LETTERS

Murine typhus: the first reported case from Victoria

Stephanie L Jones, Eugene Athan, Daniel O'Brien, Stephen R Graves, Chelsea Nguyen, John Stenos 482

Sublingual glyceryl trinitrate as prehospital treatment for hypertension in Irukandji syndrome

Mark Little, Peter L Pereira, Richard Mulcahy, Teresa Carrette, Jamie Seymour 482

Geoffrey K Isbister 483

Peter J Fenner 483

Richard J G Bonham 484

Travel insurance and medical evacuation

Peter A Leggat, Robin Griffiths 484

Fred Gilligan, Peter Sharley, Andrew Berry 486

Howard Roby 486

Are the Australian guidelines asking too much of the Pneumonia Severity Index (PSI)?

Kirsty L Buising, Karin A Thursky, James F Black, Graham V Brown 486

El Niño Southern Oscillation and the transmission of hepatitis A virus in Australia

Wenbiao Hu, Anthony J McMichael, Shilu Tong 487

Murine typhus: the first reported case from Victoria

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TO THE EDITOR: Murine typhus (caused by *Rickettsia typhi*) has not been previously described in the state of Victoria, although it is well known in Western Australia, Queensland and South Australia.

In 2002, a 49-year-old man presented to Geelong Hospital, Victoria, with a 10-day history of fever, myalgia, rigors, headache, rash, sore throat, dry cough and pleuritic chest pain. On examination, he had a fever (temperature, 39.2°C), hypoxia (oxygen saturation, 91% in room air), tachycardia, a central maculopapular rash and conjunctivitis. Blood tests revealed hyponatraemia, thrombocytopenia, white cell count in the reference range, with left-shifted neutrophil change (toxic granulation and increased immature forms) and a C-reactive protein level of 377 mg/L (reference range, < 10 mg/L).

The patient lived on a hobby farm close to Geelong. Two weeks before becoming unwell, he had cleaned out the contents of a shearing shed, including two rotten sheepskins in which rats had been nesting. He reported generating a lot of dust and debris in the air. He had not noticed any tick, flea or other insect bites.

Serological testing was performed for rickettsia. Baseline serum, taken 10 days after symptom onset, showed antibodies to the typhus group of rickettsiae, *R. typhi* (murine typhus) and *R. prowazekii* (epidemic typhus), with a titre of 2000. The titre rose over the following 4 days to 64 000, a fivefold increase, diagnostic of typhus group infection. Antibody titre to the spotted fever group of rickettsiae was significantly lower (peak titre, 8000).

The patient was treated with oral doxycycline and recovered completely.

Murine typhus was first described in Adelaide in 1922[1] and is now considered endemic in parts of Western Australia and Queensland.[2,3] A possible case reported from Melbourne[4] was, in retrospect, probably Brill–Zinsser disease (relapsed epidemic typhus). Murine typhus has an incubation period of 8–16 days and is generally self-limiting, although fatalities have occurred.[1] The disease typically presents with fevers, prominent myalgia, a central rash, nausea, conjunctivitis, and often significant pulmonary involvement. Unlike the tick-borne spotted fever group of rickettsiae, *R. typhi* is transmitted by rodent fleas. Transmission occurs either by aerosolisation and inhalation of infected flea faeces, often during demolition or cleaning of rat-infested environments, or, less commonly, by inoculation of faeces into a fleabite.

Murine typhus is usually diagnosed retrospectively by serological testing using microimmunofluorescence. Antibodies are usually detectable 7 to 9 days after disease onset, and IgG may persist for years. Cross-reactivity is seen between *R. typhi* and *R. prowazekii*; it is not possible to identify the pathogen by serological testing alone.[5] Specific diagnosis is based on known local epidemiology and, as epidemic typhus does not occur in Australia, we believe this was a case of murine typhus, the first described in Victoria.

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4. Penfold WJ, Corkill AB. A case of typhus-like fever. *Med J Aust* 1928; 2: 304-306.
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Sublingual glyceryl trinitrate as prehospital treatment for hypertension in Irukandji syndrome

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TO THE EDITOR: We are concerned that a recent report announced that the Queensland Ambulance Service now treats patients with hypertension caused by Irukandji syndrome with sublingual glyceryl trinitrate, based on an uncontrolled, unrandomised, unblinded "trial" of three patients.[1] We believe the authors have made the justification for such treatment on questionable assumptions.

The authors claim that venom from *Carukia barnesi* is a sodium channel agonist that causes massive noradrenaline release. This claim is based on a letter which reported whole *C. barnesi* being crudely blended and injected into piglets.[2] There is no mention in this letter of any proven sodium channel agonist action.[2] We are unconvinced that it was solely the venom of the jellyfish that resulted in a rise in noradrenaline levels in the piglets, especially since there has been a recent report of toxicity from homogenised jellyfish with the nematocysts (ie, venom) removed.[3]

While we believe that magnesium may be a promising treatment for patients with Irukandji syndrome, there has only been one case reported,[4] and we are aware of several anecdotal failures, including the first patient in the letter by Fenner and Lewin.[1] This does not constitute the justification for "proven effective treatment".

Fenner and Lewin claim that patients were envenomed by *Carukia barnesi* and yet provide no evidence. *Carukia barnesi* has yet to be discovered or reported south of Townsville. Two of us have reported that more than one jellyfish is responsible for Irukandji syndrome, and a second jellyfish has recently been identified.[5] Accurate identification of the envenoming animal is essential in toxinology.

Patients who present with Irukandji syndrome are in severe pain, and pain is a well known cause of hypertension. In a series of 116 patients with Irukandji syndrome, the mean dose of morphine administered was 31 mg.[6] In the three patients reported by Fenner and Lewin, all were in severe pain, but only received 10 mg of morphine. In light of subtherapeutic analgesia, attributing the persistent hypertension solely to an unmanageable disease process is therefore premature and possibly incorrect. As a further confounder, might the reduction of blood pressure in two

patients have resulted from the delayed onset of action of intramuscular morphine and promethazine? Without the addition of control patients to this trial, no conclusions can be drawn on the causative factors associated with this treatment.

Too often in the past, unsupported treatments have been adopted, only to be abandoned several years later when shown to be ineffectual, or even dangerous to patients. There are too many assumptions, unsupported claims and confounders in Fenner and Lewin's letter. Toxinology research needs good science and not poor anecdotes.

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3. Sher D, Fishman Y, Zhang M, et al. A new cnidarian noncnidocystic toxic protein. In: White J, Williams V, editors. 14th World Congress on Animal, Plant and Microbial Toxins.14–19 September, 2003; Adelaide. Abstract book. Adelaide: International Society of Toxinology, 2003: 90.
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5. Little M, Seymour J. Another cause of "Irukandji stingings" [letter]. *Med J Aust* 2003; 179: 654.
6. Huynh TT, Seymour J, Pereira P, et al. Severity of Irukandji syndrome and nematocyst identification from skin scrapings. *Med J Aust* 2003: 178: 38-41. ❑

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TO THE EDITOR: Fenner and Lewin describe three cases of Irukandji syndrome in which glyceryl trinitrate (GTN) was used to transiently lower blood pressure.[1] However, they provide no evidence that this relieved the patients' pain or improved their ultimate outcome (development of pulmonary oedema or myocardial injury); they therefore do not show sufficient evidence for instituting such therapy. It is worrying that, with little evidence, sublingual GTN is now recommended as a prehospital treatment in Irukandji syndrome. Although GTN has been used safely in other conditions, its use without adverse effects in three patients is not evidence that it can be used safely in treating jellyfish envenoming.

It is of concern that the treatment of bites and stings remains based on anecdotal evidence, and that clinical toxinology has not moved with the rest of medicine to developing evidence-based approaches. Fenner and Lewin state, in their second paragraph, that intravenous magnesium has proven to be effective in treating symptoms of Irukandji syndrome. This is based on a single case report and no controlled trials. It simply provides a starting point so that properly designed studies can be done to determine if magnesium is effective and safe in Irukandji syndrome. It is not appropriate to suggest that magnesium is a proven therapy without further investigation.

Are we not learning from previous problems in clinical toxinology and prehospital care? Both are areas of medicine where treatment protocols are rarely based on substantial evidence.[2,3] The pressure immobilisation bandage was introduced for the first-aid treatment of *Chironex fleckeri* stings with no supporting evidence, and it has taken two well designed animal studies to show that this is dangerous.[4,5] A recent well designed study in the United States showed that prehospital intubation by paramedics of patients with head injury resulted in transient desaturation in 57% of patients and significant bradycardia (< 50 bpm) in 19%.[6] This occurred despite the paramedics describing the intubation as "easy" in 84% of the patients who desaturated. This shows that it is essential to properly evaluate the safety and efficacy of treatments before recommending them for prehospital use.

Although the infrequency of many envenoming syndromes makes controlled studies difficult to do, it is essential that clinical investigators collaborate to institute such studies. This is now happening in Australia with the commencement of randomised controlled trials on redback spider antivenom, magnesium in Irukandji syndrome and hot-water first-aid in jellyfish stings. Hopefully we will move from anecdote to evidence in clinical toxinology with collaborative studies such as these.

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IN REPLY: We did not provide evidence of any effect on pulmonary oedema, myocardial injury or any effect in stopping pain, as that was not our intention. Also, even intravenous nitrates that have been used for many years in hospital are ineffective in reducing the muscle-cramping pains of Irukandji envenomation, and, although they reduce hypertension and ischaemic myocardial pain, there is no correlation or evidence that they prevent pulmonary oedema or myocardial injury.

The assumption that the Queensland Ambulance Service (QAS) introduced this treatment after our trial is incorrect. The QAS Medical Director will confirm that it was introduced independently of our trial and actually before we treated our first case.

While pain causes hypertension, in our Patient 1 effective analgesia had been attained without any effect on his hypertension. Also, many patients with Irukandji syndrome have pain without hypertension, while others have severe hypertension even with effective pain control.[1] Further evidence that the venom from *Carukia barnesi* is a sodium channel modulator has been submitted for publication (Ken Winkel, Director, Australian Venom Research Unit, Melbourne, personal communication).

Carukia barnesi has now been caught in the Mackay region, 400 kilometres south of Townsville (identified by me, and confirmed by L A Gershwin, PhD student in cubozoan taxonomy, James Cook University, 2003, personal communication). A number of other species can cause the Irukandji syndrome, but the jellyfish causing the sting is rarely caught, so cause and effect are hard to establish at present.

Our aim was to present early evidence that sublingual nitrates *may be* effective in reducing the hypertension that

occurs in some cases of Irukandji syndrome; our suggestion was that they *be further trialled*.[2] It seems worthwhile to do so, especially as sublingual nitrates have also been shown to be effective in dysreflexia from spinal injury,[3] where hypertension also occurs from the massive release of similar catecholamines.

At least two deaths have occurred after Irukandji envenomation, both as a result of cerebrovascular accidents from severe hypertension.[4] When treating a patient with severe hypertension from Irukandji envenomation in a prehospital situation, it is reasonable to try to reduce the hypertension. Doing so might reduce the risk of a cerebrovascular accident and possible death, and so further trials of this prehospital treatment must take place. Other prehospital treatments must also be trialled, especially those for pain. Early promising leads need to be published early for all to evaluate. The Irukandji syndrome is a dreadful experience for the victim; all possible prehospital relief measures must be tried and evaluated.

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COMMENT: I would like to clarify a few points. Firstly, all Queensland Ambulance Service (QAS) paramedics use glyceryl trinitrate (GTN) frequently to treat cardiac chest pain, and have done so for many years. They are very familiar with its effects and interactions.

Secondly, a small number of intensive care paramedics (ICPs), the top tier of officer, with many years of training and experience and tertiary qualifications, have, for a number of years, been authorised to consult with the senior doctor at the receiving hospital for use of GTN to treat acute severe hypertension. In Queensland, GTN has been used for this indication in one or two patients per year, compared with its use in 25 000 patients with cardiac chest pain.

Finally, QAS reviewed its protocol for managing marine envenomation after two people died from Irukandji syndrome two years ago. The current protocol for suspected Irukandji stings is now standard care, including oxygen, and vinegar if there is a visible sting, followed by aggressive pain management, including up to 30 mg of morphine intravenously. If the patient has significant hypertension (> 200/120 mmHg) after effective analgesia, ICP officers are able to consult with a senior doctor to administer GTN according to the above protocol. Obviously, the concern of the QAS Medical Advisory Council (comprising representatives of all medical specialty colleges and other medical groups) was to prevent avoidable cerebral haemorrhage or acute heart failure, as occurred in the two deaths from Irukandji syndrome, but, to date, such use of GTN has not been invoked. ❑

Travel insurance and medical evacuation

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TO THE EDITOR: Grace and Penny present some fascinating "travellers tales" concerning travel insurance and medical evacuation.[1] Although I recognise that there may have been difficulties with individual cases, the article probably paints an unnecessarily bleak picture of travel insurance and medical assistance companies. About a fifth of travel insurance claims involve the successful use of the emergency assistance service, which mostly does not involve aeromedical evacuation.[2] Evacuation by a dedicated air ambulance is uncommon among travellers.[2] Almost all travellers in need of medical and dental treatment source treatment locally, for which they are generally reimbursed. Aeromedical evacuation, where needed, is more likely by scheduled airlines with or without an escort.

The article may raise unrealistic expectations among travellers for aeromedical evacuation. Air ambulances do not operate and respond in the same way as ground ambulances. Time is required to assess and prioritise cases, select suitable aircraft, obtain flight plan clearance, check and equip aircraft, brief retrieval and receiving hospital personnel, and develop contingency plans. Aircraft may be required to refuel en route, as air ambulances tend to be based where they can be maintained and staffed adequately and safely — this may be far from the retrieval site.

It is useful to raise travellers' awareness of the possible difficulties in accessing adequate medical facilities in many developing countries, especially where tourism is promoted, and create a sense of travellers' responsibility for their own health, safety and welfare. The International Society of Travel Medicine has articulated this in a recently released policy statement.[3] Readers should be wary of generalising from a small number of case studies from a "popular tropical island holiday destination".[1] It would be useful to collect data on medical retrievals and emergency assistance provided from various sources and consult all interested parties before attempting to establish guidelines for medical evacuation.

Grace and Penny do raise the important issue of the need for appropriate travel insurance for all travellers. This message needs to be conveyed by the travel industry and by travel health advisers. The article referred to a study of the travel health advice provided by general practitioners in New Zealand, but did not mention that only about half the GPs in that study routinely discussed travel insurance.[4] A similar study in Australia indicated that less than 40% of GPs routinely give advice on travel insurance.[5]

Competing interests: None identified.

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2. Leggat PA, Leggat FW. Travel insurance claims made by travellers from Australia. *J Travel Med* 2002; 9: 59-65.
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5. Seelan ST, Leggat PA. Health advice given by general practitioners for travellers from Australia. *Travel Med Infect Dis* 2003; 1: 47-52. ❑

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TO THE EDITOR: As consultants in intensive care, experienced in transporting critically ill patients within Australia and internationally, we have also received complaints from clients about some travel insurance organisations.[1]

Poor service appears due partly to economic restrictions and partly to the paucity of experienced staff and specialised aircraft available.

Assistance companies implement travel insurance policies for underwriters, quoting 24-hour emergency call centres. Some companies economise by subcontracting (eg, episodic diversion of calls to another organisation). This can result in coordinators lacking an understanding of regional geography, population and medical services and omission of the early, vital input of senior medical advisers. Information relayed between several people can be lost or distorted. Failure to ask key questions can result in inappropriate clinical planning. Furthermore, time zone differences can result in calls being received late at night, further reducing availability of immediate expert opinion. Only a well-organised (and thus expensive) control centre with a critical care focus can manage all these variables.

Furthermore, it is difficult for a company to permanently employ current, high-grade healthcare staff in adequate numbers. Current critical care retrieval staff in Australia are confined to a few stand-alone aeromedical organisations or public hospitals which run aeromedical services for state governments and other organisations, using their regular anaesthesia, intensive care or emergency medicine staff. Reliable assistance companies tend to contract with these retrieval organisations or their off-duty staff.

In Australia, ambulance aircraft able to travel offshore, with adequate oxygen systems, stretcher attachments, electrical power, and so on, are uncommon and expensive to equip. Portable equipment to care for a critically ill patient represents a capital investment of over $100 000, and some services try to achieve results with inadequate tools or by borrowing.

ISAS (the International Society of Aeromedical Services, Australasian Chapter) promotes standards on staffing and equipment,[2] including the Australian and New Zealand College of Anaesthetists/Australian College for Emergency Medicine standard for transporting the critically ill.[3] Based on critical care practice, the standards are not legally binding, but one suspects they would be quoted in any litigation.

It behoves all travellers to scrutinise their travel insurance policies closely — many think of them only in terms of lost or stolen baggage. Following serious injury or illness, the policy may dictate what kind of care is offered.

Competing interests: None identified. The authors' retrieval services are based in public hospitals and government funded.

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TO THE EDITOR: As the medical director of Customer Care Medical Assistance, which manages the travel insurance policies of most travelling Australians, I wish to reassure readers that none of the experiences chronicled by Grace and Penny[1] related to our company. I have previously described the activities of Customer Care in the Journal.[2]

I would welcome any enquiries from colleagues about the way our company functions.

I note that there was no declaration by Grace and Penny of their competing interests.

Competing interests: Customer Care Medical Assistance is a private commercial enterprise.

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2. Roby HP. Aerial evacuation of sick travellers. *Med J Aust* 1994; 161: 636-637. ❑

EDITOR'S NOTE: In their statement about competing interests, Grace and Penny wrote:

"For 5 years Grace worked for the Ministry of Health in Vanuatu on a program sponsored by AusAID. At the time of publication of the article, he was not employed.

Penny worked for the Ministry of Health for 2 years, then ran a private ambulance service (Promedical) in Vila. Promedical is not a retrieval service. At the time of publication, Promedical was a community-funded service and Penny was a salaried ambulance officer.

Neither of us have offered for sale or recommended or had any pecuniary interest in any form of travel or medical evacuation insurance." ❑

Are the Australian guidelines asking too much of the Pneumonia Severity Index (PSI)?

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TO THE EDITOR: The 2003 Australian guidelines on antibiotic therapy suggest that the Pneumonia Severity Index (PSI) may be used to triage site of care and antibiotic selection for patients with community-acquired pneumonia.[1,2] The PSI was developed as a mortality prediction tool, using data from over 14 000 patients with community-acquired pneumonia.[3] The antibiotic guidelines suggest specifically that PSI classes I and II represent patients suitable for outpatient therapy, and that class V can identify patients likely to require intensive care and broad-spectrum antibiotic therapy. We believe this is beyond the previously recommended applications of the PSI and advise caution about its use to identify patients with severe pneumonia.

In the cohort used to validate the PSI, only 32% of patients with severe

Suggested alternative approach to assessing patients with community-acquired pneumonia

Step 1: Does the patient need admission to hospital?

Assess with the Pneumonia Severity Index (PSI).

- Class I or II: consider outpatient management (but also need to consider comorbidities, social supports, likelihood of compliance).
- Class III-V: likely to need inpatient management.

Step 2: Does the patient need admission to the intensive care unit?

Assess with the modified British Thoracic Society (BTS) Severity Score.

- Class as severe if two or more of the following features are present on initial assessment or within 24 hours of presentation (and are not attributable to another cause):
  - ➤ Confusion (acute onset)
  - ➤ Serum urea level > 7 mmol/L
  - ➤ Respiratory rate ⩾ 30 breaths/minute
  - ➤ Systolic blood pressure < 90 mmHg or diastolic blood pressure ⩽ 60 mmHg
- If severe, discuss the case with a senior clinician and consider intensive-care review and aggressive broad-spectrum antibiotics.

pneumonia (requiring intensive care) were in class V, indicating that the PSI has poor sensitivity for severe pneumonia.[3] This finding has been reflected in other studies.[4] The strength of the PSI lies in its ability to identify low-risk patients, as the title of the validating article suggests.[3] The PSI is so heavily weighted by age and comorbidities that younger patients needing intensive care are unlikely to accumulate enough points to reach class V. This is important, as early identification of patients with severe pneumonia and initiation of broad-spectrum antibiotic therapy and intensive-care support improves outcomes.

We are concerned that the PSI may be widely accepted for a purpose for which it was not intended and has not been validated. In underestimating the severity of illness in two-thirds of patients with "severe pneumonia", the guidelines may provide false reassurance, while clinicians may lose confidence in the PSI if they find it "misses" most patients requiring intensive care.

Current evidence does support use of the PSI to guide decisions about inpatient or outpatient therapy. However, the modified British Thoracic Society (BTS) Severity Score is a simpler, better-validated tool to identify patients with "severe pneumonia" who are likely to need intensive care assessment[5] (Box). This tool is useful for junior staff to "flag" patients with potentially severe pneumonia and ensure that they are discussed with a senior clinician. As always, the final management and antibiotic selection should be guided by clinical judgement. We believe that the antibiotic guidelines are valuable to encourage appropriate antibiotic use; our aim is to promote discussion of their content relating to this particular condition.

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El Niño Southern Oscillation and the transmission of hepatitis A virus in Australia

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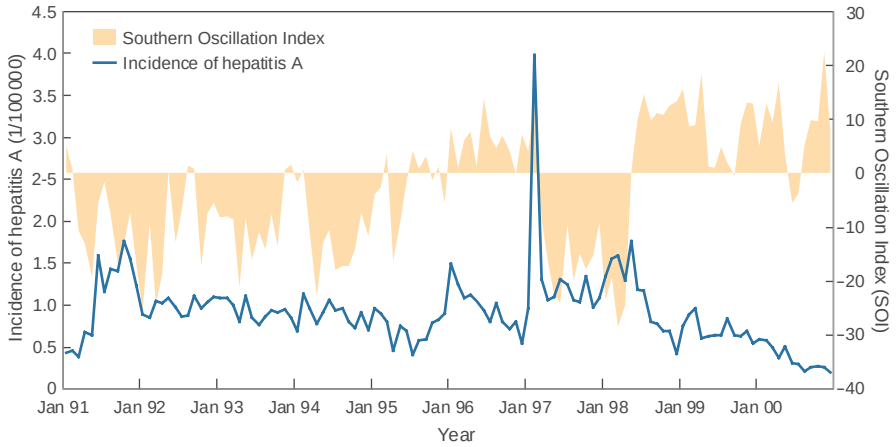
TO THE EDITOR: We examined the possible association between the Southern Oscillation Index (SOI) and the occurrence of hepatitis A in Australia using a Seasonal AutoRegressive Integrated Moving Average (SARIMA) regression model.[1] Our results indicate that the SOI is statistically significantly associated with the transmission of hepatitis A.

We obtained data on the monthly counts of hepatitis A cases in Australia and the monthly SOI between 1 January 1991 and 31 December 2000 from the Commonwealth Department of Health and Ageing and the Australian Bureau of Meteorology, respectively. Data on population sizes were obtained from the Australian Bureau of Statistics. Cross-correlations were used to compute a series of correlations between SOI and the incidence of hepatitis A over a range of time lags (defined as the time span between the SOI and the incidence of hepatitis A).

A SARIMA model was used to estimate the independent contribution of SOI in our study. We adjusted for seasonality by "seasonally differencing" (ie, replacing each observation by the difference between it and the observation from the previous year). In the modelling process, attention was paid to observations well outside the main body of the data (outliers) and the only outlier was excluded in the final SARIMA model. We used the SARIMA $[1,0,0]$ $[1,1,0]_{12}$ model (ie, first-order autoregressive combined, first-order seasonal autoregressive, after adjustment for first-order seasonal integration) to assess the association between SOI and the incidence of hepatitis A.

Incidence of hepatitis A in Australia and the Southern Oscillation Index between January 1991 and December 2000



Negative values of the SOI are associated with El Niño conditions (dry and warm in Australia), and positive values with La Niña conditions (wet and less warm in Australia).

We found that a decrease in the SOI (ie, warmer and drier conditions) was statistically significantly associated, at a lag of 1 month, with an increase in the monthly incidence of hepatitis A ($\beta = -0.01$; $P = 0.001$). Two El Niño events (1991–92 and 1997–98) were also clearly associated with an increased incidence of hepatitis A (Box).

The results suggest that there was an increase of about 360 cases per year in Australia for an, on average, interquartile range decrease in the SOI. The residuals in the model fluctuated randomly around zero, and there was no apparent autocorrelation between residuals at different lag times (data are available from the corresponding author).

These results indicate that the model fitted the data well, with no violation of assumptions. The significant association between SOI and the incidence of hepatitis A remained when the outlier was included in the model ($\beta = -0.013$; $P = 0.001$).

El Niño Southern Oscillation (ENSO) has been found to be related to various health outcomes, including waterborne disease, vectorborne disease, and natural disaster-related deaths (eg, floods, bushfires and cyclones).[2-4] Our study adds further evidence of ENSO-related health effects. Infectious diseases are, in general, sensitive to climate variability, as climate can influence the development and transmissibility of pathogens, and can also affect people's behaviour.[3,5] If the relationship between ENSO and hepatitis A is confirmed by other studies, these findings may facilitate the development of early warning systems for controlling and preventing this widespread com-municable disease.

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