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## Bulk-billing GP clinics did not significantly reduce emergency department caseload in Mackay, Queensland

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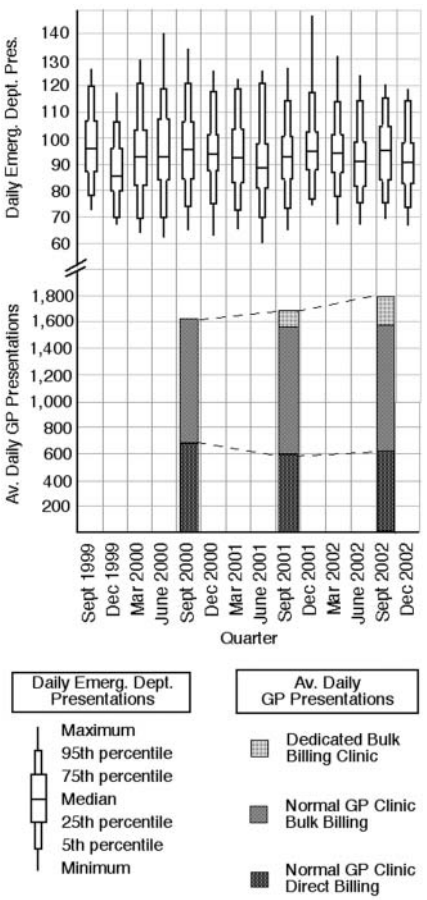
TO THE EDITOR: It has been argued that reduced levels of bulk billing have resulted in emergency department (ED) overcrowding due to an increase in non-emergency, primary care ED presentations.[1] In the 2001–02 financial year, Queensland EDs experienced a 7.1% growth in caseload compared with 2000–01 (Mr D Searle, Surgical Access Team, Queensland Health, personal communication, Nov 2002). During the same period, there was a 1.5% decline in the proportion of general practice services bulk billed in Queensland.

**Effect of general practice bulk-billing presentations on average daily emergency department presentations, Dawson electorate, Queensland**




Before December 2000, no dedicated general practice bulk-billing clinics existed in Mackay. The opening of two bulk-billing clinics, one within 1 km of Mackay Base Hospital, provided an opportunity to assess the effect of the increased availability of bulk-billing services on ED presentations.

The Mackay region had a full-time-equivalent GP : patient ratio of 1 : 1648 in 2002, compared with a Queensland average of 1 : 1143.[2] In the September quarter of 2000, 58.4% of GP consultations were bulk billed in Mackay, compared with 85.7% in Brisbane (Ms D-A Kelly, Federal Member for Dawson, personal communication, Jun 2003).

The Mackay Base Hospital ED provides 24-hour, 365-day emergency medical services to the Mackay region, and managed 34 558 presentations in the 1999–00 financial year, admitting 15% of its caseload. On the Australasian Triage Score (ATS) classification, 0.2% of presentations were category 1, 4% category 2, 20% category 3, 54% category 4, and 21% category 5.

Since the bulk-billing clinics were established, there has been an average of 237 extra bulk-billing consultations per day, with a resultant 7.3% increase in the proportion of GP consultations bulk billed in the federal electorate of Dawson (Ms D-A Kelly, Federal Member for Dawson, personal communication, Jun 2003) (91% of the electorate lives in the Mackay region). However, ED presentations have remained stable, with a median 93 presentations per day (Box). Changes in the proportion of ATS 3, 4 and 5 presentations were observed (25%, 55% and 14%, respectively), but were associated with internal organisational changes (shifting surgical and orthopaedic dressing clinics out of the ED and into the outpatient department, and a review of triage policy), and cannot be reliably attributed to the influence of the bulk-billing clinics.

In Mackay, the implementation of two bulk-billing GP clinics did not result in a measurable reduction in the absolute number of ED presentations. These results are consistent with previous studies that suggest that non-emergency, primary care ED presentations are not a major determinant of ED overcrowding.[3]

*Acknowledgement:* This study was funded under the Priority Driven Research Program of the Australian Health Ministers Advisory Council.

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*COMMENT:* The report by Hanson et al[1] comes at an important time in the debate regarding emergency departments, especially at the onset of winter, a time of high demand and stretched resources. The authors describe the effect of the opening of two bulk-billing clinics on attendances at the emergency department (ED) of a provincial Queensland hospital. Despite the clinics seeing 2.5 times the number of patients seen at the emergency department each day, this resulted in no reduction in ED attendances. This should give pause for thought to those who maintain there is a simple and direct relation between the level of bulk billing of general practitioner services and ED workloads.

The nature of the relation between GP services and ED attendances has never been clearly defined, but it is likely to be complex. Similarly, the nature of ED workloads is also complex, and more than just a matter of the total attendances.[2]

In the Journal last year, Cameron and Campbell cited the major causes of access block and overcrowding as being the reduction in hospital beds and aged care facilities, along with changes in workforce and community attitudes.[3] In that issue, the Journal published a series of articles that essentially represented a national audit of responses to ED overcrowding. Only one article described the opening of a GP clinic as a response; the authors noted that this was unsuccessful in reducing access block.[4]

Thus, the findings of Hanson and colleagues are neither new nor surpris-

ing. Overcrowding is the single most important barrier to quality in ED care. It is a symptom of a serious and growing mismatch between demand and supply for acute healthcare services. Solutions require a whole-of-systems approach. Efforts to improve the flow of patients through acute-care hospital beds are needed, as are strategies to divert some current inpatient flow to community-based subacute services. There is an important role to be played by GPs in coordinating the management of patients with chronic and complex health problems, to reduce the demand for acute-care admissions to hospital, and in working in partnership with the acute-care sector in coordinating community-based subacute alternatives to hospital care. Such initiatives will only come about if state and Commonwealth governments and health departments work together.


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## What explains falling asthma mortality?


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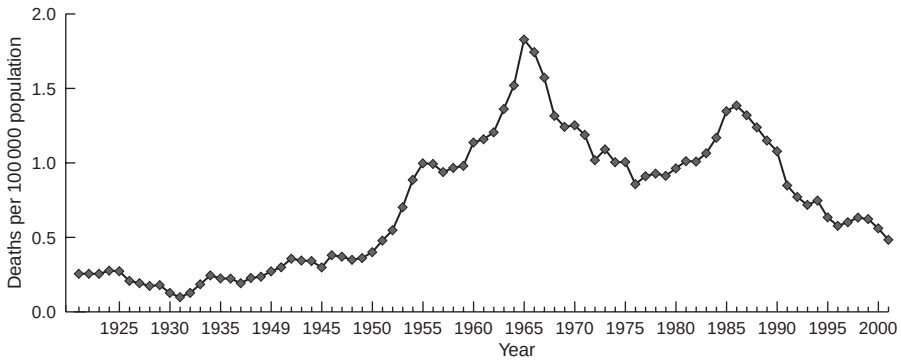
TO THE EDITOR: The Australian Bureau of Statistics recently released details of asthma mortality for 2002. These figures indicate that asthma mortality has continued to decline in 2002 and that deaths in young people aged 5–34 years are at their lowest level since the early 1950s (Box). In this age group, the number of deaths fell from 43 in 2001 to 33 in 2002 (a 23.3% drop), and for all ages the number of deaths fell from 422 in 2001 to 397 in 2002 (a 5.9% drop). This suggests that the various asthma awareness activities, spearheaded by the National Asthma Council and other interest groups, have been successful in raising awareness of asthma and its management. Or does it?

**Asthma mortality in Australians aged 5–34 years, 1920–2002***




* Points on the graph represent 3-year "moving" averages — for example, the 2001 value is the average of 2000, 2001 and 2002 data; the 2000 value is the average of 1999, 2000 and 2001 data, etc. This technique is used to smooth annual fluctuations that occur in data of this kind.

Emerging evidence suggests other changes in the epidemiology of asthma in Australia. Robertson recently reported a 26% decrease in the prevalence of asthma in Melbourne school children between 1993 and 2002, but increased reporting of rhinitis and eczema over the same period.[1] The significance of these findings is difficult to interpret without measures of airway function. A second study supported these results and also observed a small decline in the prevalence of parent-reported asthma, but found little change in atopy or airway hyperresponsiveness.[2] Age-adjusted hospital separation rates for asthma decreased by 31.1% in young people and 31.4% in all ages between 1989–90 and 1999–00.[3]

There is little evidence of improved management of asthma in the general practice setting. Data published from the BEACH (Bettering the Evaluation and Care of Health) survey of general practice activity indicates a significant reduction in rates of presentation for asthma among children but not adults, with no changes in indicators of severity over time.[4] Our recent research in south-western Sydney, examining uptake of the "asthma 3+ visit plan"[5,6] by general practitioners and their patients, is disappointing. It suggests reluctance on the part of both GPs and patients to participate in the plan.

Clearly, there remains much that we do not understand about the natural history of asthma.[7] We need to continue to monitor asthma through regular surveys and routine data collection in order to understand more about fluctuations in asthma prevalence, the relationship to changing child-rearing practices (such as use of childcare facilities) and the impact of management practices.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## Central venous catheters: optimal patient care or convenience?


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TO THE EDITOR: In most serious infections associated with intravascular devices, the device is a central venous catheter (CVC).[1,2] Good clinical practice dictates that these devices should be removed when no longer needed.[3]

**Characteristics of patients and central venous catheters (CVCs) in the audit**

| | General ICU patients | | | Cardiothoracic patients | | |
|---|---|---|---|---|---|---|
| | CVC removed | CVC retained | *P* | CVC removed | CVC retained | *P* |
| Number of patients | 20 | 40 | | 30 | 13 | |
| Number of CVCs | 23 | 51 | | 35 | 17 | |
| APACHE II score (mean [SD]) | 14 (3) | 16 (6) | 0.13 | 13 (4) | 13 (2) | 1.00 |
| Length of ICU stay (d) (mean [SD]) | 3.9 (5.4) | 2.4 (3.5) | 0.2 | 1 (0) | 2.1 (3.7) | 0.1 |
| Ventilation time (h) (mean [SD]) | 61 (86) | 47 (51) | 0.5 | 17 (3.5) | 20 (15.6) | 0.3 |
| % Of patients ventilated* | 60% | 41% | | 100%‡ | 100%‡ | |
| CVC in-situ time (d) (mean [SD]) | 3.2 (3.2) | 7.0 (5.6) | 0.003 | 2.1 (0.25) | 4.5 (3.8) | 0.001 |
| Number of peripheral IV catheters† (mean [SD]) | 1.8 (1) | 0.6 (0.6) | < 0.001 | 0 | 0.07‡ | |
| Number of CVCs reinserted | 0 | na | | 0 | na | |

ICU = intensive care unit. IV = intravenous. na = not applicable.
* Mechanically ventilated in the intensive care unit.
† At 7-day follow up.
‡ Only one peripheral catheter was inserted in one patient.

Our intensive care unit maintains a clinical practice of prompt removal of CVCs once they are no longer required. In addition, CVCs are to be removed before patients are discharged from the unit (for "general" patients) or within 24 hours (for cardiothoracic surgical patients). We conducted an audit to determine how often this practice was followed and whether it had unintended adverse clinical consequences (eg, need to reinsert a CVC).

The audit was conducted over 8 weeks in 2001 and included 126 CVCs in 103 patients. Fifty-eight CVCs (46%) were removed by the predetermined time, and 68 (53%) were retained past this time (Box).

The data demonstrated:

- Low removal rates, with 31% (23/74) of CVCs removed in general patients, but higher rates in cardiothoracic surgical patients (67%; 35/52).
- APACHE II scores, ventilation times and lengths of stay in the intensive care unit were similar in the group who had the CVC removed and the group who retained the CVC, implying that severity of illness was not a factor biasing retention.
- Among patients who had a CVC removed, none had another CVC reinserted; cannulation rates with short peripheral catheters were low and acceptable.
- Retention of the CVC past the predetermined time resulted in significant prolongation of CVC in-situ time (eg, general patients 7.0 v 3.2 days).

The reason given for CVC retention in general patients was antibiotic administration for 37/40 (93%) (vancomycin, 6; β-lactams, 20; aminoglycosides, 9; and quinolones, 2), while total parenteral nutrition and poor peripheral access were factors in only seven (18%). In the 13 cardiothoracic surgical patients, the reason given was inotrope infusion in four (30%) and amiodarone infusion in four; no reason could be ascertained in the other five.

Two issues emerge from this audit. Firstly, there did not appear to be good reasons for retaining many of these CVCs. Drug therapy was most often quoted, but many of these drugs (antibiotics and amiodarone) could have been safely administered via a short peripheral intravenous cannula, with markedly lower risk of infection.[2-5] We believe that when a patient left the intensive care unit with a CVC, it most likely remained in place as a "convenience" factor for busy nursing and junior medical staff on the wards. However, this was at the cost of a significant increase in CVC in-situ times, increasing the risk of both mechanical and infectious complications.

Secondly, implementation of predetermined CVC removal appears safe in our hospital. Although removal rates were lower than expected, nearly half of all CVCs inserted over the 8-week study period were removed as per "clinical practice", without any need for CVC reinsertions.

We have now further refined our clinical practice and introduced a formal written policy that:

- All CVCs should be removed and replaced with a suitable alternative before patient discharge from the intensive care unit, unless there is a specific indication for retention.
- Retention of a CVC should be based on simple guidelines, such as need for total parenteral nutrition, poor peripheral venous access, or use of drugs that require central access.

We recommend implementation of this simple policy in other intensive care units.


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## Q fever in children: an emerging public health issue in Queensland


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TO THE EDITOR: Queensland has a small but increasing number of Q fever notifications in children. This is of concern to public health services in the Darling Downs and south-west Queensland where most Q fever notifications originate (Box). Little is known about Q fever in children, especially in Australia, as the disease is primarily diagnosed in adults following occupational exposure to *Coxiella burnetii*. Here we report a case series examining clinical presentation, exposure to risk factors and disease outcomes in children.

Twenty-one children aged 3–14 years notified with Q fever from the Darling Downs and south-west Queensland in 2001 and 2002 were followed up. In acute cases the febrile illness was similar to that in adults from the same region. The one child with chronic Q fever had no known acute illness and presented with osteomyelitis of the wrist. All the children recovered, although relapsing symptoms were reported in two children and three reported persisting fatigue for 3 months or more after diagnosis.

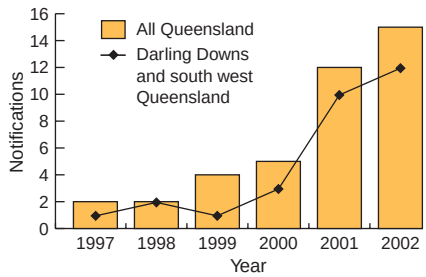
All but one patient reported contact with cattle, sheep or goats (13 lived on a farm and 7 had visited a farm). The single exception reported contact with kangaroos and feral pigs through hunting. Prolonged exposure to animals or the farm environment was not necessary for infection. Twelve of the children had high risk exposure to *C. burnetii* (contact with animal births, newborn animals, or animal carcasses).

With increasing age, exposure of children from rural properties approaches that of their parents as they participate in the same activities. An effective vaccine is available,[1] but use in people younger than 15 years is not recommended because of the lack of safety and efficacy data. Although avoidance of high risk situations such as shearing, animal births or on-farm butchering will decrease the chance of infection, these measures may be impractical for rural children. Realising this, some practitioners choose to vaccinate younger children who assist with animal births and butchering.

Increasing notifications in children may reflect increased awareness that Q fever is not confined to adults with occupational exposures. Increased awareness leading to recognition of infection would give children access to effective treatment and may contribute to prevention of chronic disease. However, much remains unknown about Q fever in children, including how often the infection is asymptomatic, the spectrum and outcome of disease, and if there are effective preventive strategies.

As many rural children cannot avoid potential exposure to Q fever, our study highlights the need for a safe and effective vaccine for children.

**Q fever notifications in children aged 0–14 years, 1997–2002**

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## Border screening for SARS

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TO THE EDITOR: In their article describing the Australian experience of border screening for severe acute respiratory syndrome (SARS), Samaan and coworkers add new insights about the low efficacy of this measure in identifying SARS cases at entry into a country.[1] To our knowledge, this is the first report on this issue from a low-risk area for SARS.[2] Indeed, as summarised by Samaan et al, other available data derive from countries where people with SARS, entering at the early stage of the epidemic, generated a sustained local transmission of SARS-associated coronavirus (SARS-CoV) disease. Among the reasons for a low sensitivity of entry screening, Samaan suggests that subjects may evade screening by making false declarations or by taking antipyretic drugs, or by simply being in the incubation period with no symptoms or only mild symptoms.

To contribute to this debate, we report the experience of our Institute, which was designated as a referring centre for SARS by the Italian Ministry of Health. In Italy, where only four imported probable cases of SARS were identified and no local transmission occurred,[3] entry screening was implemented at the two international airports of Milan and Rome. In particular, travellers and crews arriving from World Health Organization SARS-designated areas, directly or after transiting in other EU countries, were provided with health alert cards and screened for body temperature.[4] Suspected SARS cases identified at Rome airport were to be referred to our institute.

However, of the 72 subjects attending our admission unit for clinical evaluation for possible SARS, none was referred by the airport authorities. Among these patients was one of the four people with SARS arriving in Italy: an airline flight crew member coming from a SARS-designated area who passed both exit and entry screening, despite complaining of mild fever before his departure. He was admitted 6 days after arrival, at which time the clinical picture had evolved into full-blown SARS. He was discharged after 2 weeks.

Among the measures recommended by WHO to reduce SARS-CoV spread, the identification of symptomatic subjects at border departure screening was the only measure with some evidence of efficacy, although this only reduced on-flight transmission.[5]

Conversely, evidence from several sources, including Samaan et al, showed that screening travellers (visual inspection and screening for fever) as they disembark identifies very few SARS cases and is of questionable value.[1] We agree with the conclusions of Samaan et al that, in the light of a possible resurgence of SARS or similar diseases (avian flu), entry screening should, at least, be more focused, and needs further evaluation, including cost-effectiveness analysis.

*Acknowledgement:* Ministero della Salute Ricerca Finalizzata e Ricerca Corrente IRCCS.

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## Using AUDIT to classify patients into Australian Alcohol Guideline categories

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**Alcohol consumption patterns in one general practice in western Sydney, as defined by the recently revised Australian Alcohol Guidelines[1]**

| Characteristic | Males (%) | Females (%) | Total (%) |
|---|---|---|---|
| Abstinent | 18.2 | 29.6 | 25.2 |
| Long-term harm | | | |
| Low-risk | 68.2 | 67.6 | 67.8 |
| Risky | 11.4 | 1.4 | 5.2 |
| High-risk | 2.3 | 1.4 | 1.7 |
| Short-term harm | | | |
| Low-risk | 61.4 | 52.1 | 55.7 |
| Risky | 9.1 | 8.5 | 8.7 |
| High-risk | 11.4 | 9.9 | 10.4 |

Bold text represents categories that cannot be distinguished using AUDIT alone.

TO THE EDITOR: Revised Australian Alcohol Guidelines[1] were released in 2001. Although general practitioners (GPs) can be influential in initiating and supporting behaviour change to reduce levels of alcohol misuse among their patients,[2,3] the extent to which their advice remains relevant and effective depends largely on the extent to which screening tools can be modified to take account of revised versions of such guidelines.

The Alcohol Use Disorders Identification Test (AUDIT)[4] is a clinical instrument used widely to screen patients for problematic alcohol use. The aims of our study were to examine the ability of AUDIT to classify general practice patients' alcohol consumption into the categories specified in the revised Australian guidelines, and to identify any additional information needed for such classification.

Patients aged at least 16 years attending a general practice surgery in western Sydney were asked by receptionists to complete a health-related survey by means of a hand-held computer while waiting for their consultation. Items covered a number of domains, including demographics, the AUDIT, and two additional questions about consumption of specified quantities of alcohol. The use of computers ensured patients were only asked questions relevant to them.

***Risk of harm in the long term:*** Respondents' average number of standard drinks per week was calculated from the first two AUDIT questions, using a previously devised method.[5]

***Risk of harm in the short term:*** AUDIT question 3 is not specific enough to distinguish short-term risk of harm, so additional, sex-specific questions on how many occasions in the previous 30 days the patient had consumed "7–10" and "11 or more" (men) or "5–6" and "7 or more" (women) standard drinks were asked.

Of the 115 patients who completed the survey, 62% were female; their mean age was 42 years; 10% were unemployed; 34% had had tertiary education; 65% were married or in a *de facto* relationship; and 80% were born in Australia. Their alcohol consumption patterns are shown in the Box.

AUDIT is a reliable and valid instrument, and is widely used as a clinical tool. However, as national guidelines are updated, clinical tools such as AUDIT need to remain consistent with them. Ideally, revisions would build on the benefits of existing tools rather than rendering them obsolete. For example, a major advantage of AUDIT is that it measures a number of drinking dimensions within the one, brief, validated instrument. This multidimensionality could be preserved while promoting AUDIT's consistency with new guidelines by adding two items, with high face validity, to more accurately assess risk of harm in the short term. Incorporating the two additional consumption items we used in this study with AUDIT allows drinkers to be classified according to the guidelines as "low-risk", "risky" or "high-risk" both in the long term and short term, with minimal additional response time.

*Acknowledgements:* Thanks to receptionists and GPs in the participating practice in western Sydney, and Hugh Garsden at the Centre for Health Informatics (UNSW) for programming.

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## Acute liver failure associated with the use of herbal preparations containing black cohosh

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TO THE EDITOR: We wish to comment on the case report by Lontos and colleagues on the proposed causal relationship between the herb black cohosh (*Cimicifuga racemosa*) and acute hepatic failure.[1] One other case has been reported in Australia,[2] and the evidence linking black cohosh to liver toxicity was weak and contested.[3]

The medication in the case report presented by Lontos and colleagues[1] included a herb (ground ivy) containing a known liver toxin (pulegone). Pule-

gone is considered a strong hepatotoxin and should not be dismissed, even though it was reported that there was less pulegone in ground ivy than in pennyroyal.

The authors do not indicate the daily dose of the pulegone ingested. The Therapeutic Goods Administration (TGA) made only qualitative analyses of three of the five herbs in the mixture. In our opinion the most suspect ingredient was ground ivy, and it was not assayed. The argument that the TGA could not find a standard for pulegone or ground ivy is untenable given the level of expertise and the capacity of the TGA and its laboratories.

Was the supply company asked to provide analytical evidence of the contents of the herbal extracts? Ground ivy is not known to be hepatotoxic, but it is possible that the extract could have contained ground ivy with pennyroyal, which might explain the hepatoxicity of the mixture. Although uncertain without thoroughly investigating all ingredients in the herbal mixture, this alternative is a possibility. Without thorough investigation of herbal preparations, adverse events attributed to certain herbs remain dubious at best.

Black cohosh has a very good safety record. There is a large body of clinical evidence and research which suggests that this herb has no hepatotoxic effects. Indeed, a German manufacturer has sold more than 350 million daily doses of black cohosh preparations worldwide since the pharmacovigilance system was introduced, and no comparable cases have been reported until these two reports in Australia. An Ames test (salmonella microsomal assay) showed no in-vitro evidence of mutagenic potential of an extract of black cohosh,[4] and no chemical or organ toxicities were observed in Wistar rats given up to 5000 mg of a *Cimicifuga racemosa* extract granulate per kilogram body weight for 26 weeks.[5]

What may also be of concern is that the TGA may not have the full capabilities to test ingredients used in Australian herbal products. That substitution of herbs with potentially toxic herbs may be a common event, and that neither the TGA nor the manufacturers may have the expertise to prevent deleterious contaminants, is of even greater concern.

*Competing interests:* Michael Thomsen had paid consultancy in 2003 with Phytomedicine Pty Ltd, which has a product containing black cohosh; he has no stock, stock options or other financial interests in Phytomedicine Pty Ltd. Michael Thomsen is a director of HerbResearch Pty Ltd, which sells herbal medicines but does not currently have any products containing black cohosh.

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*COMMENT:* Thomsen and colleagues have pointed to what they believe were uncertainties in the case described by Lontos et al, associating the use of a herbal preparation containing black cohosh with acute liver failure.[1] They assert that on the basis of these uncertainties, and other evidence, black cohosh was unlikely to have been the cause of the liver failure.

There are indeed uncertainties about the cause of the illness. The first, and quite critical one, is that we cannot be certain that the patient was given the same herbal materials as those supplied to the Therapeutic Goods Administration (TGA) for analysis. The pharmacist who dispensed the herbal formula for the patient provided the TGA with samples for analysis of each of the five individual extracts in the formula. Documentary evidence supporting the correct identity of the five herbs was supplied in the form of certificates of analysis from the manufacturers of the individual herbal extracts. However, it was not possible to ascertain whether the exact batch of the exact formulation taken by the patient was that which was tested by the TGA.

While the title of the article by Lontos et al suggests a "problem" with black cohosh, the body of the article makes clear that the causative agent(s) are unknown,[1] which is in agreement with the letter by Thomsen et al.[1]

Thomsen and colleagues claim that the TGA did not test the ground ivy extract supplied by Lontos et al, and said that this was because the TGA did not have a standard for pulegone or ground ivy. This is not the case, and the letter by Lontos et al[1] does not state that either. The reason the TGA did not test initially for pulegone was because it is a minor constituent of the essential oil of ground ivy. We did not expect there to be any significant levels of this compound. In fact, we have subsequently confirmed that pulegone was not detectable (limit of detection 5 ppm) in the sample of ground ivy extract provided by Lontos et al.

Thomsen and colleagues refer to the good safety record of black cohosh in Australia and internationally, and claim there has been only one other case in Australia where black cohosh was linked to liver toxicity. Some caution is needed in drawing broad conclusions about the safety of herbal medicines. Most countries do not have adverse reaction reporting systems which include herbal remedies. In Australia, where we have a well-developed reporting system, there have been several reports of liver problems in patients taking various preparations containing black cohosh. However, causality has not been established beyond doubt in these cases.

The TGA has state-of-the-art testing facilities and a team of internationally recognised scientific staff. The formulation supplied to the patient was extemporaneously dispensed by a pharmacist. Such medicines are not subject to the regulatory controls of the TGA and would not normally be included in the TGA's testing program. However, the TGA offered to test the herbal formulation supplied by the pharmacist to assist the clinical team. The results of the TGA's testing confirmed the absence of undeclared pharmaceuticals in the samples of herbal extracts provided.

In view of these uncertainties, it is not possible to conclusively identify the cause of the patient's liver failure. On the evidence available it cannot be concluded that black cohosh was a cause. It is simply not possible to rule in or rule out black cohosh, or indeed any of the other herbal extracts in the formulation taken by the patient, as a cause. Where practitioners suspect a complementary medicine is involved in an adverse reaction, providing the exact product and batch taken by the patient is essential if laboratory testing is to help in confirming causality.

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