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A2 milk is allergenic

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TO THE EDITOR: Recent media reports have claimed numerous health benefits for A2 milk^{1,2} (eg, “new wave milk”, “wonder milk”). It is becoming more widely available, particularly in health food shops, and is advertised on Queensland television. We believe it is important to offer clear information about this product and cows’ milk allergy.

A2 milk is produced by cows homozygous for the A2 polymorphic variant (his→pro) at amino acid 67 of the β -casein gene. A difference in degradation patterns of the A1 and A2 variants is purported to lead to differences in immunological or pharmacological effects,³⁻⁵ which we will not comment on here. Regarding cow’s milk allergy, β -casein is one of at least seven proteins in cows’ milk with allergenic significance (α -, β - and κ -casein, α - and β -lactoglobulin,

lactoferrin and transferrin). One would not expect a single amino-acid difference in one protein to have a significant effect on milk allergenicity.

We have found in discussion with parents of milk-allergic children, as well as from inquiries from the community to AllergySA, that there is a perception that A2 milk may be less allergenic than “normal” milk (which contains A1 and A2 β -casein). Although most proponents of A2 milk have made no explicit claims about allergenicity — and indeed some have cautioned against the use of A2 in milk-allergic individuals — there have been media reports that may have led to this perception.⁶ However, these reports are misleading. For example, it is quite likely that children with a previous history of cow’s milk allergy who have been found to tolerate A2 milk have in fact “grown out” of the allergy, which is the usual natural history. Others may never have had true milk allergy.

We obtained a sample of pure A2 milk from A2 Dairy Marketers (Acacia Ridge, QLD) and used it for skin-prick testing of 11 consecutive milk-allergic children (Box). The tests compared A2 milk with “normal” (A1/A2) milk and cow’s milk protein extract. The mean diameter of the wheal raised by normal milk was not significantly different to that raised by A2 milk (8.2 mm for normal milk v 10.7 mm for A2 milk; $P=0.09$, paired t test). No patient had a negative reaction to A2 milk when the reaction to normal milk was positive.

We did not perform an oral challenge with A2 milk in these children, as many had experienced severe allergic reactions, and the predictive value of a positive skin-prick test in the presence of a clear recent history of clinical allergy is high.

We therefore caution that A2 milk should not be used by those with IgE-mediated cow’s milk allergy, particularly those who have had recent severe reactions to milk.

1 Today Tonight [television broadcast]. Channel 7. Episodes broadcast on 31 Mar 2003, 1 Apr 2003, 21 Jul 2003, 15 Sep 2003, 9 Feb 2004. Transcripts available at: <http://seven.com.au/todaytonight> (search for “A2”) (accessed Jul 2004).

2 Autism, milk link research hidden. *The Australian* 2002; Nov 13: 1.

3 McLachlan CN. beta-casein A1, ischaemic heart disease mortality, and other illnesses. *Med Hypotheses* 2001; 56: 262–272.

4 Laugesen M, Elliott R. Ischaemic heart disease, Type 1 diabetes, and cow milk A1 beta-casein. *N Z Med J* 2003; 116: U295.

5 A2 Corporation. About A2 milk. Available at: www.a2corporation.com (accessed Aug 2004).

6 Collins S. Milking the health advantages of A2. *The New Zealand Herald* 2003; 7 Apr. Available at: www.nzherald.co.nz/storydisplay.cfm?storyID=3351045 (accessed Aug 2004). □

Prescribing of amino acid infant formula

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TO THE EDITOR: There appear to be regional differences in the prescribing of amino acid infant formula in Australia. This is possibly due to differing practices in use of this formula as a first-line treatment for cow’s milk allergy or as a strategy for preventing allergy. This has financial implications, as the cost to the PBS of amino acid formula is \$371 per pre-prescription, compared with \$106 for hydrolysed protein formula.¹

In infants at high risk of allergic disease who are unable to be completely breastfed, there is evidence that prolonged feeding with a formula based on hydrolysed cow’s milk protein rather than conventional cow’s milk formula reduces infant and childhood allergy.^{2,3} There is no clear evidence that amino acid formula should be substituted for extensively hydrolysed protein formula as a primary preventive strategy.³ The current PBS indication for hydrolysed protein formula is treatment of intolerance to both cow’s milk and soy protein, but not primary allergy prevention. Similarly, current PBS guidelines restrict the use of amino acid formulas to proven intolerance to cow’s milk, soy protein and protein hydrolysate. Among children who are allergic to cow’s milk, 10% or less are also sensitive to protein hydrolysate formula.⁴ Thus, if current guidelines were followed, one might expect nine times the use of hydrolysed protein formula compared with amino acid formula.

I obtained statistics on PBS items supplied for the period January 2003 to January 2004 from the Health Insurance Commission (www.hic.gov.au/statistics/dyn_pbs/forms/pbs_tab1.shtml) for hydrolysed protein formula (item numbers 2676W and 8259Q) and synthetic amino acid formula (item numbers 3066J, 8443J, 8574G and 8575H). These showed that 8374 hydrolysed protein formula items were supplied, half the number of amino acid formula items (16 886).

Numbers of amino acid formula items supplied per 1000 children aged 4 years and younger were calculated using population statistics from the Australian Bureau of Statistics census figures 2001. These are compared in the Box with numbers of paediatric physicians per 1000 children (obtained from the Royal Australasian College of Physicians 2004) and paediatric allergists (derived from the Australasian Society of

Mean wheal diameter* (mm) on skin-prick testing

Patient	Normal milk [†]	A2 milk [†]	Cow's milk extract [‡]	Histamine positive control
1	12	10	8	4.5
2	11.5	12	11	5.5
3	4	8	6	15
4	8	11	10.5	3
5	12	8	6	9
6	3	5	2	9
7	7	15	7	10
8	7	7.5	5	7.5
9	6	7.5	4	3.5
10	13	25	4.5	3
11	7	9	3	5
Mean	8.2	10.7	6.1	6.8

* As wheals produced are not necessarily circular, it is standard to report diameter as the mean of two measurements taken perpendicular to each other. Results for all negative controls were 0 mm.

[†] Normal and A2 milk were stored frozen, and aliquots thawed for testing. They do not produce wheal reactions in non-allergic individuals.

[‡] Cows’ milk extract is manufactured for skin-prick allergy testing by Hollister-Stier, Wash, USA, and purchased from Richard Thomson, Sydney, NSW.

Amino acid formula prescription rates, January 2003 to January 2004, compared with numbers of paediatric physicians and allergists per 1000 children aged 4 years or younger

	Amino acid formula items per 1000 children	Paediatric physicians per 1000 children	Paediatric allergists per 1000 children
Australian Capital Territory	22.3	0.79	0
New South Wales	18.8	1.02	0.033
Victoria	17.8	1.00	0.030
Tasmania	12.3	0.53	0.033
South Australia	9.3	1.01	0.067
Northern Territory	9.1	0.92	0
Queensland	5.9	0.72	0.008
Western Australia	3.3	0.99	0.049

Clinical Immunology and Allergy membership handbook 2003).

Prescribing practice varied markedly between states and territories. The Australian Capital Territory, New South Wales and Victoria had six to seven times more amino acid formula items per 1000 children than Western Australia. This did not appear related to numbers of paediatricians or paediatric allergists, as Western Australia had a similar number of paediatricians and more paediatric allergists per 1000 children than NSW and Victoria.

The differences found were unlikely to be related to variation in numbers of adult immunology/allergy specialists, who are unlikely to treat many infants aged under 2 years. Nor were they likely to be due to differing prevalence of combined milk, soy and protein hydrolysate intolerance, as the prevalence of allergic disease does not differ markedly between Australian states. For example, the prevalence of atopic eczema at age 6 years in four cities (Adelaide, Melbourne, Sydney and Perth) was very similar, ranging from 10.1% to 11.4%.⁵ It seems unlikely that 80% of cases of combined intolerance are being missed in Western Australia. The estimated cost to the PBS for amino acid formula for 2003–2004 of \$7 107 627 was 10 times that of hydrolysed formula (\$757 570).

1 Australian Government Department of Health and Ageing. Schedule of pharmaceutical benefits for approved pharmacists and medical practitioners. Effective from 1 August 2004. Available at: www1.health.gov.au/pbs/ (accessed Oct 2004).

2 Osborn D, Sinn J. Formulas containing hydrolysed protein for prevention of allergy and food intolerance in infants. *Cochrane Database Syst Rev* 2003; 4: CD003664.

3 Host A, Halken S. Hypoallergenic formulas — when, to whom and how long: after more than 15 years we know the right indication! *Allergy* 2004; 59 Suppl 78: 45-52.

4 Giampietro PG, Kjellman NI, Oldaeus G, et al. Hypoallergenicity of an extensively hydrolyzed whey formula. *Pediatr Allergy Immunol* 2001; 12: 83-86.

5 Williams H, Robertson C, Stewart A, et al. World-wide variations in the prevalence of symptoms of atopic eczema in the International Study of Asthma and Allergies in Childhood. *J Allergy Clin Immunol* 1999; 103: 125-138

Rectal perforation from colonic irrigation administered by alternative practitioners

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TO THE EDITOR: Colonic irrigation is the introduction of a large volume of fluid into the colon via the rectum. This volume may be up to 50 litres, run in and out by means of a rectal tube, in an effort to empty the bowel. This treatment is often administered by a practitioner of complementary or alternative medicine, without medical advice. The fluid may be driven by gravitational or mechanical force.¹ Recognised risks from

colonic irrigation are electrolyte imbalance, bowel perforation and communicable diseases such as amoebiasis.²

Colonic irrigation is different from a standard enema given to relieve constipation or to treat a primary bowel disease. An enema involves a small amount of fluid and is usually authorised by a medical practitioner and administered by a trained nurse, attendant or is self-administered. Perforation of the rectum has rarely been reported.³

We document three cases of perforation of the rectum from colonic irrigation, treated by different surgeons at different institutions (Box). All have required surgical intervention. Each patient underwent colonic irrigation to relieve chronic constipation, to “cleanse” or “clear out stale faeces”. None had primary colonic or rectal pathology. None of the three patients were warned about the complication of perforation. Importantly, one patient initially denied the use of colonic irrigation, even with direct enquiry (Case 1), presumably because of embarrassment. This has the potential to delay the diagnosis or lead to inappropriate treatment.

Perforation may occur in the rectum by direct injury from the irrigation device (Case 1), or after the irrigation has commenced (Cases 2 and 3), and may be caused by the generation of a high pressure within the lumen of the bowel.

Rectal perforation from colonic irrigation may be diagnosed from the history, plain abdominal x-rays or a computed tomography scan with or without meglumine diatrizoate enema. A high degree of suspicion by the attending physician will prompt the diagnosis. Intensive medical therapy with appropriate antibiotics and surgery is necessary. Plain abdominal x-ray did not show an abnormality at 12 hours in the one case where x-ray was taken.

We feel that colonic irrigation is of dubious benefit, especially when delivered to remove so-called “toxic waste” when bowel

Correspondents

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There should be no more than 5 references. The reference list should not include anything that has not been published or accepted for publication. Reference details must be complete, including: names and initials for up to 4 authors, or 3 authors et al if there are more than 4 (see [mja.com.au/public/information/uniform.html#refs](http://www.mja.com.au/public/information/uniform.html#refs) for how to cite references other than journal articles).

LETTERS

function is satisfactory. There is potential for serious harm. The apparent failure of the operators to warn patients about a risk of any serious complication, the failure to diagnose the possible perforation at the time of injury, and the failure to provide any subsequent follow-up, which might have led to an earlier diagnosis of any complication, probably indicates subopti-

mal practice. Cases 2 and 3 occurred at the same clinic within a few weeks of each other, suggesting a possible systems failure of the irrigation device.

Primary healthcare practitioners need to be aware of the dangers of this treatment. Colonic irrigation should be urgently and formally assessed from an evidence-based, risk-benefit perspective.

- 1 Colonic irrigation and the theory of autointoxication: a triumph of ignorance over science [editorial]. *J Clin Gastroenterol* 1997; 24: 196-198.
- 2 National Health and Medical Research Council Medicine Advisory Committee. Colonic irrigation. Report of the Session (NHMRC) 1982 October Canberra. Canberra: NHMRC, 1982. (Indexed in *Australian Medical Index* Jan 2004.)
- 3 Parun H, Butnarug G, Neufeld D, et al. Enema induced perforation of the rectum in chronically constipated patients. *Dis Colon Rectum* 1999; 42: 1609-1612. □

Case descriptions for three women who had rectal perforation after undergoing colonic irrigation

Case	Age (years)	Timing of symptoms	Clinical features	Investigations	Management
1	59	Pain immediately on insertion of enema tube. No irrigation. Attended emergency department 24 hours after the tube insertion.	Lower abdominal and deep pelvic pain. Sepsis.	Abdominal computed tomography scan showing perirectal oedema and extrarectal gas.	Intravenous antibiotics and transrectal drainage of perirectal abscess.
2	51	Pain started during irrigation. Attended emergency department 4 days after irrigation.	Lower abdominal pain. Sepsis.	Abdominal computed tomography scan showing gas and fluid in the perirectal fat and retroperitoneum.	Intravenous antibiotics and initial transrectal drainage of perirectal abscess. Recurrent abscess formation required laparotomy and rectal resection with stoma formation.
3	56	Pain started during irrigation. Attended emergency department the same day, but was discharged. Re-presented 7 days later.	Lower abdominal and deep pelvic pain. Constipation and urine retention leading to urinary infection. Sepsis.	Abdominal computed tomography scan showing pelvic abscess posterior to the rectum.	Emergency laparotomy, sigmoid loop colostomy and drainage of abscess. Residual abscess drained transrectally 2 weeks after initial surgery.

Critical shortage of injectable thiamine in Australia

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TO THE EDITOR: There is no substitute for injectable thiamine in the treatment and prevention of Wernicke's encephalopathy, for which the oral form of thiamine is considered inadequate.¹ If the condition is not treated promptly with parenteral thiamine, permanent brain damage can occur.

A shortage of injectable thiamine noted in a South Australian hospital led us to enquire into the extent of the problem in Australia. In the first week of July 2004, we undertook an Australia-wide survey of major teaching hospital pharmacies. Sixteen hospitals were contacted by phone, and 15 chief hospital pharmacists provided information about thiamine stock, normal thiamine usage over a 6-month period, shortages of other drugs,

and reasons for shortages. Data on thiamine are shown in the Box.

Most hospitals (11/15) were unable to provide injectable thiamine for periods ranging from a few weeks to 5 months. Rationing reduced the use of injectable thiamine in 13/15 hospitals. There was a total shortfall of 2000 ampoules per month for the 13 hospitals. Given an average of six ampoules used per admission, we estimate that 330 patients a month were untreated or inadequately treated.

Half the hospitals surveyed obtained some ampoules either directly from suppliers or through the Special Access Scheme (SAS) protocol of the Therapeutic Goods Administration (TGA). This protocol is time-consuming and cumbersome, while the non-SAS system is expensive (10 times the usual price per ampoule). Pharmacists reported having many other drugs (40–60) on back order.

The pharmacists stated that drug shortages were caused by scarcity of raw materials and TGA restrictions. However, the current shortage of thiamine in Australia was foreseeable in 2003, when the main

Stocks and usage of injectable thiamine in 15 Australian hospitals, as at 3 July 2004*

Hospital	Number of vials		Use/month	
	Lowest	Current	Previous 2 months	Usual
1	0	0	0	16
2	0	0	0	50
3	0	0	0	50
4	0	0	0	65
5	0	12	0	20
6	0	10	0	35
7	0	25	0	20
8	0	200	0	130
9	0	120	0	1200
10	0	25	25	70
11	0	10	10	150
12	1	35	40	120
13	5	160	17	180
14	25	86	100	100
15	30	90	30	30

*The table compares the level of stock at its lowest during the shortage with the level at July 2004, along with estimates of use at July 2004 and before the shortage.

manufacturer stopped thiamine production. The TGA did not alert pharmacists or doctors to the potential shortage in writing, nor provide comprehensive help to prevent or alleviate the shortages.

The public health response to shortages of essential medicines should include surveillance and a systematic analysis of the causes. Better communication between pharmacists, clinicians and government authorities, and the formation of contingency plans and guidelines, are needed. It was only through informal networking and the quick thinking of hospital pharmacists that a crisis was averted in Australia.

It is unconscionable that an inexpensive essential medicine is not available to those Australians who may need it. In this respect, our public health system has failed. Because injectable thiamine has been unavailable or rationed, an increase in the incidence of alcohol-related brain damage may have occurred. Australian health ministers should act immediately to prevent critical shortages of essential medication, which could be tragic and costly.

1 Thomson A, Cook C, Touquet R, Henry J. The Royal College of Physicians report on alcohol: guidelines for managing Wernicke's encephalopathy in the accident and emergency department. *Alcohol Alcohol* 2002; 37: 513-521. □

Pertussis vaccination for new parents?

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TO THE EDITOR: Pertussis (whooping cough) is a readily transmissible respiratory infection that may cause severe respiratory illness. The burden of severe pertussis affects infants, often resulting in hospitalisation (especially those aged under 6 months) and death (1 in every 200 patients aged under 6 months).^{1,2}

In Australia, there were nine deaths from pertussis between 1993 and 1997, predominantly in young infants, and a further five young infant deaths during the 2001–2002 epidemic.^{3,4} Epidemics occur every 3 to 4 years.² Pertussis cases and hospitalisations

in children aged under 6 months continue to occur in south-east Queensland, with 19 notifications since January 2003.

There has been a shift in the epidemiology of pertussis in Australia and the United States, from a disease of young children to a disease of adolescents and adults of child-bearing age.^{1,5} In Australia, there has been a preponderance of pertussis notifications in adult females.⁵

Pertussis vaccine is already provided free to children at ages 2, 4 and 6 months, 4 years and 15 years, as part of the National Immunisation Program.² However, young infants remain incompletely protected by vaccination, as the third, completion dose of the primary course of pertussis vaccination is not given until 6 months of age. A national study of hospitalised infant pertussis cases in 2001 indicated that parents were the presumptive source of pertussis infection for their children in more than 50% of cases.⁶ This has led the National Health and Medical Research Council to recommend that both parents should receive a (once-only) adult booster dose of pertussis vaccine, either when planning pregnancy or as soon as possible after delivery of an infant.² The cost of the vaccine is about \$30.

As yet there is no suggestion that funding will be made available to provide this vaccine to all new parents as part of the National Immunisation Program. However, the amount is not a high price to pay for the protection of a new baby and its parents, particularly now that new parents will receive additional financial support from the federal government. The potential exists to promote opportunistic maternity-ward-based administration of this vaccine to postpartum mothers and their partners. We encourage all medical practitioners, especially obstetricians and paediatricians, to discuss this important issue with parents.

1 Guris D, Strebel PM, Bardenheier B, et al. Changing epidemiology of pertussis in the United States: increasing reported incidence among adolescents and adults, 1990-1996. *Clin Infect Dis* 1999; 28: 1230-1237.

2 National Health and Medical Research Council. The Australian immunisation handbook. 8th ed. Canberra: Commonwealth of Australia, 2003.

3 McIntyre P, Amin J, Gidding H, et al. Vaccine preventable diseases and vaccination coverage in Australia, 1993-1998. *Commun Dis Intell* 2000 Suppl: 24.

4 Australian Institute of Health and Welfare. The AIHW national mortality database. Canberra: Australian Government, 2004.

5 Communicable Diseases Surveillance Highlights. Vaccine preventable diseases. *Commun Dis Intell* 2000; 24: 11.

6 Elliot E, McIntyre P, Ridley G, et al. National study of infants hospitalized with pertussis in the acellular vaccine era. *Pediatr Infect Dis J* 2004; 23: 246-252. □

To exercise or not to exercise in chronic fatigue syndrome?

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TO THE EDITOR: A recent editorial¹ and article² continue to promulgate and link the unproven concepts that patients with chronic fatigue syndrome (CFS) are “deconditioned” and exercise is beneficial in treatment. The cited study by Fulcher and White³ is open to opposite conclusions, depending on their use of the outcome descriptor “better”. If the term is restricted to “much better” and “very much better”, then, as cited by Lloyd,¹ 16 of 29 people with CFS rated themselves as “better” after a graded exercise program, compared with only 8 of 30 in the control group who completed a flexibility treatment regimen. However, if the “better” descriptor combines “a little better”, “much better” and “very much better”, which is the interpretation used by Wallman et al,² then the scores for the exercise versus flexibility groups are not different, being 27 of 29 and 26 of 30, respectively, agreeing with the conclusion of Wallman et al.²

Whichever interpretation is applied, any beneficial effect of the graded exercise program in people with CFS in these studies must be independent of any training effect or change in level of “conditioning”, as this was reported in one study,² but not in the other.³

A fundamental flaw with most exercise studies in CFS is the use of submaximal or symptom-limited tests, which provide notoriously misleading data when compared with maximal exercise testing procedures.^{4,5} Wallman et al² correctly identify maximal oxygen consumption as the “gold standard” measure of exercise capacity, yet such measurements were not made in the three articles they cited. When such procedures are applied, the exercise capacity of people with CFS is not significantly different from either measured or age-predicted values for healthy sedentary people.⁶ Wallman et al² suggested that maximal testing procedures could favour the recruitment of “more robust or healthier” patients and provide misleading information. In the first place this is denied by the study of Sargent et al,⁶ in which the illness status reported by patients who completed the maximal tests was similar to that in previous CFS studies. In the second place, the maximal test proto-

col chosen for a given population should be designed to exclude any influence of fatigue on the metabolic measurements. This is confirmed by the results from the study cited,⁶ in which the metabolic measurements met the published criteria of a maximal test.^{4,5}

In summary, patients with CFS are not “deconditioned”. Neither their muscle strength nor their exercise capacity is different from that of other sedentary members of the community (>70%). We remain unaware of any incontrovertible evidence that the various “exercise training” programs suggested in previous articles improve either the physiological or clinical status of people with CFS.

- 1 Lloyd AR. To exercise or not to exercise in chronic fatigue syndrome? No longer a question [editorial]. *Med J Aust* 2004; 180: 437-438.
- 2 Wallman KE, Morton AR, Goodman C, et al. Randomised controlled trial of graded exercise in chronic fatigue syndrome. *Med J Aust* 2004; 180: 444-448.
- 3 Fulcher KY, White PD. Randomised controlled trial of graded exercise in patients with the chronic fatigue syndrome. *BMJ* 1997; 314: 1647-1652.
- 4 Sargent C, Scroop GC. Defining exercise capacity, exercise performance and a sedentary lifestyle. *Med Sci Sports Exerc* 2002; 34: 1692-1693.
- 5 Sargent C, Scroop GC. VO_{2peak} versus VO_{2max} ? An important distinction. *Med Sci Sports Exerc* 2002; 34: 1215-1216.
- 6 Sargent C, Scroop GC, Nemeth PM, et al. Maximal oxygen uptake and lactate metabolism are normal in chronic fatigue syndrome. *Med Sci Sports Exerc* 2002; 34: 51-56. □

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TO THE EDITOR: The claim in Lloyd's editorial¹ that “the criteria for diagnosis are well accepted internationally” ignores the recent publication of the Canadian consensus guidelines for the diagnosis and management of myalgic encephalomyelitis/chronic fatigue syndrome,² which were sponsored by Health Canada and written by an international group of well published researchers. The Canadian definition of chronic fatigue syndrome (CFS) requires the concurrent presence for six months of fatigue, post-exertional fatigue, sleep dysfunction, pain (including headaches) and neurological/cognitive manifestations, as well as at least one symptom from two of autonomic, neuroendocrine and immune manifestation categories (pp 12–13). These requirements add clinical specificity to the Fukuda criteria and exclude subjects who

may have chronic fatigue for other reasons, such as psychiatric disorder without multiple physical symptoms.

Lloyd refers to the “recent refinements to improve reliability” in the revision of the research case definition by Reeves et al.³ The SPHERE screening instrument recommended by that article was designed for psychiatric screening in primary care. It arbitrarily classifies people with multiple physical symptoms, often severe in degree and associated with major disability, as having somatisation disorder. This is akin to subclassifying people with severe multiple sclerosis as having somatoform disorder and those with fewer and less severe symptoms as the “core” multiple sclerosis group, a finding which is not supported by the evidence.

Conclusions from the article by Wallman et al⁴ cannot be generalised to the severely ill. Recruitment was from “notices placed in medical surgeries and by advertisements in local newspapers”. Patients with severe CFS, who can barely venture outside their homes and are often too ill to read, would be unlikely to participate. Loblay, Chair of the Royal Australasian College of Physicians Working Group for CFS Clinical Practice Guidelines, urges caution about generalising from exercise studies, which never include people with severe CFS: “All these studies involve people willing and able to participate. The people who find it makes them feel lousy drop out.”⁵

Lloyd asserts exercise is no longer a question (“... graded physical exercise should become a cornerstone of the management approach for patients with CFS”). To promote such a strong, unqualified message to busy general practitioners who may be unfamiliar with the range of severity in CFS risks serious harm to patients.

- 1 Lloyd AR. To exercise or not to exercise in chronic fatigue syndrome? No longer a question [editorial]. *Med J Aust* 2004; 180: 437-438.
- 2 Carruthers BM, Jain AK, De Meirleir K, et al. Myalgic encephalomyelitis/chronic fatigue syndrome: clinical working case definition, diagnostic and treatment protocols. *J Chronic Fatigue Syndr* 2003; 11: 7-116. Available at: www.mefmaction.net/documents/journal.pdf (accessed Sep 2004).
- 3 Reeves WC, Lloyd A, Vernon SD, for the International Chronic Fatigue Syndrome Study Group. Identification of the ambiguities in the 1994 chronic fatigue syndrome research case definition and recommendations for resolution. *BMC Health Serv Res* 2003; 3: 25.
- 4 Wallman KE, Morton AR, Goodman C, et al. Randomised controlled trial of graded exercise in chronic fatigue syndrome. *Med J Aust* 2004; 180: 444-448.
- 5 Maegraith D. Pros and cons of exercise in fighting CFS. *The Weekend Australian* 2004; Jul 3-4: C32. □

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IN REPLY: Scroop and Burnet correctly identify the vagaries of the necessarily subjective measurement of outcomes in intervention studies of chronic fatigue syndrome (CFS). Given that muscle strength, endurance and recovery are essentially normal in patients with CFS,¹ rather than become too focused on the best approach to measurement of exercise capacity the key issue is whether patients benefit in terms of self-reported symptom severity or functional status.

The weight of evidence indicates that graded physical exercise does provide such benefits. Whether this occurs via improvements in aerobic fitness or via the well-recognised psychological and social benefits of exercise is something of a side-issue.

Stein and Hunter draw attention to the recently published Canadian consensus guidelines for the diagnosis and management of myalgic encephalomyelitis/CFS. Although this document may provide a welcome recognition for Canadian patients with the disorder, unlike the Australian guidelines,² it is devoid of an evidence base for the recommendations. Sadly, rather than “add[ing] clinical specificity”, it is also highly likely that the modified diagnostic criteria fall into the trap of preferentially identifying patients with somatisation disorder,³ as such individuals often report large numbers of unexplained symptoms, and hence the addition of 20 or more symptoms to the diagnostic criteria may well bias towards inclusion of such patients.

Stein and Hunter are incorrect in the assertion that SPHERE was designed for psychiatric screening in primary care, as the instrument arose out of our studies in CFS specifically seeking to identify clinically significant fatigue states.⁴

I support the recommendation about caution in generalising from existing published data regarding graded exercise to patients who are severely ill, as such patients are indeed likely to be under-represented in published studies. Nevertheless, it is noteworthy that the recommendations made in the Canadian document cited by Stein and Hunter also clearly support the notion of graded physical exercise: “Patients should gently and gradually increase their level of activity.” Thus, rather than leave the severely

affected to continue to “barely venture outside their homes”, I would recommend a carefully designed graded exercise program in the home, with a goal of improving functional performance sufficiently to escape those confines.

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Institutional racism in Australian healthcare: a plea for decency

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TO THE EDITOR: While the article by Henry and colleagues provides food for thought and possible action,¹ do they exhibit the fairness they exhort to solve the problem they perceive?

There appears to be a distinct lack of logic in some of their deductions in the Box on page 517. “Body part funding” is not confined to Aboriginal health. For the 43 years I was associated with NSW Health, it was an integral part of the system and, together with its variations, increased as the years passed.

The authors claim that as only \$80 per head being spent on medical and pharmaceutical benefits in a remote Aboriginal community compared with the \$900 spent in Double Bay is an example of racism. Surely, it is only a reflection of the lack of both a pharmacy and doctor in the remote community compared with the easy access to both in the inner-Sydney suburb. Comparison between the remote Aboriginal community and an all-white community of similar characteristics would have more validity.

- 1 Henry BR, Houston S, Mooney GH. Institutional racism in Australian healthcare: a plea for decency. *Med J Aust* 2004; 180: 517-520. □

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TO THE EDITOR: In their challenging article, Henry and coauthors assert that the poor health of Australian Aboriginals is the result of the “divided, divisive, racist, socially unjust society” of “this Australia”.¹

I cannot agree. The health standards enjoyed by “white Australia” are not an isolated phenomenon, but rather a part of the fabric of an advanced technological society. Efforts to bring Australian Aboriginal health to the same standard without the Indigenous Australians being fully part of this 21st-century society will never be successful, even with limitless resources and endless goodwill.

It is possible to maintain cultural identity and remain cognizant of past hurts while playing a full, if not leading, role in this technological society.

If the Aboriginal elders were to lead their people into mainstream society they would find, I’m sure, an inclusive, tolerant, exciting and advancing society where they could play a full role, enjoy the same health as the rest of Australia, while still maintaining their unique identity.

- 1 Henry BR, Houston S, Mooney GH. Institutional racism in Australian healthcare: a plea for decency. *Med J Aust* 2004; 180: 517-520. □

Three Australian whistleblowing sagas: lessons for internal and external regulation

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TO THE EDITOR: We write in response to the article by Faunce and Bolsin on the lessons to be drawn from three Australian whistleblowing sagas.¹ Their summary of events at King Edward Memorial Hospital, Perth, deserves comment.

Michael Moodie, the Chief Executive Officer (CEO) of King Edward Memorial Hospital, was also CEO of Princess Margaret Hospital for Children (PMH). He was stood down from PMH because of the concerns of

workers in response to events at PMH unrelated to those at King Edward Memorial Hospital, as Faunce and Bolsin implied.

Moodie was the senior administrator charged by the government with ensuring that appropriate standards were in place and were being met. Staff at PMH believed he was unable to fulfil his brief, culminating in votes of no confidence from the PMH Clinical Staff Association, the PMH Medical Advisory Committee, and a petition signed by 80 PMH doctors.

- 1 Faunce TA, Bolsin SNC. Three Australian whistleblowing sagas: lessons for internal and external regulation. *Med J Aust* 2004; 181: 44-47. □

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IN REPLY: Our reference to Michael Moodie as a “whistleblower” merely reiterates his description as such in the report of the Inquiry into Obstetrics and Gynaecological Services at King Edward Memorial Hospital by the Australian Council for Safety and Quality in Health Care.¹

That report states: “Both the Bristol and King Edward case arose from ‘whistle-blowers’ reporting serious problems rather than from established safety and quality monitoring systems. In Bristol’s case, the whistleblower was an anaesthetist and, in King Edward’s case, it was the recently appointed Chief Executive. In both cases, either directly or indirectly, the department of health received information about management and clinical performance problems that had not been addressed over a significant period of time.”

The report then lists nine examples of problems established at both institutions, ranging from a “closed culture and environment unsupportive of openly disclosing errors and adverse events” to “poor clinical and emotional outcomes for patients and families”. The report continues: “However, there were differences in the Hospitals’ response to the inquiries. Bristol welcomed an inquiry and actively supported the process. In contrast, King Edward tolerated the process and the Western Australian branch of the Australian Medical Association actively and publicly fought it.”

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Ethical and legal issues at the interface of complementary and conventional medicine

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TO THE EDITOR: The complementary and alternative medicine (CAM) series raised awareness and provided balanced and thoughtful debate. The article by Kerridge and McPhee in that series¹ is no exception, but we would like to question their conclusion that “not only is it unclear whether a true integration of conventional and unconventional medicines is possible, but, more importantly, whether it is even desirable”. For a variety of reasons we believe that it is both possible and desirable.

There are increasing examples of situations in which medical practitioners can integrate ethical, evidence-based CAM into practice. Apart from the well-known and validated examples, such as *Hypericum perforatum* (St John's wort) for depression, ginger for nausea in pregnancy, and *Ginkgo biloba* for intermittent claudication, there are other, less well known, but increasingly investigated, examples of CAM for common conditions. With quality information and a little training, these can be readily incorporated into medical practice.

To illustrate, Hippocrates was known to use the herb *Vitex agnus-castus* (chasteberry) for treating symptoms of premenstrual syndrome. Today we have a randomised controlled trial (RCT) to support its use.² There are RCTs to support the use of *Serenoa repens* (saw palmetto) for symptomatic relief of benign prostatic hypertrophy,³ and good evidence is accumulating for the use of glucosamine for osteoarthritis⁴ and mindfulness meditation for preventing relapse in recurrent depression.⁵

With systematic reviews on these CAMs doctors should be informed about them. However, the resources for promoting them are minimal compared with those used to promote pharmaceuticals. Considering side-effect profiles and patient autonomy, why shouldn't trained medical practitioners offer effective CAM remedies as first-line therapy instead of a pharmaceutical? To say these therapies should only belong to the realm of CAM practitioners would be to deprive the

medical practitioner and patient of a wider choice of treatments.

Communication, holism, balance and individualised care are the hallmarks of quality general practice and do not just belong to CAM therapists. If orthodox medical practice is to remain current, evidence-based and relevant, general practitioners have no option but to integrate safe, validated and ethical forms of CAM into their practice. If they are not adequately trained in the relevant discipline they may wish to refer to an appropriately qualified CAM practitioner, although statistics indicate that GPs prefer to refer to GPs already trained in CAM.⁶

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TO THE EDITOR: Although Kerridge and McPhee stress the need to find an evidence base (if there is any) for CAM, they nevertheless claim “medical practitioners and students no longer have any choice but to gain some knowledge about CAM and the interface between conventional and complementary medicine.”¹

I suppose that archaeologists, geologists, palaeontologists and biologists now need to gain some knowledge about the interface between Darwinism and Creation Science. And our astronomers need some knowledge about the interface between astronomy and astrology.

Science, including effective medical care, is not advanced by pandering to unscientific consumerism about unproven theories, especially if it manages to get the law on its side. Galileo was persecuted for “his heretical

view” that the earth revolved around the sun. Have we learnt nothing from his experience?

Competing interests: Member, Australian Skeptics.

1 Kerridge IH, McPhee JR. Ethical and legal issues at the interface of complementary and conventional medicine. *Med J Aust* 2004; 181: 164-166. □

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IN REPLY: We agree with Kotsirilos and Hassed that there are many examples of successful integration of “proven” CAM into conventional medical practice. Our question, however, is whether it is possible to integrate CAM where its theoretical maxims and practices are incommensurate with allopathic medicine (eg, homoeopathy) and whether “integrative medicine” will ultimately fragment and diminish CAM, further isolate “non-evidence-based” CAM practitioners and make less visible those views of health and disease that are not consistent with modern medicine.¹

It is misleading for Arnold to imply that there may be no evidence base for complementary and alternative medicines (CAMs). We suggest that medical practitioners should ask themselves not whether an “evidence base” exists, but what the existing evidence shows. The picture that emerges from a review of the literature is one of variable clinical efficacy. Thus, there is no evidence to support the use of chiropractic for childhood asthma,² but there is good evidence that phytomedicines may reduce crises in sickle-cell disease,³ that cranberry juice may reduce the frequency of symptomatic urinary tract infections in women,⁴ and that horse chestnut seed extract is an efficacious treatment for chronic venous insufficiency.⁵ There is also clinically important evidence about harmful interactions, for example that St John's Wort, garlic and ginseng may lower blood levels of warfarin.⁶

Medical practitioners should be critical and sceptical of all untested claims of therapeutic benefit. We suggest they acquaint themselves with evidence about risks and benefits of CAMs, particularly in their own area of practice. This is not pandering to anything. It is evidence-based practice. By the same token, use of CAM may reflect evidence-based decision-making by doctors and patients. It is simply divisive to dismiss it as “unscientific consumerism about unproven theories”, and

it is foolish in any case to dismiss the latter. Medicine and science must compete with non-scientific perspectives in the public sphere, for the contest of ideas is never over in human history.

Ideological positions are black and white. Science prefers shades of grey. We have indeed learnt much from Galileo's experience.

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Timing of health assessments

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TO THE EDITOR: I read with interest the article by Byles and colleagues that shows the minimal impact of health assessments in a section of the older Australian community.¹ While these assessments may not be identical to the assessments covered by Enhanced Primary Care (EPC) items on the Medicare Benefits Schedule, my experience performing the latter in older people leads me to believe that they also have limited impact.

I am now in part-time clinical practice, with a reasonably well-defined practice population, comprising mostly older patients with complex problems. My practice philosophy is closer to the (perhaps old-fashioned) notion of continuing, comprehensive care, which means I have not been afraid to

spend the time needed to understand those patients and to document their health information. So far, I am not sure I have learned anything new in any of the EPC health assessments in which I have participated, although they have been useful for initial assessments of newer patients, as at least they remunerate practices better for the time-consuming task of doing this well.

However, EPC assessments may be performed every 12 months. Is this really necessary, unless patient circumstances change? In my practice the answer is probably no, although they may be more useful in practices with less stable doctor-patient relationships. Would it not be a more effective use of resources to instead allow for better-funded initial assessments and assessments when a patient's condition changes, irrespective of the timing?

- 1 Byles JE, Tavener R, O'Connell RL, et al. Randomised controlled trial of health assessments for older Australian veterans and war widows. *Med J Aust* 2004; 181: 186-190. □

Should telemedicine in eye care be funded in Australia?

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TO THE EDITOR: Telemedicine in eye care (teleophthalmology) is one of the established technologies in medicine, providing the means for undertaking sophisticated eye care and for maintaining contact with patients in rural and remote areas.¹

Telemedicine in Australia has been primarily facilitated by government, against a background of complex funding arrangements and interwoven healthcare responsibilities (it is funded mostly by project grants and state government telehealth initiatives).² This funding mechanism impedes the efficient use and integration of telemedicine services.²

The current healthcare environment demands a detailed economic evaluation to justify continuous funding for teleophthalmology. However, some of the economic benefits of teleophthalmology may not be directly visible in the healthcare system itself. Significant benefit may be obtained by, for example, savings in time and travel expenses, thereby contributing to society indirectly. Furthermore, the cost-effectiveness of a telemedicine service improves considerably when it is integrated with existing routine healthcare services.³ But organisational and attitudinal barriers and lack of funding have delayed such integration.⁴ These barriers relate to human resource allocation issues in an already overstressed healthcare system and the mindset of some critics who view telemedicine as a peripheral activity and a “novelty” area for technological enthusiasts. The cost-effectiveness of telemedicine will not be improved unless the perception that it is an “add on” is changed.⁴

The question of whether teleophthalmology should be integrated into routine services, with Medicare reimbursement, can be judged by four criteria:⁵

- Is the technology sound? (ie, does it fulfil its purpose?)
- Is the program effective compared with existing care?
- Is the program cost-effective?
- Is the program practical? (ie, are there any significant problems associated with it?)

On the basis of our own comprehensive evaluation of teleophthalmology in Western Australia,⁶ we believe that all four questions can be answered affirmatively, and that teleophthalmology would be most efficiently provided if integrated into existing healthcare services. Its inclusion in the Medicare Benefits Schedule would benefit many patients in remote and rural areas in Australia.

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UK health inequalities: the class system is alive and well

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TO THE EDITOR: The Postcard from Heller, Weller and Jamrozik¹ may reflect a nostalgic and unrealistic view of how good things are back home. They suggest that, in New South Wales, the health chances of both advantaged and disadvantaged populations are improving, and, in relative terms, social inequalities in health may also be showing “some improvement”.

In fact, despite impressive overall declines in mortality, there remain important differences in health status between NSW populations. Figures for the mid-1990s show that life expectancy at birth for both Aboriginal males and females is markedly less (by 20 years and 18 years, respectively). Similarly, socioeconomic disadvantage shortens life expectancy for both rural men and women (by 14 and 10 years, respectively) and urban

men and women (by 10 and 7 years, respectively).²

The relative gap is also widening for some important health indices. For example, from 1980 to 2000, the percentage difference in premature death rates (<70 years of age) between high and low socioeconomic groups has increased from 30% to 52% for men and from 24% to 32% for women, and for potentially avoidable mortality from 34% to 63% for men and from 27% to 40% for women.³

How should one respond to such inequalities? Heller et al suggest universal rather than targeted programs, as they are based on sound population health principles.

To construct this as a simple choice is not helpful. Unless we recognise and address the barriers facing people in adverse social circumstances, universal programs may unintentionally widen health inequalities. For example, universal access to healthcare in the UK and Australia has not equally benefited those from the most disadvantaged circumstances compared with wealthier and better-educated populations.⁴

The Postcard authors suggest that Australia is saved from class divisions by the established “fair go” tradition, where shared values overcome structural inequalities in “socioeconomic status”. In fact, social class continues to be a powerful but complex and changing influence in Australia.⁵ It is important to acknowledge the evidence that structural inequalities are significant and worsening in Australia,⁶ and that the most

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disadvantaged experience continued social exclusion.⁷

We need to shift from a "trickle down" perspective that sees the greatest health gains accruing to the most advantaged — with a hope that these benefits will eventually be achieved by everyone — to a more explicit social justice perspective that ensures that resources for health are allocated in ways that produce fair outcomes. This may help address "socially entrenched self-denial of the chance for better health".

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Drugs, sport and the Olympics 2000-2004

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TO THE EDITOR: Pseudoephedrine is no longer a banned substance in sport.¹ It was originally banned to protect athletes from overuse and its dangers. Has it become harmless or are athletes more intelligent?

This highlights much of the confusion in drug testing. Athletes with diabetes are permitted to use insulin for therapy, but those with hypertension are not allowed to take β -blockers. Both drugs are popularly believed in athletic circles to improve performance. What is to stop an athlete with diabetes from taking extra insulin for performance enhancement? Why do we discriminate against those with hypertension?

There is a ban on oxygen-transport drugs and on physical environment enhancers such as hypobaric chambers. Both are

alleged to produce the same result, but only use of the drug can be tested. The penalty for the drug user is disqualification, but for the hypobaric enthusiast a rousing cheer for a drug-free effort. The crime is the same, so why vary the penalty?

There is never likely to be a level playing field under the present system, in which one reads of positive test results being swept under the table. How will drug testing eliminate the genetic inequalities between athletes? How will testing improve the availability of top-level coaches and training facilities to all? How can it eliminate the inequality in financial incentives, allowing some athletes to train for 6 hours daily while others have to work to enable them to train for even 2 hours daily? We have swimming costumes that decrease drag in the water,¹ resulting in faster times. These are not universally available, giving their owners an advantage. A level playing field will never exist in our present system. It is incongruous that in all this mess, only drugs are available to all.

The current frenzy to test blood has ethical problems which have not been addressed.² What is to happen to an athlete who develops an infection from a dirty needle? Who is responsible for the tester who has a needlestick injury from an HIV-positive athlete? It is worth remembering that this diagnosis will only be made 3 months after the Games, when everyone has dispersed.

The whole area needs to be reviewed by an outside body with no vested interest in the outcome.

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