune panel blood test and had raised measles titres. Also, Eric had inflammation of the colon. Many parents report the same results with their autistic children. There are no independent, long-term, safety studies involving any of the vaccines given in such variety during childhood (MMR vaccine included) to assure us that nothing is wrong. Are our children now acting as unwitting participants for a safety study?

Evidence for an autism pandemic is clear, especially in highly-vaccinated countries. In California, USA, there was a 273% increase in the number of autistic children in 1987–98. For Brick, CDC had documented that one in 149 children are autistic. In east Surrey, UK, one in 69 children aged 3 years are autistic. Eric goes to a special school for autistic children, which, in 1992, had 20 pupils; now there are over 100. To say this increase in numbers of autistic children is a result of better diagnosis shows insensitivity towards parents who know their own children and the education authorities entrusted with their education and long-term care.

We need exhaustive research funded now. Do we want to wait until one in five children are diagnosed with autism? Can our communities afford the increase in human suffering and the dire economic consequences? To ignore the findings that Wakefield, Singh, and O'Leary presented at the hearings will only worsen our dilemma.

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- 1 Editorial. Measles, MMR, and autism: the confusion continues. *Lancet* 2000; **355**: 1379.
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Role of sleep apnoea in epilepsy in elderly people

Sir—Linda Stephen and Martin Brodie (April 22, p 1441)¹ presented a comprehensive review of the aetiology and treatment of seizure disorders in elderly people. Although ischaemic brain injury was mentioned as a potential precipitating factor, the role of nocturnal hypoxaemia as a result of sleep apnoea (central or obstructive), was not addressed. The following case illustrates this association.

A man aged 69 years with stable coronary artery disease presented after having three episodes of generalised tonic-clonic seizures that occurred at 0300 h or 0400 h during sleep. Brain computed tomography was normal; a cardiovascular assessment did not show myocardial ischaemia or cardiac arrhythmias. He was subsequently found to have nocturnal hypoxaemia as a result of obstructive sleep apnoea, which was ameliorated by treatment with continuous positive airway pressure during sleep.

Seizures have been reported as the presenting sign of sleep apnoea.^{2,3} Also, treatment of sleep apnoea has been shown to improve seizure control in children.⁴ Moreover, the development of obstructive sleep apnoea after pathological weight gain caused an increase in seizure frequency in one patient.⁵

Since hypoxaemia as a result of sleep apnoea can precipitate seizures or exacerbate an underlying seizure disorder, it may be worthwhile to consider this diagnosis in patients being assessed or treated for seizures.

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Imaging hypnotic paralysis

Sir—Peter Halligan and colleagues (March 18, p 986)¹ showed that right orbito-frontal and anterior cingulate cortex were selectively activated without comparable activation in the motor cortex when a study participant attempted but failed to move his left leg during hypnotic paralysis. The findings were consistent with those of their patient with hysterical paralysis.² Although hypnosis and hysteria shared partially similar findings for motor paralysis, we cannot agree with their suggestion that the psychological mechanism underlying hypnotic phenomena provide a model for understanding and treating conversion hysteria symptoms. This model is insufficient for the following reasons.

First, the suggestions that produce hypnotic phenomena are usually administered by another individual, whereas the source of suggestions in hysteria lies primarily within the internal dynamics of the individual or in implicit societal and interindividual processes.³ Moreover, hypnotic phenomena are conventionally short-lived and are contained within hypnotic context, whereas conversion symptoms are usually of much longer duration. These differences suggest that conversion symptoms, but not hypnotic phenomena, may result from the primary lesions which continuously and automatically enhances (or cannot suppress) the production of symptoms.

Second, Spence and colleagues⁴ have reported that left dorsolateral prefrontal cortex hypofunction was common to all patients with hysteria when they moved an affected limb, irrespective of symptom lateralisation, and that right prefrontal hypofunction characterised feigned disorder of either side. Although we acknowledge that the tasks of the two studies^{1,2} differed somewhat, Halligan and colleagues' findings¹ of activation in the right orbito-frontal and anterior cingulate cortex are not consistent with those of hysterical patients. Rather, from the viewpoint of laterality, their findings (abnormality in right cortex) are most consistent with those of feigners.

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