LETTERS

Overweight and obesity from childhood to adulthood: a follow-up of participants in the 1985 Australian Schools Health and Fitness Survey

Margaret A Allman-Farinelli, Lesley King and Adrian E Bauman

To the Editor: The recent article by Venn et al reported that childhood overweight carries through into adult overweight and obesity, but that most obese young adults in their study were "healthy" weight as children in 1985.[1] As demonstrated by National Health Surveys, age is one of the strongest predictors of overweight,[2] with body mass index (BMI) increasing as we grow older. However, there are two additional time-related components influencing obesity.

Since 1985 (when Venn et al reported the prevalence of overweight and obesity in children was less than 10%), the environment appears to have become more obesogenic — a 2004 survey in New South Wales showed that 26% of children were overweight or obese.[3] It is not only children who are vulnerable — the percentage of overweight adult Australians increased for almost all age groups from 1990 through 2001, and the mean BMI at which Australians enter adulthood has increased with each subsequent survey. For example, for women aged 20–24 years, mean BMI increased from 22.1 kg/m^2 (1990) to 22.5 kg/m^2 (1995) to 23.2 kg/m^2 (2001) to 23.3 kg/m^2 (2004). As the heights and weights were self-reported in these surveys, true BMI values may be even higher.

We recently reported that year of birth (birth cohort) also predicts prevalence of overweight and obesity, independent of age and survey period; the prevalence of overweight and obesity in adults increased progressively with birth cohorts born since 1960.[4] This birth span includes the cohort in the study by Venn et al.[1] While obesity begins in childhood for only a small proportion of adults, the so-called healthy weight children now have a higher mean BMI, giving little margin for the seemingly inevitable increases in weight with ageing, before the population mean BMI reaches the cutpoint for overweight and later obesity. The 2004–2005 National Health Survey showed that men reached the overweight cutpoint at 25–29 years and women reached it at 30–34 years.[5]

Given increasing child and adult obesity, the need for allocation of public health resources to improve dietary and physical activity habits is undisputed. However, these data[1,4] indicate that efforts should be directed to the hard-to-reach group, young adults, to prevent weight gain at this point. This will pose considerable challenges, because this group has minimal contact with health services, and perceives the threat of chronic illness as irrelevant. However, swift intervention is required, not only for their own health and that of their children as they become parents, but also because they will become overconsumers of health care for chronic diseases within a generation.

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In reply: Allman-Farinelli et al make an important point about the influence of age, survey period and cohort effects on the prevalence of overweight and obesity. While age and cohort effects could not be clearly separated in the 1985 Australian Schools Health and Fitness Survey, the prevalence of overweight and obesity increased with age in 7–15-year-olds.[1] Our data collected from 4571 of the individuals in that survey at follow-up about 20 years later also showed an increase in the prevalence of overweight and obesity with age, although these findings were not presented in our report.[2]

In the Box, we show the distribution of body mass index (BMI) values for men and women in three age groups (24–27 years, 28–30 years and 31–34 years). Mean BMI values across the age groups were 25.2 kg/m^2, 25.6 kg/m^2, and 26.5 kg/m^2 in men and 23.5 kg/m^2, 24.2 kg/m^2, and 24.6 kg/m^2 in women. The prevalence of obesity (BMI $\geq$ 30 kg/m^2) increased with increasing age as follows:

- age 24–27 years — men 12.2%, women 9.9%;
- age 28–30 years — men 12.3%, women 12.0%; and
- age 31–34 years — men 15.6%, women 14.6%.

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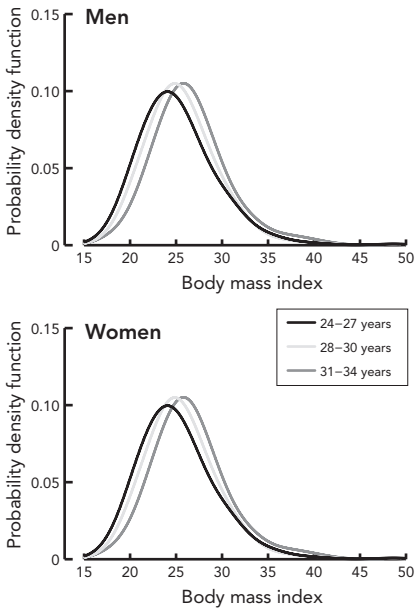
Distribution of body mass index values for men and women in three different age groups*



* 24–27 years, 757 men and 854 women; 28–30 years, 767 men and 807 women; and 31–34 years, 673 men and 691 women in the 20-year follow-up of the 1985 Australian Schools Health and Fitness Survey. ◆

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A review of policies on alcohol use during pregnancy in Australia and other English-speaking countries, 2006

Ruth Morley, Jane L Halliday and Susan M Donath

To the Editor: O'Leary and colleagues[1] rightly point to the need for better evidence on whether low to moderate maternal alcohol intake affects the fetus. Evidence to date is weak and inconsistent, largely because alcohol consumption in pregnancy is generally poorly documented,[2] and few studies have recorded data on factors that could potentially modify fetal exposure. Evidence could come from large pregnancy cohort studies, usually designed to address other issues, but there are no published guidelines on how to collect information relevant to fetal alcohol exposure during gestation. There is, therefore, a great need to define a core dataset for research studies, as well as one that is sufficiently simple to use in routine pregnancy care settings.

We have identified a number of issues that need to be addressed. To stimulate discussion, the Box shows our suggested core dataset.

Maternal alcohol intake: Alcohol questionnaires have largely been designed to identify women who are heavy drinkers, misuse alcohol or are alcohol-dependent.[3] Questionnaires are needed that capture information on alcohol intake across the range, from minimal to heavy drinking, as well as information on drinking patterns and alcohol intake at different periods of gestation.

Factors that may modify the relationship between maternal intake and fetal alcohol exposure: For a given maternal intake over a given period, maternal blood alcohol level and hence fetal alcohol exposure may vary according to maternal size and body composition. Other factors can affect maternal alcohol absorption and elimination, such as whether alcohol is taken with food,[4] and possibly maternal genotype.[5] This information is rarely reported in pregnancy studies.

Factors that may modify effects of alcohol on the fetus: There is animal evidence that maternal micronutrient supplementation may protect the fetus against some of the adverse effects of gestational alcohol exposure.[6] We need to consider recording supplement use and measures of maternal nutritional intake or status. In well resourced studies, fetal genotype could also be considered.[6]

Researchers with expertise in the field need to reach consensus and provide guidelines on the best way to assess fetal alcohol exposure, so that pregnancy researchers and clinicians with little experience of alcohol research do not need to create their own. Better data should provide better evidence on which to base advice to women who are (or may be) pregnant. Good studies may also provide explanations for the apparently variable link between maternal alcohol consumption and adverse sequelae in the offspring.

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1 O'Leary CM, Heuzenroeder L, Elliott EJ, Bower C. A review of policies on alcohol use during pregnancy in Australia and other English-speaking countries, 2006. *Med J Aust* 2007; 186: 466-471.
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Suggested core dataset for studies of maternal alcohol intake during gestation and outcome of offspring

As a basis for discussion, we suggest the folllowing dataset:

Baseline

- Weeks of gestation at pregnancy recognition, or expected date of delivery and date that pregnancy was recognised (to allow calculation)
- Height

For specific periods of gestation (eg, from date of start of last menstrual period until pregnancy recognition; from pregnancy recognition to 12 weeks' gestation; from 13 to 28 weeks; and from 28 weeks to term)

- Body weight (eg, at 12 and 28 weeks)
- Alcohol intake — we suggest:
  - ➢ Average number of standard alcoholic drinks per week;
  - ➢ Average number of days per week on which alcoholic drinks are taken; and
  - ➢ Maximum number of drinks on one occasion.
- Whether alcohol is taken with or soon after food (never, sometimes, usually or always)
- Dietary intake of fruit and vegetables
- Use of nutritional supplements ◆

Sue Miers

To the Editor: I would like to put forward a consumer's perspective in response to the recent article by O'Leary and colleagues.[1]

I am puzzled that they concluded that the National Health and Medical Research Council (NHMRC) guideline is in step with policies of the United Kingdom and Canada. My research of Canadian policy suggests that it is in direct contrast to current NHMRC guidelines. Health Canada states very clearly, "Whether you are trying to get pregnant or are pregnant already, stop drinking alcohol",[2] and "No amount or type of alcohol during pregnancy is considered safe".[3,4]

The potential harm to the birth mother that results from an abstinence-based message was also raised by O'Leary and colleagues. Elizabeth Russell, the birth mother of two sons affected by prenatal exposure to alcohol, offers an alternative viewpoint:

> By not discussing alcohol and pregnancy through misplaced compassion, we are hurting one person for the sake of another. Very few mothers would want that but that is exactly what is happening — children are being sacrificed to ensure that the anxiety level of a mother is kept within acceptable limits — neither mother nor child will benefit from this methodology.[5]

Many pregnant women give up eating shellfish and processed meats and drinking coffee but still continue to consume alcohol, thinking it is safe because they have not been told otherwise. I would like to pose the following questions.

What is the potential harm of not having an abstinence message? Might this prevent women who are alcohol-dependent from seeking help to alter their drinking behaviour when pregnant because they are not aware of the risks?

Should further research to "elucidate the true association between low to moderate alcohol consumption and fetal harm" really be a priority? What are the benefits to the unborn child of trying to ascertain a safe level of consumption of a teratogen and neurotoxin that is known to disrupt fetal development, particularly the fetal brain, throughout the three trimesters of pregnancy?[6]

While I acknowledge the importance of scientifically sound research on the risks of prenatal exposure to alcohol, I am concerned that this argument is diverting attention and dollars away from the urgent need for diagnosis and management of fetal alcohol spectrum disorder (FASD) in Australia.

The reality is that children, adolescents and adults with FASD are seldom recognised, seldom treated effectively, and seldom connected to service dollars. Addressing this situation needs to be the priority.

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Colleen M O'Leary,
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In reply: Morley and colleagues and Miers raise a number of interesting discussion points. As we reported in our policy review, the Canadian, United Kingdom and Australian guidelines have similar intent but differ in emphasis.[1] Health Canada's policy position is that, although abstinence is the prudent choice, fetal risk is relative to the amount of alcohol consumed and is minimal with low levels of maternal alcohol intake. Australian policy addresses the same issues, with less emphasis on abstinence and more on avoiding intoxication and ensuring low-level alcohol consumption. Since our review was published, UK guidelines have been reframed to emphasise abstinence, but their message has not changed — they now place more weight on avoiding alcohol during pregnancy.[2]

We strongly agree with Morley and colleagues that screening for alcohol should be routine in all pregnant women, and that standardised items should be included in a core dataset.[3] If this were implemented, Australia would be in a unique position to make a valuable contribution to alcohol and pregnancy research.

Miers comments that research into the impact of low to moderate alcohol exposure during pregnancy should not be a priority, because it may direct "attention and dollars away from the urgent need for diagnosis and management of fetal alcohol spectrum disorder". Although we agree that specialised clinical services are important — and lacking — in Australia, it is short-sighted to suggest that there is no need for further research to establish the true risks from low to moderate alcohol consumption. Rates of alcohol consumption in Australia are high: about 80% of women report alcohol consumption in the 3 months before pregnancy, 14% report binge drinking, while 47% report that pregnancy was unplanned.[4] Many fetuses may thus be exposed to alcohol before women are aware they are pregnant. Unfortunately, many women are unable to stop drinking and may expose their babies to high alcohol levels. Health professionals need to have a true estimate of risk to the fetus and know what additional factors (eg, genetics and nutrition) may alter risk, and women deserve to be well informed. The need for research is well articulated by Morley et al.

Research evidence in humans does not clearly indicate a risk to the fetus from low levels of alcohol consumption, and this has led to inconsistent policy.[5,6] As we point out, the potential for harm from an abstinence message should be considered when Australian alcohol guidelines are reframed. Whatever the policy, it needs to be widely disseminated, in a "digestible" format, to health professionals and the community. Research being conducted at the Telethon Institute for Child Health Research is evaluating educational materials for health professionals about alcohol and pregnancy.

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A case of Kawasaki disease mimicking acute appendicitis

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To the Editor: Kawasaki disease (KD) is an acute vasculitis of unknown aetiology occurring mostly in infants and young children. KD is characterised by fever of more than 4 days' duration; conjunctivitis; rash;

cervical lymphadenopathy; erythema of the lips, oral mucosa, palms and soles; and oedema of the hands and feet.[1]

Coronary artery aneurysms develop in 15%–25% of untreated children,[2] with attendant risk of ischaemic heart disease, myocardial infarction and sudden death.[3,4] Treatment with intravenous immunoglobulin (IVIG) within the first 10 days reduces the incidence of aneurysm to less than 5%.[4] A KD diagnosis is clinical, based on the recognition of a characteristic set of signs and symptoms.[4] The 10%–45% of children who meet only some of the classical criteria are said to have "atypical" or "incomplete" KD. These children have a higher risk of coronary artery aneurysm than children with typical KD.[4] Abdominal symptoms, including acute appendicitis and appendicular vasculitis, can occur before the development of classical features of KD.[4,5] A 50% coronary artery aneurysm rate has been reported in children with KD and acute abdomen. It is still unclear whether this reflects a delay in diagnosis and treatment or is a marker of a more severe vasculitis involving the intestinal tract.

A 3-year-old boy presented with a 2-week history of remittent, high-spiking fever (37.5–39.0° C; 2–3 spikes/day), right lower quadrant abdominal pain, and McBurney's sign with rebound tenderness. Abdominal ultrasonography suggested a diagnosis of acute appendicitis with peritonitis. The postoperative diagnosis was appendicular vasculitis with peritoneal inflammation and serous secretion.

Fever persisted despite treatment with intravenous cephalosporin. Several days later, the boy developed conjunctivitis, cracked lips, a raised erythrocyte sedimentation rate and C-reactive protein level, and thrombocytosis (715 × 10^9 platelets/L). KD was suspected, and an echocardiogram revealed two sacciform coronary artery aneurysms (diameters, 3.1 mm and 2.9 mm) in the proximal part of the common trunk. The child was given IVIG (2 g/kg) and oral acetylsalicylic acid (100 mg/kg per day in four divided doses). As the fever failed to resolve, the patient was given a second dose of IVIG,[4] this time leading to a dramatic clinical improvement. Five days later, he developed oedema of the hands and periungual peeling of the fingers. His aspirin dose was reduced to 5 mg/kg/day. Follow-up echocardiograms at 3 months and 6 months demonstrated persistent coronary artery dilatation.

Persistent fever with conjunctivitis and cracked lips should alert clinicians to the possibility of KD. At our patient's age, acute appendicitis is rare, and other causes of abdominal pain must be excluded. In this case, the unusual postoperative course, with persistent fever even after antibiotic treatment, was another clue to establishing the correct diagnosis.

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Late-term abortion: what can be learned from *Royal Women's Hospital v Medical Practitioners Board of Victoria*?

Joanna M Flynn

TO THE EDITOR: Gerber's article[1] about the case *Royal Women's Hospital v Medical Practitioners Board of Victoria* raises important issues but contains significant errors.

The *Medical Practice Act 1994* (Vic) states that the main purposes of the Act are "to protect the public by providing for . . . investigations into the professional conduct . . . of registered medical practitioners" — Section 1(a). Section 22(1) makes it clear that any person may notify the Board if they believe a person may have engaged in unprofessional conduct. Section 25(1) states that the Board must investigate notifications unless they meet certain criteria, which the Board did not believe were met in this case.

Gerber is not correct in stating that the Board made no attempt to question the medical practitioners involved. In the absence of the patient's consent, all but one of the doctors refused to provide any information in response to the complaint.

Gerber's statement that the subcommittee of the Board that conducted the preliminary investigation concluded that the complaint was "frivolous and vexatious" is wrong. The subcommittee recommended that the matter be closed. Later, the full Board chose not to accept this recommendation, as the investigation had been hampered by lack of information, including access to the original hospital records.

While formal hearing panels have the power to subpoena documents or persons, this power does not extend to the Board's preliminary investigations. The Board does have the power to apply to a magistrate for the issue of a search warrant.

Gerber refers to the powers of the Board under Section 48 and Section 49 of the Act. These powers only come into play if the Board has determined that it will conduct a formal hearing. He is also incorrect when he states that the Board can compel medical practitioners to appear before the Board. This power is not available during preliminary investigations.

The considerable delay in the matter being finalised was due to the legal appeals mounted by the Royal Women's Hospital against the decision of the Magistrate to allow the Board access to the hospital records.

Another issue raised by Gerber requires clarification: the Board does not currently have the power to conciliate disputes or conduct mediations.

The Board did not ultimately dismiss the matter as frivolous and vexatious. When the subcommittee, having been provided with the records, reported to the Board that it did not find evidence of unprofessional conduct, the Board closed the investigation.

I trust that this information will correct the public record on this important matter.

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1 Gerber P. Late-term abortion: what can be learned from *Royal Women's Hospital v Medical Practitioners Board of Victoria*? *Med J Aust* 2007; 186: 359-362. ❑

Paul Gerber

IN REPLY: Flynn points to some minor technical differences in my historical recount of the handling, by the Medical Practitioners Board of Victoria, of the complaint against the medical specialists

involved in a late-term abortion.[1] None requires a reply, save for Flynn's assertion that "the Board does not currently have the power to conciliate disputes or conduct mediations".

The Board does not require statutory power to approach a hospital in a conciliatory manner so as to explore whether an impasse, involving confidentiality, can be resolved without recourse to litigation. Was the Board's only remedy to raid the hospital, trawling for evidence to decide whether there were grounds for the possible suspension or cancellation of registration of the doctors involved in the complaint?

We both agree that the relevant legislation mandated the Board, on the material before it, to investigate the charge of serious professional misconduct. Where we disagree is that, having overruled its own subcommittee's recommendation that the matter be closed, the Board failed (I maintain) in its statutory duty to promptly institute a formal hearing. Had the various specialists been subpoenaed, this would have cleared them of professional misconduct, thereby preventing the considerable and unnecessary stress to these witnesses over a period of 5 years.

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1 Gerber P. Late-term abortion: what can be learned from *Royal Women's Hospital v Medical Practitioners Board of Victoria*? *Med J Aust* 2007; 186: 359-362. ❑

Medical professionalism: it is really under threat?

George Halasz

To the Editor: Breen's timely call for a reality check on medical professionalism noted major global changes that affect contemporary doctor–patient relationships: new technology, changing market forces, evidence-based treatment protocols, and resource-driven health services and policies.[1] The call by our colleagues in the United States and United Kingdom to restore "trust that the public used to have in the profession" was urgent.

Breen's thesis posits that lost trust is due to an "altered balance" of ethical issues faced by doctors because of a generational shift from the ethical principle of "beneficence" to "autonomy" to "justice" and "distributive justice".

This view contrasts with Green and Bloch's analysis of the mental health care system's ethical concerns arising from an adherence to "efficiency-driven" policies that started in many countries during the 1980s.[2] They suggest the system itself is flawed.

Green and Bloch suggested that the legacy of efficiency-driven policies created two current moral compromises for our profession: first, a threat to the "ethic of agency"; second, the constraints those policies imposed on ethical principles precisely because they were not based on justice, instead being created to meet wider socioeconomic and political considerations. Their views point to the heart of the matter, beyond Breen's suggested remedy to be found in "stronger leadership", which may be necessary, but is not sufficient without an urgent update on personal medical ethics.

At the individual doctor's experience, Green and Bloch locate conflict arising from competing interest when doctors' "principle of fidelity is juxtaposing their financial interests alongside patients' needs". In the US, a study found 28% of physicians receive direct payment for consulting, lectures or enrolling patients in trials, and 94% report "some type of relationship with the pharmaceutical industry".[3] To avoid the conundrum posed by the ethics of conflict of interests we face when confronted by these physician–industry relationships, or by efficiency-driven policies, risks perpetuating the very loss of trust that we need to restore.

Breen's analysis, an important step in the needed debate on medical professionalism, should account for not merely shifts in the ethical balance, but also the incremental erosion of trust. As it stands, he expressed our very Australian attitude "she'll be right". Our overseas colleagues, as well as locals, have suggested that "she won't be right, mate" when it comes to managerialism eroding the ethical foundations of medicine.[4]

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Kerry J Breen

In reply: My recent article was submitted under the category of "For Debate", so it is pleasing that Halasz has joined the debate. I am disappointed that he interprets my view as "she'll be right". My point is that revising or repackaging existing ethical codes will not, on its own, fix any of the perceived problems of "managerialism eroding the ethical foundations of medicine". Working constructively, consistent with existing ethical codes, within our health care system, as is also suggested by Green and Bloch,[1] is more likely to achieve better outcomes for our community. As I stated and as Green and Bloch imply, this will not always be a simple matter.

I am in fierce agreement with Halasz over steps to reduce erosion of trust,[2] but that was not the focus of my article.

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1 Green SA, Bloch S. Working in a flawed mental health care system: an ethical challenge. *Am J Psychiatry* 2001; 158: 1378-1383.
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Inequitable provision of optimal health services for patients with chronic heart failure: a national geo-mapping study

Elizabeth A Dodd

To the Editor: Clark et al have claimed to map the distribution of services for people with chronic heart failure (CHF) against the distribution of these people.[1] An examination will show that they have mapped the distribution of people likely to have CHF, using age and Aboriginality as surrogate markers. The stated mapping of the services shows the services probably available to these people.

A map is drawn to show us what the cartographer wants us to see.[2] The authors note that high prevalence in remote regions has been shown on the maps, but they have not considered different mapping methods to provide a better representation of their results.[3] This has led to an anomaly so that, when calculating numbers of people with CHF, the maps show remote areas with giant households containing between 24 and 300 people.

CHF programs were located by a snowball sampling technique, which by its nature

will miss isolated examples.[4] Isolation is a feature of rural and remote practice, so this method is biased to finding metropolitan examples. In my own rural practice in Griffith, I found a local program that had been operating in 2004 and a distance program run by a health fund, neither of which had been identified by Clark et al.

On the medical front, the authors asserted that access to a CHF management program is a mark of equity in health services. This is based on a metro-centric model looking at admission with CHF to a major teaching hospital, and showing that rates of readmission and death were reduced where a nurse and a pharmacist made a single visit to someone with CHF in their home after hospital discharge. The relevance of this activity to a rural person with a single community pharmacist who may make a home visit, a single point of contact with medical services in their general practitioner, and the possible availability of a community nurse to visit them regularly is unproven. Perhaps the city folk were copying the principles of the services we already had?

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CORRECTION:

Re: "**Chocoholic**" in *In Other Journals* in the 4 June issue of the Journal (*Med J Aust* 2007; 186: 604). The item referred to "an intake of between 46 and 100 mg of polyphenol-containing dark chocolate per day". The intake amount should have been stated as "between 46 and 100 g". ◆

Robyn A Clark, Andrea Driscoll, Justin Nottage, Skye McLennan, David M Coombe, Errol J Bamford, David Wilkinson and Simon Stewart

In reply: We thank Dodd for her commentary on our article.[1] We concur with many of the highlighted issues relating to our suboptimal response to the burden and management of chronic heart failure (CHF) in rural and remote Australia. These include the lack of rigorous epidemiological data and lack of specialist services "beyond city limits".

Unfortunately, we have limited space to respond fully. However, we re-emphasise that, although our previous estimates[2] complement that of the Canberra Study,[3] neither can replace an Australia-wide study of CHF that samples metropolitan, regional, rural and Indigenous communities. We also stand by (within the context of the stated limitations) the accuracy of our mapping of the CHF programs and the location of general practice services in Australia for the study period. Our geomapping approach and data have been well validated by the National Centre for Social Applications of Geographic Information Systems (GISCA).[4] For example, Jenks' (natural breaks) classification is used for all sociodemographic thematic mapping at GISCA. Overall, we identified only four CHF programs which were located in regional areas. Other rural programs were excluded as they did not meet our prespecified definition of a CHF program.

In summary, we acknowledge the need for better data to describe the burden of CHF throughout Australia. We also explicitly acknowledge the need for a less "metro-centric" approach to CHF service: perhaps by using remote monitoring techniques.[5]

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Compulsory helmets for school-age skiers and snowboarders

Graham M Slaney, Judith Finn, Angus Cook and Philip Weinstein

To the Editor: With the ski season in Australia drawing to a close for another year, it is a good time to reflect on the injury prevention benefits of wearing helmets when skiing or snowboarding.

Skiing falls can be fatal. Two people have died from head injuries on Australian skifields in recent years: a skier died after colliding with a tree branch on an intermediate run at Mt Buller, Victoria, in 2003; and in 2006, a novice snowboarder died after falls sustained while snowboarding at Thredbo, New South Wales.[1] Neither person was wearing a helmet. In Australia in 2002–03, 3.5% of skiing-related hospital admissions and 6.2% of snowboarding-related admissions were due to intracranial injuries.[2]

During the 2004 and 2005 ski seasons we collected data on the use of helmets in snowboarders presenting to the Mt Buller Medical Centre. Of 494 snowboarders, 17.6% had been wearing helmets, and none had sustained a head injury. Of the nine patients with head injuries, none had been wearing helmets. These figures are similar to those reported in overseas studies, which have shown that wearing a helmet can reduce the snow-sport head injury rate by up to 60%.[3,4]

The use of helmets for snow sports makes intuitive and biological sense, as it does for cyclists, but, unfortunately, Australia is yet to issue a snow-sport helmet performance standard, as it does for bicycle helmets. Helmet use should be strongly recommended for all snowboarders and skiers. In particular, helmets should be made compulsory for children, who are more susceptible to head injury[4] and who are often present at ski resorts in large organised school groups that could readily be made to comply. At present in Australia, helmet use is not compulsory for children attending skiing or snowboarding lessons, as it is in North America. This is in spite of the fact that helmet use is compulsory in Australia for school skiing and snowboarding competition events.

Some skiers and snowboarders are gradually getting the message about helmets, and a recent informal survey at Mt Buller (Buller Ski Lifts personnel, personal communication) estimated the rate of helmet use to be 20% among adults and 68% among children — but this still leaves over 30% of children vulnerable.

Snow-sports helmets now come in many colours, shapes and sizes, and are increasingly acceptable to young people. A helmet is probably the cheapest individual item of clothing for a ski holiday. And it may save your life.

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Probiotic treatment of vancomycin-resistant enterococci: a randomised controlled trial

H Reginald Magee

TO THE EDITOR: It was interesting to read of the trial conducted by Manley et al[1] using yoghurt containing *Lactobacillus rhamnosus* to clear vancomycin-resistant enterococci.

I would like to add an historical note.

The use of yoghurt in restoring bowel flora was practised by Dr J H Kellogg (of Corn Flakes fame) around the end of the 19th century. Kellogg was the chief physician at the Battle Creek Sanitarium in Michigan and was an advocate of high colonic irrigation, for he believed the colon was a sewer of toxic materials that were the causal factor in many diseases. Following this procedure, the patient was given a pint of yoghurt — half to be taken orally, and the remainder given by enema.[2,3]

By these measures, Kellogg claimed to have cured many conditions, from cancer of the stomach to psychiatric problems.

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