LETTERS

How much cervical cancer is being prevented?

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TO THE EDITOR: It was estimated in 1989 that cervical screening in Australia was preventing only 46% of squamous malignancies, against a theoretical capacity of 90%.[1] This suboptimal achievement after almost 25 years of cervical screening led to a major reorganisation of the program.

The 1989 analysis has been repeated, using the most recent year (1998) for which incidence rates have been published (Box). The more recent figures suggest that cervical screening in Australia is now preventing 70% of squamous carcinoma of the cervix.

This remarkable improvement can probably be attributed to the improved participation by women in regular screening, improved standards within laboratories, and better follow-up of cytological abnormalities. While there is still scope for improvement, there is clear evidence of a substantially better gain from cervical screening. Continued efforts to increase the participation rate among women aged 60–69 years is appropriate given that the prevented proportion appears to be lower in this age range.

1. Australian Health Ministers' Advisory Council. Cervical Cancer Screening Evaluation Committee. Cervical cancer screening in Australia: Options for change. Australian Institute of Health: Prevention Program Evaluation Series No 2. Canberra: AGPS, 1991.
2. Australian Bureau of Statistics 1998. Estimated resident population by age and sex: Australian States and Territories, June 1997 to June 1998. Canberra: ABS, 1998. (Catalogue No. 3201.0.)
3. Australian Bureau of Statistics 2001. National Health Survey. Canberra: ABS, 2001. (Catalogue No. 4364.0.)
4. Day NE. The epidemiological basis for evaluating different screening policies. In: Hakama M, Miller AB, Day NE. Screening for cancer of the uterine cervix. Lyon: International Agency for Research on Cancer, 1996: 199-209.
5. Australian Institute of Health and Welfare (AIHW). Cervical screening in Australia 1997-1998. Canberra: AIHW, 2000.❑

Ethics and research participation

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TO THE EDITOR: The recent article by Scott and colleagues[1] described a retrospective analysis by postal questionnaire of the attitudes of family members to participation (about a year earlier) in a face-to-face interview about their child's diagnosis of Ewing's sarcoma. This was accompanied by an editorial exposing the complexities of research participation, including the potential risks of interviews as well as the role of altruism.[2]

Although results derived from the questionnaire have only recently been published (November 2002), the questionnaire was distributed in November 1997, before introduction of the *National statement on the ethical conduct of research involving humans*.[3] Some ethical uncertainties and questions of historical interest arise. First, what was the nature of the original consent obtained for the initial interviews? Presumably it involved written informed consent in which the risks of participation were clearly mentioned, *including* the possibility of distress associated with the interview. Did it mention a procedure for aborting or complaining about the interview? Second, did the patients (aged up to about 35 years) *and* their families give permission to be contacted again by the same research group? Third, for the follow-up on research participation, was consent implied simply by return of the questionnaire? Finally, how would the conduct of the initial interviews and the follow-up questionnaire differ in the light of recent developments in the ethics of research involving humans?

Importantly, the respondents (84% of those surveyed) indicated that participation in the original study had not "upset them".[1] While the attitude of non-responders is unknown, this would

Percentage of squamous carcinoma of the cervix prevented, by age group, Australia 1998

Age group	Number of women (estimated number with a cervix*) in Australia[2]	Expected rate (per 100 000 women) of squamous carcinoma without screening†	Expected number of squamous carcinomas‡	Estimated number of squamous carcinomas observed in 1998§	Percentage prevented
20–24	665 691 (665 025)	5	33.3	9.6	71.1%
25–29	733 145 (732 412)	15	109.9	34.8	68.3%
30–34	706 925 (687 838)	25	172.0	62.9	63.4%
35–39	748 913 (728 692)	45	327.9	74.7	77.2%
40–44	702 629 (608 477)	45	273.8	76.2	72.2%
45–49	649 539 (562 501)	45	253.1	81.4	67.8%
50–54	570 287 (410 607)	45	184.8	48.1	74.0%
55–59	431 183 (310 452)	45	139.7	40.7	70.9%
60–64	370 123 (251 314)	45	113.1	40.7	64.0%
65–69	348 707 (236 772)	45	106.6	44.4	58.3%
Total	5 927 142 (5 194 090)		1714	514	70.0%

*Calculated by multiplying the number of women in Australia by the estimated age-specific hysterectomy fractions.[3] †Methodology as for the study by the International Agency for Research on Cancer,[4] using incidence in Norway at a time when the rates would have been little affected by screening. ‡Calculated by multiplying the estimated number of women in Australia with a cervix by the expected rate of squamous carcinoma of the cervix in the absence of screening.[4] §Calculated by multiplying the number of cases of cervical cancer observed in 1998 by 0.74, which was the proportion of all cervical cancers that were squamous.[5]

seem to confirm that the original process had been sensitive and appropriate. This is supported by the finding that families whose child had died after the initial interview were more likely to respond to the questionnaire.

As a long-time member of a university human research ethics committee, I have often been required to evaluate research protocols that involve potentially threatening or distressing interviews. This has occurred more frequently since introduction of the *National statement*, as much qualitative research previously conducted under different jurisdictions (such as quality control or clinical audit) has been submitted for formal ethical review. The risk of harm to participants in qualitative research cannot be trivialised. Its impact can be minimised by wording the consent form to warn of possible adverse psychological reactions to interviews and questionnaires, using trained interviewers and providing counselling support if needed.

1. Scott DA, Valery PC, Boyle FM, Bain CJ. Does research into sensitive areas do harm? Experiences of research participation after a child's diagnosis with Ewing's sarcoma. *Med J Aust* 2002; 177: 507-510.
2. Braunack-Mayer A. The ethics of participating in research. *Med J Aust* 2002; 177: 471-472.
3. National Health and Medical Research Council. National statement on the ethical conduct of research involving humans. Canberra: NHMRC, 1999. ❑

A Quality Use of Medicines program for continuity of care in therapeutics from hospital to community

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TO THE EDITOR: The article by Mant et al[1] and the letters following its publication focus attention on a long-standing problem that, despite concerted efforts by governments, healthcare providers and other stakeholders to address it, remains a major obstacle to effective and appropriate continuity of pharmacotherapy following discharge from hospital.

There is little doubt that many of the difficulties arise as a result of poor communication between hospitals and general practitioners. This is further exacerbated by a lack of standard protocols for the preparation and dissemination of discharge summaries. Nowhere is this more evident than in the case of residents of aged-care facilities returning from hospital with radically changed medication regimens.

To establish appropriate communication channels, Mant et al mention hospital GP liaison officers.[1] New canvasses the potential problems and suggests sensible solutions, including the involvement of community pharmacists.[2]

However, every hospital has a ready-made resource that requires only a set of formal protocols and procedures to make it function — clinical pharmacists. Clinical pharmacists view every patient's chart at least once every day. The chart not only provides information for dispensing, but also gives pharmacists an opportunity to monitor Quality Use of Medicines and communicate with hospital doctors regarding existing or potential problems.

By the time the "prescription" is processed and the medication dispensed, quality issues have been addressed and a detailed record created. From the dispensing record, a "medication profile" could be generated This can provide the patient with consumer information regarding each drug, its dose, frequency of administration and mode of action, and can act as an accurate and up-to-date discharge summary. In addition, a clear, legible copy can be faxed, mailed or electronically transmitted to the patient's GP, pharmacist, specialist, allied healthcare professional, rehabilitation hospital or aged-care facility.

Our organisation provides medication management services to a large number of aged-care facilities as well as to public and private hospitals, and correctional facilities. Quality Use of Medicines monitoring constitutes a vital part of our clinical pharmacists' duties. It provides an effective, accurate and timely method of communicating detailed discharge summaries (which have undergone thorough Quality Use of Medicines screening) to GPs and other interested parties.

By including hospital clinical pharmacists in the Quality Use of Medicines monitoring and evaluation process, meaningful information can be obtained, appropriate judgements made, effective communication conducted and optimum pharmacotherapy outcomes achieved.

1. Mant A, Kehoe L, Cockayne NL, et al. A Quality Use of Medicines program for continuity of care in therapeutics from hospital to community. *Med J Aust* 2002; 177: 32-34.
2. New PW. A Quality Use of Medicines program for continuity of care in therapeutics from hospital to community [letter]. *Med J Aust* 2002; 177: 575. ❑

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IN REPLY: Gelb points out that clinical pharmacists can, and in some cases do, provide useful communication to general practitioners following hospitalisation, as well as for their patients in aged care facilities. Regrettably, clinical pharmacists are in short supply, even in teaching hospitals; thus, in practice, their expertise often cannot be fully utilised.[1] Attention to this shortage is clearly warranted to safeguard patient care.

We agree that clinical pharmacists (hospital and community based) should be included in the Quality Use of Medicines monitoring and evaluation process. In addition, we urge all healthcare

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 for how to cite references other than journal articles).

providers to take responsibility for careful and timely communication to ensure continuity of patient care.

1. Kaye KI, Mant A, Brien JE, Kehoe L. Evaluation of the implementation and effectiveness of the Australian Pharmaceutical Advisory Council (APAC) national guidelines to achieve the continuum of quality use of medicines between hospital and the community. Report to the Commonwealth Department of Health and Ageing. October 2002. ❑

Clinical practice guidelines for depression in young people

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TO THE EDITOR: We would like to comment on a recent article by Chan et al on clinical practice guidelines for depression in young people.[1] We disagree with their proposal to amend National Health and Medical Research Council (NHMRC) guidelines[2] to include a statement that "SSRIs [selective serotonin reuptake inhibitors], particularly fluoxetine and paroxetine, should also be considered as a first-line treatment" for major depression in young people. We believe that there is insufficient evidence to assign a grade of "E1"[2] to this statement. Chan et al[1] quote three randomised controlled trials (RCTs) and one systematic review in support of their argument, but as yet the results of only two of the three RCTs have been published.[3,4] Unfortunately, Chan et al do not include a critical appraisal of the significant methodological and analytical problems with each of the studies. Nor is any comment made about risk–benefit ratios, or the fact that even if the results were sound the clinical relevance of such small differences between active drug and placebo is questionable.[5] The following brief commentary on the two studies highlights the dangers of carrying out sophisticated procedures such as meta-analysis without sufficient attention to the quality of the trials included in the analysis.

The very high dropout rates (46% of 48 for placebo; 29% of 48 for fluoxetine) in the study by Emslie et al[3] raise questions about the reliability of the results. Other interpretations of the findings are plausible. For example, in their study, significant advantage to fluoxetine over placebo on the Clinical Global Impressions improvement rating (a primary outcome measure) was lost when only patients completing the trial were counted ($P = 0.2$).

More worrying is that Chan and colleagues do not seem to have noticed the dangerously distorted reporting in the study by Keller et al.[4] On neither of the two designated primary outcome measures (*change* from baseline in Hamilton Rating Scale for Depression [HAM-D], and *response*, set as "fall in HAM-D to $\leqslant 8$ or by $\geqslant 50\%$") did paroxetine differ significantly from placebo. But Keller and colleagues never report this negative finding. Instead, the criteria for *response* are covertly altered (to "fall in HAM-D to $\leqslant 8$", which does achieve significance). The authors then erroneously claim significance on this (altered) primary outcome measure, ignoring the lack of significant *change*. Thus, a study that showed no significant improvement on either of two primary outcome measures is reported as demonstrating unqualified efficacy.

Similar problems can be found in a more recent article by Emslie and colleagues[6] (published after the review by Chan et al[1]), in which the authors openly acknowledge that the difference between fluoxetine and placebo on their prospectively defined primary outcome measure did not reach statistical significance, yet claim to have demonstrated the drug's efficacy.

Another worry is that Chan and colleagues, in their list of proposed changes to NHMRC recommendations,[1] suggest that the availability of SSRIs obviates the need for more expert and thoughtful assessment and management of depression. We are uncomfortable that the prescribing and management of psychotropic medication is portrayed as requiring relatively few skills and resources, to be carried out by those general practitioners who lack training in mental health and/or access to expert mental health services.

We urge the NHMRC to maintain a conservative approach to the use of psychotropic drugs in children with depression unless more convincing evidence is forthcoming.

1. Chan RTW, Rey JM, Hazell PL. Clinical practice guidelines for depression in young people: are the treatment recommendations outdated? *Med J Aust* 2002; 177: 448-451.
2. National Health and Medical Research Council. Depression in young people: clinical practice guidelines. Canberra: Australian Government Publishing Service, 1997.
3. Emslie GJ, Rush AJ, Weinberg WA, et al. A double-blind, randomized, placebo-controlled trial of fluoxetine in children and adolescents with depression. *Arch Gen Psychiatry* 1997; 54: 1031-1037.
4. Keller MB, Ryan ND, Strober M, et al. Efficacy of paroxetine in the treatment of adolescent major depression: a randomized, controlled trial. *J Am Acad Child Adolesc Psychiatry* 2001; 40: 762-772.
5. Kirsch I, Moore TJ, Scoboria A, Nicholls SS. The emperor's new drugs: an analysis of antidepressant medication data submitted to the U.S. Food and Drug Administration. *Prevention & Treatment* 2002; Vol. 5, Article 23. Available at: http://www.journals.apa.org/prevention/volume5/pre0050023a.html (accessed Jan 2003).
6. Emslie GJ, Heiligenstein JH, Wagner KD, et al. Fluoxetine for acute treatment of depression in children and adolescents: a placebo-controlled, randomized clinical trial. *J Am Acad Child Adolesc Psychiatry* 2002; 41: 1205-1215. ❑

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IN REPLY: We thank Jureidini and Tonkin for their comments. In relation to their criticism of the study by Emslie et al,[1] intention-to-treat analysis is the accepted standard. In relation to the study by Keller et al,[2] their criticism about the criteria for response has already been answered elsewhere. (Criteria were defined in the report as a final Hamilton Rating Scale for Depression [HAM-D] score that was $\leqslant 8$ or a reduction from baseline of $\geqslant 50\%$. Dual criteria were selected because the scores at entry could range from a minimum of 12 [set by protocol] to a maximum of 53 [highest scores for the 17-item HAM-D]. Limiting response to either a 50% reduction or a specified cut-off point would impede patients at the lower end of the ranges from meeting the criterion.[3]) The concern about the absence of differences in change scores on the HAM-D cannot be resolved, as mean change in scores and standard errors of the means were not reported. However, 63.3% (57/90) of subjects taking paroxetine ($P = 0.02$ versus placebo) achieved a HAM-D total score of $\leqslant 8$ at endpoint. With respect to the size of difference in response rate to active treatment versus placebo, the results of trials involving selective serotonin reuptake inhibitors (SSRIs) in children

and adolescents are similar to those reported in adults (ie, SSRIs achieve a response in about 20% more participants than placebo,[4] which is similar to the 26% difference between cognitive behavioural therapy and various control conditions[5]). Consistent with these results, the US Food and Drug Administration has recently approved fluoxetine to treat children and adolescents aged seven to 17 years for major depression. We believe that there are sufficient new treatment data (including a study,[6] published since the submission of our article, showing fluoxetine's superiority over placebo in symptom improvement and in remission rates) to warrant revision of the 1997 guidelines.

Depression affects one in 20 Australian teenagers, few of whom will access specialist mental health services.[7] Contrary to the view of Jureidini and Tonkin, we believe general practitioners must provide treatment for young people suffering depression. A strength of the National Health and Medical Research Council guidelines is that they offer comprehensive advice to promote thoughtful assessment and management of depression in young people.

1. Emslie GJ, Rush AJ, Weinberg WA, et al. A double-blind, randomized, placebo-controlled trial of fluoxetine in children and adolescents with depression. *Arch Gen Psychiatry* 1997; 54: 1031-1037.

2. Keller MB, Ryan ND, Strober M, et al. Efficacy of paroxetine in the treatment of adolescent major depression: a randomized, controlled trial. *J Am Acad Child Adolesc Psychiatry* 2001; 40: 762-772.
3. Keller MB, McCafferty JP. Paroxetine in the treatment of major depression — reply [letter]. *J Am Acad Child Adolesc Psychiatry* 2002; 41: 1270.
4. Walsh BT, Seidman SN, Sysko R, Gould M. Placebo response in studies of major depression: variable, substantial and growing. *JAMA* 2002; 287: 1840-1847.
5. Harrington R, Whittaker J, Shoebridge P, Campbell F. Systematic review of efficacy of cognitive behaviour therapies in childhood and adolescent depressive disorder. *BMJ* 1998; 316: 1559-1563.
6. Emslie GJ, Heiligenstein JH, Wagner KD, et al. Fluoxetine for acute treatment of depression in children and adolescents: a placebo-controlled, randomized clinical trial. *J Am Acad Child Adolesc Psychiatry* 2002; 41: 1205-1215.
7. Rey JM, Sawyer MG, Clark JJ, Baghurst PA. Depression among Australian adolescents. *Med J Aust* 2001; 175: 19-23. ❑

Measuring outcomes in patients with depression or anxiety: an essential part of clinical practice

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TO THE EDITOR: I do not believe that Dinnen's comments[1] should be so easily dismissed as suggested by the academics proposing that general practitioners should do questionnaires,[2,3] at least in New South Wales.

I write this as barely a month has passed since a most damning report was released by a NSW Parliamentary Inquiry into Mental Health Delivery.

A prime example of arrogance and loss of contact with the reality of clinical services by academia and administration is that, during the demise of mental health services in NSW, the services have been forced to complete a new 30-page admission process for every admission. Just what was needed by registrars spending hours trying to find beds for seriously mentally ill patients!

The gulf between academia and administration on the one hand and real clinical services on the other is now huge in NSW, at least in mental health services. No one outside real clinical services has any credibility or right to demand doctors, let alone hard-pressed GPs, engage in dubious and very likely useless research projects without very special funding to support the project.

I found the K10 questionnaire extraordinarily simplistic compared with a Mental State Examination (MSE). Surely, if there is concern, doctors should be encouraged to revise how the MSE is carried out, and not encouraged to adopt "cookbook" medicine.

1. Dinnen A. Measuring outcomes in patients with depression or anxiety: an essential part of clinical practice [letter]. *Med J Aust* 2003; 178: 48.
2. Hickie IB, Andrews G, Davenport TA. Measuring outcomes in patients with depression or anxiety: an essential part of clinical practice. *Med J Aust* 2002; 177: 205-207.
3. Andrews GA, Hickie IB, Davenport TA. Measuring outcomes in patients with depression or anxiety: an essential part of clinical practice [letter]. *Med J Aust* 2003; 178: 48. ❑

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TO THE EDITOR: As a general practitioner I was gratified to see Dinnen's letter[1] in which he questioned the "urgent need" for GPs to use more questionnaires for depression management. Reading the professorial reply,[2] I despair. As more and more specific health promotions are introduced (eg, Asthma 3-Step Plans, Diabetic Care protocols, Health Assessments, Care Plans) we have to consider not just our patient's problems, but which forms to fill out or numbers to put down to fulfil Health Insurance Commission requirements or be correctly remunerated.

By the time a depressed patient is sitting in my room, he or she wants to be correctly diagnosed and treated, not to be given a form to fill out. In my opinion, to hand a form to a depressed patient who has tearfully told me his or her problems is an insult. We do not (yet) expect patients to fill out a checklist for heart failure. As to the suggestion that the form be filled out in the waiting room, how is this to be done? Should the patient be sent out again with form in hand? Privacy concerns do not allow reception staff to hand out such forms, and the waiting room is not the best place to fill them out if they are needed. In reality, the GP is likely to give the patient a form and then go have a coffee or make a telephone call.

No psychiatrist will ever receive a referral from me based on a K10 score. I may, however, mention that my patient still has suicidal impulses, cries a lot, has trouble sleeping and cannot concentrate at work. That should be easy enough for anyone to understand.

1. Dinnen AH. Measuring outcomes in patients with depression and anxiety: an essential part of clinical practice [letter]. *Med J Aust* 2003; 178: 48.

2. Andrews G, Hickie IB, Davenport TA. Measuring outcomes in patients with depression and anxiety: an essential part of clinical practice [letter]. *Med J Aust* 2003; 178: 48. ❑

What is pathography?

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TO THE EDITOR: I read with great interest about your search in dictionaries for the word "pathography".[1]

Pathography[2] originates from reflections on genius and its possible association with insanity, a question that has occupied experts in many fields since Socrates, Plato and Aristotle. The first psychiatric scientific treatise concerning this question was contributed by Moreau de Tours in 1859.[3] Inspired by him, Cesare Lombroso, in 1863, coined the famous expression *genio et follia*, and contributed many, albeit somewhat uncritical, pathographies.

The term pathography was first used about 1899 by the German psychiatrist Paul Julius Möbius, who contributed with several seminal pathographies, including Rousseau, Goethe, Schopenhauer and Nietzsche.

Among other famous pathographers should be mentioned Freud, W Lange, Jaspers, Birnbaum and Kretschmer.

Pathography can be defined[4] as

> . . . historical biography from a medical, psychological and psychiatric viewpoint. It analyses a single individual's biological heredity, development, personality, life history, and mental and physical pathology, within the sociocultural context of his/her time, in order to evaluate the impact of these factors upon his/her decision-making, performance and achievements. No preconceived format can be assumed as the method depends on the nature of the various available materials and on the specific inquiry. A prerequisite for plausible pathographical results is a thorough knowledge and understanding of psychopathology, and of the borderland between normal and abnormal mental life, combined with a capacity for [sober] historical judgement. . . . The pathographical method is applicable to any personality, sick or sound, provided that sufficient biographical sources are available. The pathographical result is a facet but often an indispensable one.

Subjects of pathography have traditionally been famous people in all areas of human achievement. Pathography is also indispensable in assisting historians, political scientists and other groups in their quest for a better understanding of events where leaders or other "very important persons" have played a significant role, and where personality or illness, physical or mental, has been decisive, at times with far-reaching consequences for nations.[5,6] History is replete with such examples.

1. van der Weyden MB. Extinguishing empathy [From the Editor's desk]. *Med J Aust* 2002; 177: 401.
2. Schioldann J. Den patografiske tradition og metode. *Dansk Medicinsk Årbog (Copenhagen)* 1983; 91-104.
3. Moreau (de Tours) J. *La psychologie morbide dans ses rapport avec la philosophie de l'histoire ou l'influence des névropathies sur le dynamisme intellectual.* Paris: Masson, 1859.
4. Schioldann J. The Life of D. G. Monrad, 1811–1887. Manic-depressive disorder and political leadership. Odense: Odense University Press, 1988.
5. Lawrence J. The psychiatry of leadership and the psychiatrist as leader. *Aust N Z J Psychiatry* 1988; 22: 245-256.
6. Schioldann J. The psychiatry of leadership. *Aust N Z J Psychiatry* 1988; 22: 344-346. ❑

Is asthma prevention possible with dietary manipulation?

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TO THE EDITOR: In the abstract of his article on asthma prevention with dietary manipulation,[1] Mellis states that "we know" that the major modifiable dietary environmental risk factors for childhood asthma are not having been breastfed and low intake of omega-3 fatty acids.

In his discussion of the evidence, Mellis suggests that breastfeeding may be protective and, importantly, acknowledges the controversy. He further states (in the abstract) that observational studies have shown a reduction in childhood asthma in children who eat fish regularly (that is, have a high intake of omega-3 fatty acids), similar to those who were exclusively breastfed for three months. However, he provides no references for these observational studies, and nor does he discuss any specific evidence in support of including omega-3 fatty acids for reducing childhood asthma.

While there are some suggestions of such an association, the evidence is extremely limited compared with the extensive literature on the potential for the protective effect of breastfeeding. Further, there are substantial methodological issues associated with the few studies that do exist, not the least of which is the measurement of the relevant dietary parameters.

Australian studies have suggested a protective influence of at least two fish meals per week on bronchial hyperresponsiveness in 7–11-year olds[2] and of eating oily fish[3] on the prevalence of childhood asthma. However, neither of these studies had the capacity to measure omega-3 fatty acid nor fish intake in a valid way. These limitations were acknowledged by the authors of the studies, and have been noted by others;[4] they need to be included in any discussion of a putative protective effect. It should also be noted that the biological plausibility of such an association has been challenged.[4] There are many valid reasons for promoting the consumption of omega-3 fatty acids, but shouldn't we wait for the outcome of the randomised clinical trial currently under way before accepting the statement that "we know" that a low intake of these fatty acids increases the risk of childhood asthma?

1. Mellis CM. Is asthma prevention possible with dietary manipulation? *Med J Aust* 2002; 177 (6 Suppl): S78-S80.
2. Peat JK, Salome CM, Woolcock AJ. Factors associated with bronchial hyperresponsiveness in Australian adults and children. *Eur Respir J* 1992; 5: 921-929.
3. Hodge L, Salome CM, Peat JK, et al. Consumption of oily fish and childhood asthma risk. *Med J Aust* 1996; 164: 137-140.
4. Thien FCK, Woods RK, Walters EH. Oily fish and asthma — a fishy story? [editorial]. *Med J Aust* 1996; 164: 135-136.❑

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IN REPLY: Sherriff is correct in pointing out that the studies showing protection from bronchial asthma (and bronchial hyperresponsiveness) are based on consumption of fish meals rather than a direct measure of omega-3 fatty acid intake. This protection has been observed consistently in cross-sectional studies of New South Wales primary school children. Thus, the level of evidence is at best Level III, albeit using a proxy for omega-3 fatty acid intake.

Results of a randomised-controlled trial of omega-3 fatty acid supplementation currently under way in western Sydney are now in the public arena at 18-month follow-up.[1,2] At this early stage, it is uncertain who has genuine asthma rather than other wheezing syndromes. Nevertheless, the group who received omega-3 fatty acid supplementation have differences in rates of wheeze compared with those not supplemented.[1,2] For example, the rate of "ever" having had wheeze was 52.6% in the controls versus 42.8% in the supplemented group (absolute risk reduction, 9.8%; number need to treat, about 10).

In the table of recommendations in my article,[3] I carefully pointed out that supplementing infants with omega-3 fatty acid is something to "consider" rather than strongly recommending it. It should also be noted the level of evidence is low (Level III). Stronger recommendations will depend on the long-term results of randomised trials, such as the western Sydney trial.[1,2] In summary, at this stage the only strong dietary recommendations which can be made are:

- not to use strict elimination diets during pregnancy (Level I evidence); and
- to consider using lactobacillus probiotic supplements.

The evidence for lactobacillus is Level II (from a single randomised controlled trial), although the protection shown is for atopy rather than asthma. Clearly, the children in the lactobacillus study will need further follow-up, and the trial will need to be repeated in other populations.

All of this highlights the need for better-quality studies in the area of primary prevention of asthma, based on dietary factors during pregnancy or early infancy.

1. Mihrshahi S, Peat JK, Marks GB, et al. Effect of house dust mite reduction and dietary Omega-3 fatty acid supplementation from birth on atopy and wheeze in 18 month old children. Available at: http://www.thoracic.org.au/p35.pdf (accessed February 2003).
2. Mihrshahi S, Peat JK, Marks GB et al. Eighteen-month outcomes of house dust mite avoidance and dietary fatty acid modification in the Childhood Asthma Prevention Study (CAPS). *J Allergy Clin Immunol* 2003; 111: 162-168.
3. Mellis CM. Is asthma prevention possible with dietary manipulation? *Med J Aust* 2002; 177 (16 Sep Suppl): S78-S80. ❑

The verdict from ALLHAT

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TO THE EDITOR: The publication of the main results of the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT), and the accompanying editorial, triumph the role of thiazide diuretics as first-line management for hypertension.[1,2] It brought to mind the lines from the nursery rhyme *Old Mother Hubbard* — "And when she went there, the cupboard was bare."

Simple frequency analysis of diuretic antihypertensive medications listed in the *Australian Medicines Handbook* (1998 and 2003) revealed that the total number of thiazide diuretics available as monotherapy in 1998 was six (bendrofluazide chlorothiazide, chlorthalidone, hydrochlorothiazide, methyclothiazide and indapamide), and in 2003 three (bendrofluazide, chlorthalidone and indapamide).[3,4]

1. Appel L. The verdict from ALLHAT — thiazide diuretics are the preferred initial therapy for hypertension. *JAMA* 2002; 288: 3039-3042.
2. The ALLHAT Officers and Coordinators for the ALLHAT Collaborative Research Group. Major outcomes in high-risk hypertensive patients randomized to angiotensin-converting enzyme inhibitor or calcium channel blocker vs diuretic. The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT). *JAMA* 2002; 288: 2981-2997.
3. Australian Medicines Handbook, 1st edition. Adelaide: Australian Medicines Handbook Pty Ltd, 1998.
4. Australian Medicines Handbook, 4th edition. Adelaide: Australian Medicines Handbook Pty Ltd, 2003. ❑

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