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Further reading

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Professor Rey was a member of the advisory committees for atomoxetine (Eli Lilly) and methylphenidate (Janssen-Cilag) and was funded by Eli Lilly to attend an international conference.

Letters

Letters, which may not necessarily be published in full, should be restricted to not more than 250 words. When relevant, comment on the letter is sought from the author. Due to production schedules, it is normally not possible to publish letters received in response to material appearing in a particular issue earlier than the second or third subsequent issue.

Varicella vaccine

Editor, – Despite the risks, the article 'Frequently asked questions about varicella vaccine' (Aust Prescr 2005;28:2–5) recommends widescale immunisation. There are three arguments against this strategy. Firstly, vaccine immunity may wane over time leaving susceptible adults. Secondly, immunising part of the population may shift the disease burden to those who are not vaccinated and because they will be less likely to acquire chickenpox in childhood they risk more severe disease in adulthood. Thirdly, the effect of vaccination on the incidence of herpes zoster is unknown.

The data so far show that chickenpox in immunised individuals is less severe. However, it is too early to know how this will change as immunised infants reach adulthood.

In 2000 mathematical modelling showed that immunising 90% of infants would produce an initial 'honeymoon' period of low incidence, one or more post-honeymoon epidemics in adolescents and young adults 10–20 years later, and an equilibrium reached after 20–40 years in which the incidence in adults is similar to that in the pre-vaccine years.[1] The evidence from the USA on reduced incidence in all age groups covers only five years of experience, which is within the honeymoon period predicted by the modelling. This is insufficient time for epidemics in adults to occur through the build-up of susceptible people, as partial population immunity increases the interepidemic interval.

The impact of varicella vaccine on herpes zoster is complex. There is reasonable evidence that adults exposed to children, or exposed to chickenpox, have less chance of developing zoster, through presumed immunologic boosting by exposure to varicella zoster virus.[2] Modelling shows that immunisation causes an increase in herpes zoster for up to 50 years until immunised infants reach old age.

Due to the infectivity of reactivated herpes zoster it is not possible to eliminate varicella zoster virus in the way measles or polio could be eliminated completely. The aim of immunisation is therefore to reduce the burden of varicella disease rather than disease elimination. Since the burden of serious disease, particularly mortality, is in adults, and the modelling shows that in the long term the incidence in adults will not be affected by even high levels of vaccination coverage, the logic of universal vaccination has to be questioned.

Vaccination undoubtedly reduces childhood disease and saves the costs of medical care, childcare costs and lost income for parents while they look after sick children. Health decisions, however, should be primarily based on health considerations rather than economics.

The current low burden of disease from varicella means that it would take only a small rise in varicella in adults for us to be worse off than we were without the vaccine.

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Associate Professor Jonathan Carapetis, one of the authors of the article, comments:

Dr Ewald raises the question of how mathematical modelling should be used in determining public health policy. Should we refrain from using a vaccine that can bring immediate reductions in morbidity and mortality because of predictions that there might be ill effects in the future? Some reassurance comes from US data, which have failed to show any change in rates of zoster up to seven years after introduction of varicella zoster virus immunisation.[1] It is still early days, and this study may have taken place during the initial 'honeymoon' period. Even if the models prove correct and we begin to see early increases in adult zoster followed by later increases of varicella in adolescents and young adults, there is an obvious solution: booster doses. We already give booster doses of pertussis, diphtheria and tetanus vaccine in adolescence, and regular influenza and pneumococcal immunisation is recommended for high-risk adults.

A recent study of more than 38 000 elderly people in the USA found that a live attenuated varicella zoster virus vaccine reduced the incidence of zoster by 51%.[2] This provides reassurance that vaccination of adolescents or adults will be an effective countermeasure to the model predictions, if they eventuate.

The models of post-varicella zoster virus vaccine disease patterns are important in highlighting the need for better surveillance of varicella and zoster, the longer-term questions relating to duration of immunity, and the importance of a flexible immunisation policy that can react quickly to changes in the epidemiology of vaccine preventable diseases. The uncertainty surrounding predictions from models means that they should not be used as a reason to withhold an intervention that can prevent illness and death and save health dollars at the same time.

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First-line medicines in the treatment of hypertension

Editor, –The article by Hill and Smith (Aust Prescr 2005;28:34–7) states that when the blood pressure, on at least three separate occasions, exceeds the threshold pressures which predict an increased cardiovascular risk, treatment is required. They quote systolic and diastolic figures for triggering treatment, but then state that the patient's predicted cardiovascular risk should determine the time for intervention.

When does cardiovascular risk become 'increased'? Over what acceptable level? How is the 'predicted cardiovascular risk' used to delay the time for active intervention when one of the measurements has crossed the red line?

Why is there no reference in the entire article to discussion with the patient of their acceptable risk levels? The New Zealand Cardiovascular Risk Calculator to which they refer us has numbers needed to treat ranging from <10 to >120. The result of treatment is prevention of one cardiovascular event in five years.

This would suggest that even in a high risk 'herd' of patients, drenching all of them delivers benefits to very few. When the 'herd' consists of autonomous fellow human beings, should they not be involved in the good shepherd's calculations?

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Dr S. Hill and Professor A.J. Smith, authors of the article, comment:

Our article's focus was first-choice medicines for hypertension. We could not embark on this without a brief, but not a full, account of the assessment of absolute cardiovascular risk and its application to treatment decisions.

Blood pressure is continuously associated with cardiovascular risk and therefore there is no discrete point at which treatment is mandated. Blood pressure should not be viewed in isolation from accompanying risks such as age, gender, ethnicity, smoking, lipids, glucose, family history and body mass index – the ingredients currently used for calculating absolute cardiovascular risk.[1]

What is an 'acceptable' level of risk? The New Zealand guidelines, and our own National Heart Foundation, recommend lifestyle advice alone for individuals whose risk of a cardiovascular event over the next five years is less than 15%. Any threshold for treatment is a compromise between unnecessary intervention (the 'Number needed to treat (needlessly)'[2], culpable inactivity and economic feasibility. If, however, the approach of establishing absolute cardiovascular risk is taken it is **impossible** to leave the patient out of the discussion. We agree that this is essential for any intervention and particularly one that will last a lifetime.

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Antibiotics for surgical prophylaxis

Editor, – I read with interest the article 'Antibiotics for surgical prophylaxis' (Aust Prescr 2005;28:38–40) and the accompanying Dental notes (Aust Prescr 2005;28:41). While I do agree that surgical removal of the third molar (most often impacted) may be technically classified as 'contaminated', I think we should be more cautious with regards to routine use of antibiotic prophylaxis for this procedure.

Jawbones somehow behave differently when exposed to oral flora as compared to other bones in the body. By experience, we know that the jawbones may be exposed to oral flora as a result of periodontal disease (bony involvement may be severe in advanced cases) or as a result of dental extractions, yet they hardly get infected. I believe these exposures somehow make jawbones more resistant to infection by the oral flora, at least in healthy patients. Most patients can therefore avoid infection following routine dental extraction from a 'contaminated' area without the need for antibiotics. This 'resistance' may also explain the rareness of osteomyelitis in the jawbones even though they are frequently exposed to various dental causes such as trauma, abscesses and severe periodontal disease. A review of the need for antibiotic prophylaxis in third molar surgery concluded that there is no justification for routine prophylaxis.[1]

In view of the popularity of dental implants (technically categorised as insertion of prosthetic material), I would like to highlight a Cochrane review, mentioned in the Australian Dental Journal[2], on the use of prophylaxis to prevent complications following insertion of dental implants. It has been suggested that there is no appropriate scientific evidence to recommend or discourage the use of prophylactic systemic antibiotics. As such, we are still left in the dark on the appropriateness of prophylactic antibiotics for dental implantation. If we were to follow the criteria for surgical prophylaxis, antibiotics would be used because a dental implant is a prosthetic device and is inserted in a 'contaminated' environment.

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Valediction

Robert Moulds

In April this year Professor Robert Moulds stood down as the chairman of the Editorial Executive Committee of *Australian Prescriber*. Professor Moulds first wrote for *Australian Prescriber* in 1982 and 10 years later he joined the Executive Editorial Board of the journal. The Board appreciated Professor Moulds' pharmacological knowledge and in 2000 he became the chairman.

Under Professor Moulds' chairmanship the journal made the transition from the Department of Health and Ageing to the National Prescribing Service. Professor Moulds helped to ensure that the journal's editorial independence was maintained after this transition.

The Editorial Executive Committee became truly international when Professor Moulds became the Professor of Medicine at the Fiji School of Medicine. Despite the travel involved he remained committed to *Australian Prescriber* and regularly returned to Australia to chair the editorial meetings. His valuable contribution over the years is greatly appreciated.