LETTERS

Toxic levels of mercury in Chinese infants eating fish congee

Stephen J Corbett and Christopher C S Poon

To the Editor: We report elevated mercury levels in three infants, each the only child of Chinese parents living in Sydney. All three children had eaten fish congee (a rice and fish porridge) as a weaning food and ate fish regularly as toddlers. Their parents had sought medical advice for either developmental delay or neurological symptoms in the children.

A 2-year-old boy had demonstrated increasingly aggressive behaviour for the past 6 months. A general practitioner had diagnosed mercury poisoning in the boy's father 2 months earlier, following investigation for complaints of allergies, rashes, abdominal pain and diarrhoea. The family ate fish (usually salmon, barramundi or snapper) at least five times a week, and had used unspecified herbal medicines in the past. The child had eaten fish regularly since weaning. The boy's blood mercury level was 158 nmol/L (normal range [NR], < 50 nmol/L), a random urine mercury/creatinine (Hg/Cr) ratio was 9 nmol/mmol (NR, < 6 nmol/mmol*), and his hair mercury level was 1.42 mg% (NR, < 0.18 mg%). The boy's father and mother (who was pregnant) also had elevated hair mercury levels, of 4.3 mg% and 6.0 mg%, respectively. The father and child were treated with chelation therapy elsewhere.

A boy aged 2 years and 10 months presented with delayed speech and some autistic features. Since weaning, he had eaten fish (barramundi, sea perch, salmon and rock cod) up to eight times a week. He had no history of herbal medicine use, and his thyroid function, blood lead level, and a DNA screen for fragile X syndrome were normal. The child's blood mercury level was 350 nmol/L and urine Hg/Cr ratio was 14 nmol/mmol (NR, < 10 nmol/mmol*). The boy's father did not eat fish, and his blood mercury level was 19 nmol/L. The child's mother did eat fish, and had a blood mercury level of 27 nmol/L. Two weeks after removing fish from the diet, the child's blood mercury level had fallen to 99 nmol/L and his urine Hg/Cr ratio to 7 nmol/mmol. However, his behaviour did not improve, and he was subsequently diagnosed with classical autism.

A 15-month-old boy presented with delayed development since birth. Fish had been introduced to his diet at 8 months of age, and he had since continued to consume fish four to five times a week. He had recently eaten either ling or salmon. The boy's mother had consumed ling three to four times a week after the fifth month of her pregnancy. The child's thyroid function, DNA screen for fragile X syndrome, chromosome karyotype, and urinary metabolic screen were normal. His blood mercury level was 143 nmol/L, but fell to 19 nmol/L over a period of 1 year after ceasing fish intake. His longer-term developmental status is unknown.

Fish congee, made with either freshwater species or locally caught fish, is a common weaning food in coastal regions of southern China and South-East Asia.[1] Adding fish to the weaning diet has health benefits,[1,2] such as reducing anaemia, and is actively promoted. However, fish, particularly the large pelagic (open ocean) species more likely to be bought in Australia, may also contain mercury. Excessive consumption of mercury has been associated with neurological impairment.[3-5]

The Box shows that the consumption by infants of fish congee made from portions of large fish species may exceed the provisional tolerable weekly intake (PTWI)[7] for methylmercury of 1.6 μg/kg bodyweight/week (the limit considered sufficient to protect a developing fetus). Food Standards Australia New Zealand's most recent risk assessment con-

Estimated weekly mercury intake in infants consuming fish congee

	Mean mercury concentrations in fish tissue* (μg/kg fish)	Child's estimated weekly mercury intake† (μg/kg bodyweight/week)
Fish fillets‡		
Maximum	50	1.25
Median	16	0.40
Minimum	5	0.13
Barramundi		
Maximum	310	7.75
Minimum	40	1.00
Snapper		
Maximum	520	13.00
Minimum	52	1.05

* For species consumed in Australia.[6] † For a 12-month-old child weighing 10 kg; weekly fish consumption is estimated to be 0.25 kg, assuming 50 g servings five times per week. Provisional tolerable weekly intake for methylmercury = 1.6 μg/kg bodyweight/week.[7] ‡ Average concentrations of all fillets purchased. ◆

* The two laboratories reported different normal ranges for the urine Hg/Cr ratio.

cluded that median-level consumers of fish are unlikely to exceed the PTWI for methylmercury,[6] but frequent consumers might if all their consumption is of predatory or long-lived fish species, which tend to acccumulate higher concentrations of mercury.

It has been previously noted in the Journal that public health policy regarding fish consumption needs to balance the health benefits for cardiovascular disease and anaemia with the possible ill effects of mercury on neurological development in infants.[8]

We recommend that multilingual information about fish and mercury be made available to pregnant women and mothers, especially targeting groups who are likely to be frequent consumers of fish and who use fish in weaning and infant foods. Regulatory and health promotion activities could also be informed by surveillance of blood or hair mercury levels in infants from ethnic groups at high risk of mercury intoxication, and of the frequency of fish consumption in this age group (by type of fish).

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Prostate cancer screening and bacteraemia

Francis J Bowden, Jan Roberts and Peter J Collignon

To the Editor: Prostate cancer screening remains controversial. There is currently a lack of evidence that treating prostate cancers identified by screening leads to prolonged survival,[1] and the main screening test (serum prostate-specific antigen [PSA] concentration) has poor sensitivity and specificity. Patients with an elevated PSA level usually undergo a transrectal ultrasound-guided (TRUS) prostate biopsy for definitive diagnosis. TRUS biopsy is associated with risks that include bleeding, urinary tract infections, prostatitis and bacteraemia. Infective complications can occur even when prophylactic antibiotics are administered.[2] Gram-negative bacteraemia is usually associated with a mortality rate of at least 5%.[3]

We reviewed prostate biopsy-associated bacteraemia in Australian Capital Territory residents using data collected over the 5-year period from 2002 to 2006. All patients in ACT public hospitals who have positive blood culture results are followed as part of a bloodstream infection surveillance program. Although all prostate biopsies are performed in the private sector, nearly all patients who develop major complications are cared for in public hospitals. The number of biopsies performed on ACT residents during the study period was obtained from Medicare Benefits Schedule statistics (item numbers 37215, 37218 and 37219).[4]

Over the 5-year period, we identified 19 episodes of bacteraemia following 1843 prostate biopsies, representing a 1.0% risk of bacteraemia (95% CI, 0.6%–1.6%) (Box). No patients died, but five required admission to the intensive care unit.

A 1% bacteraemia rate associated with prostate biopsies would be an underestimate of the true rate, for a number of reasons: some patients were also admitted with severe sepsis syndromes but with negative blood cultures; some patients may have been treated in another state; and some patients may have presented to private hospitals, specialists or general practitioners and been treated empirically without blood cultures being collected. Three additional cases of bacteraemia following TRUS biopsies were excluded from our rate calculations because they involved New South Wales residents.

It has been estimated that, if a million men over 50 years of age were screened, about 110 000 would have raised PSA levels.[5] Of these, about 90 000 would undergo a biopsy and 20 000 would be diagnosed with prostate cancer. Our data show that TRUS biopsies carry a risk of serious infective complications and, notably, about 75% of these complications would occur in men without prostate cancer.

We believe that data on the likely complication rates resulting from PSA testing and TRUS biopsies must be factored into considerations of the benefits versus risks and the cost-effectiveness of any prostate cancer screening program.

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Prostate biopsies and associated bacteraemia episodes, Australian Capital Territory, 2002–2006*

	2002	2003	2004	2005	2006	Total
Prostate biopsies performed	322	324	465	377	355	1843
Bacteraemia episodes	6	3	2	4	4	19
Proportion of biopsies resulting in bacteraemia	1.9%	0.9%	0.4%	1.1%	1.1%	1.0%

* Three New South Wales residents who were diagnosed and treated for bacteraemia in the ACT were excluded (two had biopsies in Sydney and one in the ACT). Two patients had two episodes of bacteraemia (each more than 1 month apart). Fifteen episodes of bacteraemia were caused by *Escherichia coli* and one each by *Klebsiella pneumoniae*, Group C *Streptococcus*, *Citrobacter freundii* and *Proteus mirabilis*. ◆

Fatal community-associated methicillin-resistant *Staphylococcus aureus* pneumonia after influenza

Steven YC Tong, Nicholas M Anstey, Gary D Lum, Rachael A Lilliebridge, Dianne P Stephens and Bart J Currie

To the Editor: The report by Risson and colleagues of a fatal case of necrotising pneumonia caused by community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA)[1] appropriately highlights the emerging issue of CA-MRSA infections in Australia,[2] and the possibility that severe *S. aureus* sepsis may follow recurrent furunculosis. We wish to draw attention to the association between severe staphylococcal pneumonia and a preceding influenza-like illness.

In September 2006, a 56-year-old woman of European background with a history of chronic back pain and depression presented to the Royal Darwin Hospital after a 4-day influenza-like illness characterised by cough, fever and sore throat. She then developed dyspnoea and pleuritic chest pain, followed by an abrupt respiratory deterioration.

She was intubated and admitted to the intensive care unit with severe sepsis. A chest x-ray showed widespread bilateral pneumonia. We began broad-spectrum antibiotic therapy with piperacillin–tazobactam and azithromycin as per the hospital's dry-season protocol for severe community-acquired pneumonia. Further history from her husband revealed an episode of boils 1 month previously, which responded to antibiotic therapy. We added vancomycin to the therapy and, when sputum and blood cultures showed MRSA 48 hours after admission, we also added rifampicin and gentamicin. On Day 5 of admission, her clinical condition deteriorated further and we replaced rifampicin and gentamicin with linezolid. Complement fixation testing of serum taken on admission showed an influenza A antibody titre of 128, consistent with recent acute infection. Despite ongoing intensive supportive care, the patient died from refractory respiratory failure 10 days after admission.

Typing of the *S. aureus* isolates from blood and sputum showed that their single nucleotide polymorphism and variable gene profile was consistent with the Queensland clone (ST93-MRSA-IV) of CA-MRSA, and that the Panton–Valentine leukocidin gene was present.

S. aureus has long been a recognised cause of influenza-associated pneumonia. Of concern, two recent reports from the United States identified 25 patients with CA-MRSA associated with severe pneumonia following influenza-like illnesses.[3,4] Most of these patients were young (median age of 21 years[3] and 17.8 years,[4] respectively) and otherwise healthy. Combined mortality in these two studies was 40%.

With an increasing prevalence of CA-MRSA in areas of Australia,[2] CA-MRSA pneumonia should be suspected in patients presenting with worsening respiratory status and sepsis following an influenza-like illness. We stress the importance of annual influenza vaccination for those at increased risk of influenza-related complications.[5]

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Methicillin-resistant *Staphylococcus aureus* in hospitals: time for a culture change

Keith V Woollard

To the Editor: The recent editorial by Collignon and colleagues emphasised the importance of infection control mechanisms in reducing patient harm from antibiotic-resistant organisms.[1] It focused on disinfection of the hands of health care workers in hospitals. However, a vigorous education and surveillance program in a hospital in Victoria failed to achieve compliance among health care workers of even 50%.[2] Top of the list of self-reported factors leading to poor compliance is "skin irritation and dryness associated with the use of hand hygiene agents".[3]

There have been no properly controlled trials, with clinically important endpoints, of currently recommended hand-hygiene practices. With the likely poor compliance rates, such trials would likely fail.

A different approach might be more effective. Reducing skin contact between health care workers, patients and their immediate environment seems logical. Data show that skin contact produces two-step transfer of material in 82% of cases.[4] The Victorian study did include gloving as an alternative to disinfection in measuring hand-hygiene compliance.[2] However, in what might be a backward step, a recent study concluded that physicians should be encouraged to shake hands with patients![5]

Perhaps an educational campaign to avoid skin contact with environmental surfaces and other health care workers, with use of disposable gloves for patient contact, could be the basis of a successful trial to address more effectively the transmission of antibiotic-resistant organisms in hospitals.

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John K Ferguson and Helen Van Gessel

To the Editor: The magnitude and distribution of the problem of health care-associated methicillin-resistant *Staphylococcus aureus* (MRSA) in Australia can be gauged from the report of a forum on MRSA control conducted at the Australasian Society for Infectious Diseases (ASID) in March 2007.[1] This report contrasted approaches to control of health care-associated MRSA and quantified the population incidence rate of health care-associated MRSA bacteraemia across Australia from data derived from direct surveillance systems (Box). Reporting of MRSA infections is thought to be complete from all jurisdictions except Victoria and New South Wales. Figures for Victoria were extrapolated from accurate surveillance data representing 50%–60% of events. The degree of incompleteness of reporting in NSW could not be determined, and a range based on reports to NSW Health over 3 years was used. Overall morbidity of health care-associated MRSA in Australia is much higher, as only a minority of MRSA infections lead to bacteraemia.

The ASID report estimated that between 699 and 924 cases of bacteraemia would be prevented if other states and territories reduced their incidence of MRSA bacteraemia to that of Western Australia through implementation of more stringent infection control measures. The mortality of MRSA bacteraemia is 8%–50% (average, 29%).[2] A recent study showed that more than half (59%) of such deaths were directly attributable to MRSA[3] rather than other non-infective causes. These outcome proportions provide a minimum estimate of between 120 and 158 preventable deaths per annum in Australia directly caused by health care-associated MRSA — comparable to the annual South Australian road toll.

As identified recently in the Journal by Collignon and colleagues, there are significant structural barriers to achieving infection control — especially inadequate isolation resources and pressure on bed stock.[4] Other dimensions of the MRSA problem include the high incidence of MRSA in many aged care facilities, the epidemic emergence of community strains of MRSA (best described in the recent report from WA on MRSA notification data up until 2002[5]), the possibility of significant zoonotic reservoirs,[6] and the role played by imprudent antibiotic use.

MRSA colonisation or infection needs to be made a nationally notifiable disease, with a system in place to enable typing of isolates. As in WA, such a system would enable more effective identification of MRSA carriers before hospital admission, the detection of emerging epidemic strains, and timely investigation of MRSA outbreaks occurring in community groups, such as in aged care facilities. Most importantly, all states and territories need to adopt, and provide resources for, consistent, stringent approaches to surveillance, prevention and control of health care-associated MRSA that are in keeping with internationally recommended approaches. Given the scale of preventable injury occurring in many states, MRSA control must be made one of the highest priorities for patient safety.

Relative burden of health care-associated MRSA morbidity across Australia[1]

Area	Health care-associated MRSA bacteraemia events	Year(s) of data	Rate per 100 000 population
Darwin	16	2006	13.3
New South Wales/ACT*†	437–602	2003–2005	6.2–8.5
Queensland*	133	2005	3.4
South Australia*	37	2006	2.4
Tasmania*	3	2006	0.6
Victoria*†	270–330	2000–2006	5.4–6.6
Western Australia*	22	2006	1.1
Total	918–1143		4.5–5.7

MRSA = methicillin-resistant *Staphylococcus aureus*. ACT = Australian Capital Territory.
* Figures from these jurisdictions include private hospital event estimates.
† Figures from NSW and Victoria are minimum estimates, because of incompleteness of current reporting in these states. ◆

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Luke F Chen, Deverick J Anderson, Keith S Kaye and Daniel J Sexton

To the Editor: The recent editorial by Collignon and colleagues challenged Australian physicians and health care leaders to confront the rising burden of methicillin-resistant *Staphylococcus aureus* (MRSA).[1] Compared with Australia, the United States has a bigger problem with MRSA; more than 60% of all hospital-acquired *S. aureus* infections are now caused by MRSA.[2]

Appropriately, the medical community has made an urgent call for action. For example, the Institute for Healthcare Improvement (a not-for-profit organisation based in the US that aims to improve health care throughout the world) incorporated specific MRSA prevention measures into its recent 5 Million Lives Campaign (see http://www.ihi.org/ihi). One of these prevention measures, a recommendation for active surveillance, has generated controversy. Specifically, the cost-effectiveness of this strategy is still vigorously debated in the infection control literature.[3] At present, it is unclear what surveillance testing method should be used in the laboratory, and whether testing should be done for all patients or just those identified as high risk.

The cry for help from community activists in the US and the United Kingdom has reached the ears of their legislative representatives. In two northern US states, lawmakers are considering bills that require universal active surveillance in their hospitals. Mandating resource-stretched health systems to implement obligatory active screening is not a prudent use of resources. The Society for Healthcare Epidemiology of America and the US Association for Profes-

sionals in Infection Control and Epidemiology recently published a joint position paper opposing this legislative activity, noting that data in support of active surveillance have been restricted to high-risk populations.[4] We support this position and remind readers that active surveillance does not obviate the need for adherence to basic and consistent hand-hygiene practices.

Complacency and lack of clinical leadership remain the greatest challenges in the efforts to reduce the transmission of MRSA. Why do we accept this epidemic as a fact of life as our health care workers complacently contribute to the nosocomial transmission of MRSA? By implementing simple prevention policies, feedback of data on nosocomial transmission of MRSA, and increased infection-control education, we have achieved a 22% reduction in MRSA infections in our network of community hospitals.[5] Still, we acknowledge the absence of a zero-tolerance approach to failures in hand-hygiene practices. More needs to be done. We challenge our clinical leaders to demand higher standards for hand hygiene. Most cases of nosocomial MRSA transmission represent failures of basic hygiene practices. The problem is surmountable. Infection control is not a skill of a few, but the responsibility of every team member. The onus is on all of us.

Competing interests: Deverick Anderson sits on the Regional Advisory Panel for Pfizer and Schering–Plough.

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Peter J Collignon, M Lindsay Grayson and Paul D R Johnson

In reply: We thank Woollard for his comments on hand hygiene. While important, hand hygiene is just one component of what is needed to decrease the spread of methicillin-resistant *Staphylococcus aureus* (MRSA) in hospitals. Decontamination of the environment, contact precautions for colonised patients, active surveillance and screening, effective programs to prevent common infections such as intravascular catheter sepsis, good antibiotic stewardship and better hospital design are also indispensable.[1]

We do not accept that "There have been no properly controlled trials, with clinically important endpoints, of currently recommended hand-hygiene practices". For instance, the recent study quoted by Woollard showed that hand hygiene reduced infections hospital-wide, using the clinically important endpoint of serious bloodstream infections (MRSA and antibiotic-resistant gram-negative bacteria).[2] Thus, at least two large peer-reviewed studies show that alcohol-based hand-hygiene programs reduce hospital-acquired MRSA infections[2,3] (Level III evidence[4]).

There is nothing wrong with using disposable gloves, as suggested by Woollard, provided they are changed every time a health care worker moves between patients. Otherwise, gloves spread MRSA just as efficiently as unclean hands. Applying good-quality hand-hygiene products is less cumbersome than changing gloves and also allows direct human contact, which Woollard reminds us is important to patients.

How MRSA is spread in hospitals is now well known — our problem is getting health care workers to remember to practise good hand hygiene all the time, every day, before and after every patient contact.

The data shown by Ferguson and Van Gessel reaffirm how common and serious a problem we have with MRSA bacteraemia. They estimate there are about five episodes per 100 000 people annually across Australia. However, we believe that the true rate is double this.

The rate of *S. aureus* bacteraemia (ie, MRSA and methicillin-sensitive *S. aureus* combined) in Australia is around 35 per 100 000 per year,[5] with 27% caused by MRSA.[5] This crudely translates to an MRSA rate of 9.5 per 100 000. The rates are probably much higher in the states with more health care-acquired MRSA (New South Wales and Victoria). More recent data suggest that 36% of hospital-acquired invasive *S. aureus* infections were caused by MRSA, with the highest percentages in NSW (41%) and Victoria (39%).[6]

It is also worth noting that, when MRSA bacteraemia became notifiable in England in 2001, there was a 50% increase in reported *S. aureus* bacteraemia episodes.[1] This suggests that under-reporting is common in any voluntary reporting scheme, and is also likely for Australian data.

Worryingly, Chen and colleagues point out that MRSA is an even bigger problem in the United States than in Australia. However, we are not far behind.[6] Although we share their concerns about legislative impositions, some external controls and measurements can be an advantage. Western Australia, the only Australian state where MRSA is notifiable, has the lowest rates of health care-associated MRSA. While we do not want imposed "one size fits all" legislated controls, we do need change: the practice of the past 40 years has not worked. Every institution needs to have an effective MRSA control program, with components chosen according to the local situation. Institutions should measure MRSA and report centrally, especially if their rates are high and not falling continually (eg, over a year). Shop-floor quality improvement programs with empowered workers are much better than top-down management-imposed regulation (eg, from government).

We need to shake our complacency and that of our health care colleagues, accept clinical leadership and take control. Otherwise, legislative controls will be imposed on us.

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Acupuncture for persistent allergic rhinitis: a randomised, sham-controlled trial

Edzard Ernst

TO THE EDITOR: Xue and colleagues reported an interesting randomised, single-blind trial of acupuncture for persistent allergic rhinitis (PAR) and concluded that acupuncture is an effective treatment for this condition.[1] I am not entirely sure that this is true. The authors state that "once needling sensation (known as de-qi) was obtained, the needles were manipulated . . .". In the sham group they inserted needles at non-acupuncture points where, according to acupuncture theory, no de-qi can be elicited. Thus the intervention patients were experiencing de-qi, and the control patients were not. This means that neither the patients nor the therapist were blinded. Consequently, the difference in outcome between the two groups could be unrelated to acupuncture itself, and caused by patient expectation, therapist expectation or both.

In addition, the statement of Xue et al that "no other randomised controlled trial of acupuncture in adults with PAR has been reported in the English medical literature"[1] was misleading. Our review of this topic[2] included no fewer than six randomised controlled trials, all either published in English or, in one case, with an English abstract. Interestingly, three of them suggested acupuncture to be effective, while three failed to do so. I fear that the study by Xue et al does little to resolve this intriguing discrepancy.

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1 Xue CCL, An X, Cheung TP, et al. Acupuncture for persistent allergic rhinitis: a randomised, sham-controlled trial. *Med J Aust* 2007; 187: 337-341.
2 Ernst E, Pittler MH, Wider B, Boddy K. The desktop guide to complementary and alternative medicine. 2nd ed. Edinburgh: Elsevier Mosby, 2006. ❑

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IN REPLY: Ernst is concerned about the sham acupuncture procedure used in our trial.[1] Although not universally agreed, the sham/placebo control we adopted has been described as best practice.[2,3] The assumption that, without de-qi, participants would not be blinded is not supported by the literature. In fact, a sham procedure without de-qi was used in a recent trial reported by Ernst and colleagues on acupuncture for subacute stroke rehabilitation.[4] In that study, as in ours, to increase the credibility of blinding, participants with previous experience of acupuncture were excluded, and those assessing the outcomes of treatment were blinded.

With regard to previous randomised controlled trials of acupuncture for adults with persistent allergic rhinitis in the English medical literature, Ernst failed to distinguish between seasonal allergic rhinitis (SAR) and persistent allergic rhinitis (PAR).[5] Of the six studies included in his review,[5] five were on SAR, and the sixth, which was cited in our report (reference 14), was on children with PAR. These reports, therefore, are not inconsistent with our findings.

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1 Xue CCL, An X, Cheung TP, et al. Acupuncture for persistent allergic rhinitis: a randomised, sham-controlled trial. *Med J Aust* 2007; 187: 337-341.
2 Dincer F, Linde K. Sham interventions in randomized clinical trials of acupuncture — a review. *Complement Ther Med* 2003; 11: 235-242.
3 Witt C, Brinkhaus B, Jena S, et al. Acupuncture in patients with osteoarthritis of the knee: a randomised trial. *Lancet* 2005; 366: 136-143.
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5 Ernst E, Pittler MH, Wider B, Boddy K. The desktop guide to complementary and alternative medicine. 2nd ed. Edinburgh: Elsevier Mosby, 2006. ❑