Whooping cough is quite common and can be diagnosed clinically

Doug Jenkinson

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Whooping cough is quite common and can be diagnosed clinically

Enron—Harnden et al, with the help of a recently available blood test, have gone some way to confirm that whooping cough is still about. Since 1977, I have, with the help of my practice colleagues, been recording every clinically diagnosable case of whooping cough (based on a minimum of three weeks of paroxysmal coughing). This year so far we have seen six cases in a practice of 11 000. In 2002 we recorded 44 cases. Ten had blood specimens tested; nine were positive and the 10th was lost. Twenty three per cent of all whooping cough notifications in England and Wales in 2002. This surely cannot be because there is more whooping cough where I work.

I have had four papers published in the BMJ as a result of this study. I have decided to publish the incidence data on the web. Some years ago I set up a website to help patients diagnose their own whooping cough, and I can confirm from the feedback that doctors the world over seem to be equally poor at diagnosing it. I have published the Keyworth figures (www.whoopingcough.net/keyworth). Basic data on all cases are available, as well as year-on-year figures compared with national notifications (figure).

Whooping cough is a distressing illness, especially when it is undiagnosed. I am hopeful that the paper by Harnden et al will wake us up about this disease, which, if my data are correct, is just as common as it was 25 years ago after the vaccine scare settled.

Doug Jenkinson
BMJ
352
BMJ
3
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Competing interests: DJ has a website on whooping cough that charges a fee for personal advice. The fees have so far never covered the expenses for the site.


Author’s reply to review of his book on autism

Editor—The misrepresentations and omissions in Fitzgerald’s review of my book demand correction. The book overviews evidence for (and against) the apparent rise in autism and concludes that the increase is probably real—pointing to an environmental risk factor so far unknown.

A focus of the book is on physiological dysregulation that accompanies and exacerbates autism. It reviews evidence that limbic damage can both cause and reflect physiological problems, with environmental toxins including heavy metals having a causal role, although concluding that the rise in autism cannot be uniquely attributed to such exposure (but it remains a strong suspect). Just one short chapter refers to possible strategies for treatment and prevention.

Fitzgerald focuses his critical attentions only on this chapter: most of the book is not addressed. He asserts that there is no coherent scientific rationale for the treatments and little evidence of their efficacy. He omits to note the considered position taken by the book, that the field is fraught with uncertainty because few logical therapeutic approaches have been evaluated systematically. Reviewer and author are therefore substantially in agreement.

Fitzgerald then draws attention to an autistic boy who died while receiving chelation treatment. This is also misleading: use of the wrong drug was responsible for the death.

He then says that guidelines from the American Academy of Neurology explicitly reject a series of biomedical tests. The recommendations speak not of rejection but of inadequate supporting evidence, a different matter. This is an area of debate.

Regarding biomedical intervention, affected children should be spared unproved treatments until efficacy has been shown in controlled trials. Trials (examples include haloperidol, risperidone, methylphenidate hydrochloride (Ritalin)) have been conducted through mainstream medicine and not by (in the words of Fitzgerald) quacks and charlatans. Valproate continues to be prescribed for epilepsy associated with autism, even though valproic acid is a known cause of autism.

Fitzgerald’s exclusive focus on therapeutic options is unfortunate, for this is a minor aspect. The central thesis offered by the book is of a rational and plausible sequence of events that causes as well as exacerbates autism—addressed by Fitzgerald as “speculative.”

Rushing into biomedical remediation without fullest consideration of justification and efficacy is a mistake, but it is an equally grave error to dismiss without objective consideration of all the evidence (as laid out in the book), and new research under way, an environmental and physiological contribution to autism.

Richard Lathe
neuroscience consultant
Pieta Research, PO Box 27069, Edinburgh
EH10 5YW
rlathe@pieta-research.org

Competing interests: None declared.

1 Fitzpatrick M. Autism, brain and environment. BMJ 2006;333:205. (29 Jul.)
What works in schizophrenia

Competing interests: PMH has received honoraria for lecturing or attending advisory boards from Eli Lilly, Janssen-Cilag, and Novartis.


Cognitive behaviour therapy is not effective

Editor—Kingdon’s statement that more than 20 randomised controlled trials and five meta-analyses have shown cognitive behaviour therapy to be beneficial in schizophrenia gives an oversimplified picture of both the randomised controlled trials and the meta-analyses.1 Reviewing the randomised controlled trials, Tarrier and Wykes, two supporters of cognitive behaviour therapy in schizophrenia, noted that five included groups who received befriending, supportive counselling, or problem solving to control for the non-specific effects of intervention, in other words as a psychological placebo.2 They said that not one study has shown clear and significant overall difference between cognitive behaviour therapy and the non-specific control groups.3

The conclusion of the Cochrane Collaboration’s meta-analysis of cognitive behaviour therapy for schizophrenia was currently that trial based data supporting the wide use of such treatment for people with schizophrenia or other psychotic illnesses are far from conclusive.4 Compared with standard care, cognitive behaviour therapy was found not to reduce relapse and readmission; it helped mental state over the medium term but after one year the difference had gone, and it did not show a consistent effect on continuous measures of mental state. Compared with supportive psychotherapy, cognitive behaviour therapy had no effect on relapse or on the outcome “no clinically meaningful improvements in mental state” over the same time periods.5

Behind all the recent publicity surrounding cognitive behaviour therapy for schizophrenia, the truth is that it works only in poorly controlled trials and not in well controlled ones.

Peter J Mckenna  professor of psychiatry
University of Glasgow, G12 8NH
peter.mckenna@virgin.net

Competing interests: None declared.

1 Kingdon C. Psychological and social interventions for schizophrenia. BMJ 2006;333:212-3. (29 July)


Fluoroquinolone resistance in Salmonella Typhi

Editor—As Parry et al point out,1 appropriate laboratory methods are crucial in detecting clinically important quinolone resistance. We highlight the emergence of strains of Salmonella enterica serovar Typhi (S Typhi) that show reduced susceptibility to the fluoroquinolones but are susceptible to nalidixic acid (minimum inhibitory concentration <16 mg/l). In a review of 692 isolates of S Typhi sent to the Laboratory for Enteric Pathogens at the Health Protection Agency in London between 2000 and 2003 we detected 49 isolates that were susceptible to nalidixic acid but had reduced susceptibility to fluoroquinolone (minimum inhibitory concentration 0.125-1.0 mg/l). When the country of acquisition was known, 18 of these isolates were from patients who had visited India; eight, Pakistan; four, Bangladesh; and one, Kenya.

Overall, of 271 isolates with reduced susceptibility to fluoroquinolone, 18% were susceptible to nalidixic acid and therefore would not have been detected by routine screening with a nalidixic acid disc. No clinical outcome data are available and so the clinical relevance of these strains is uncertain, but an enhanced surveillance study is being undertaken by the Health Protection Agency.2

Although resistance to nalidixic acid (minimum inhibitory concentration >256 mg/l) remains an important marker for failure of fluoroquinolone treatment in typhoid fever, several isolates show reduced susceptibility to fluoroquinolone while remaining susceptible to nalidixic acid. This is particularly true for isolates from the Indian subcontinent. Furthermore, in six of the 49 such isolates (table), none of the mutations

1 S Typhi susceptible to nalidixic acid but with reduced fluoroquinolone susceptibility imported into England, Scotland, and Wales, 2000-3

<table>
<thead>
<tr>
<th>Year</th>
<th>Susceptible to nalidixic acid and reduced susceptibility to fluoroquinolones</th>
<th>Resistant to nalidixic acid and reduced susceptibility to fluoroquinolones</th>
<th>Total with reduced susceptibility to fluoroquinolone (% susceptible to nalidixic acid)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000</td>
<td>10</td>
<td>47</td>
<td>57 (18)</td>
</tr>
<tr>
<td>2001</td>
<td>0</td>
<td>55</td>
<td>63 (13)</td>
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<tr>
<td>2002</td>
<td>10</td>
<td>60</td>
<td>70 (18)</td>
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<td>2003</td>
<td>21</td>
<td>74</td>
<td>95 (22)</td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
<td>222</td>
<td>271 (18)</td>
</tr>
</tbody>
</table>
in gyrA and parC commonly associated with reduced resistance to fluoroquinolone were present using the GAMA method.\textsuperscript{1} Therefore a new mechanism of fluoroquinolone resistance, which might not confer nalidixic acid resistance, may be emerging in S Typhi. Our findings underscore the importance of estimating the minimum inhibitory concentration of the agent selected for treatment.

Fiona J Cooke\textsuperscript{2} MRC clinical research training fellow  Fiona@sanger.ac.uk

John Wain\textsuperscript{3} member of faculty  Wellcome Trust Sanger Institute, Wellcome Trust Genome Campus, Cambridge CB10 1SA

E John Threlfall\textsuperscript{4} professor  Health Protection Agency, Centre for Infections, London NW9 5HT

Competing interests: None declared.


2 Bhutta ZA. Current concepts in the diagnosis and treatment of typhoid fever. BMJ 2006;332:78-82. (8 July.)


An independent NHS?

Editor—I received 21 items of correspondence about my personal view on separating the NHS from direct government involvement, mostly supporting the idea and many urging me to take it further.\textsuperscript{1} I have worked in the NHS for 28 years and my conclusions in many of these articles in writing about the NHS as a UK model when commenting only on the NHS in England as directed from Westminster. The NHS in Scotland, in particular, is different in many respects.

A need exists to survey and audit the differences with the other nations, of course, and I communicated that suggestion to the authors of a paper last year about the NHS in England;\textsuperscript{2} Feachem et al too seemed unaware from a distance that England is not the UK.

In Scotland, I was glad that two reforms have just been made. First, the NHS in Scotland, in particular, is different in many respects.

There is no “UK health service”

Editor—Berwick and Leatherman express their steadfast support for the NHS eloquently.\textsuperscript{3} We forgive them for their error and that of the other authors in this series of articles in writing about the NHS as a UK model when commenting only on the NHS in England as directed from Westminster. The NHS in Scotland, in particular, is different in many respects.

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An independent NHS?

Editor—As a Scottish doctor I can tell Godlee that the assumption that “NHS” means “NHS in England” in not only the BMA but in government statements, the BBC, and the national British newspapers eventually produces a weary acceptance.\textsuperscript{5} Frankly, like the BMA, I have no idea what’s going on in the NHS in Scotland because it is so unreported.

The degree of divergence in NHS systems in the four countries based, as the BMA briefing paper Godlee refers to points out, on profoundly different philosophies and policy groups, provides us with a unique opportunity to learn something. What are the effects of the different NHS reforms on the experience of patients? And what are the effects of the different reforms on the experience of those who are delivering health care—the NHS employees in the four different countries? If we could at least read some comparative journalism, not to mention proper research which considered these questions, then we would be able to take the first steps in all four countries to create NHS changes which were evidence based.

Robert W Leckridge  associate specialist  Glasgow Homeopathic Hospital, Glasgow G12 0XQ  bolleckridge@gmail.com

Competing interests: None declared.

1 Godlee F. Editor’s choice. B is for British. BMJ 2006;333:0. (13 July.)

Suffering from files

Editor—Thornton says that we shouldn’t remove the NHS from politics—he says that the NHS wouldn’t exist today without politicians like Nye Bevan.\textsuperscript{6} He thinks that more democratic commissioning—perhaps even from local government—would increase NHS accountability and at the same time give more freedom to clinicians. Can we really square the circle in this way?

Or would this idea become just another initiative leading nowhere but more bureaucracy and an even higher paper mountain? What would Bevan say to the range of initiatives that the NHS has gone through in the past 10 years? Here’s what he said about a contemporary administrator: “Poor fellow, he suffers from files.”\textsuperscript{7}

Sarah J Farrow  specialist registrar in rheumatology and medicine  University Hospital Lewisham, London SE13 6HL  sfarrow@doctors.org.uk

Competing interests: None declared.

1 Thornton S. Democratic control is essential. BMJ 2006;332:212. (29 July.)


Towards evidence based changes in NHS in all UK countries

Editor—As a Scottish doctor I can tell Godlee that the assumption that “NHS” means “NHS in England” in not only the BMA but in government statements, the BBC, and the national British newspapers eventually produces a weary acceptance.\textsuperscript{5} Frankly, like the BMA, I have no idea what’s going on in the NHS in Scotland because it is so unreported.

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Robert W Leckridge  associate specialist  Glasgow Homeopathic Hospital, Glasgow G12 0XQ  bolleckridge@gmail.com

Competing interests: None declared.

1 Godlee F. Editor’s choice. B is for British. BMJ 2006;333:0. (13 July.)