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IN REPLY: Glendenning raises a number of interesting points, which require some clarification.

Are ergocalciferol (vitamin D₂) and cholecalciferol (vitamin D₃) biologically equivalent?

The statement that ergocalciferol and cholecalciferol are bioequivalent in humans is made by most authoritative textbooks, based mainly on evidence from early studies of the antirachitic efficacy of ergocalciferol and cholecalciferol compounds, and contrasts with reduced efficacy of ergocalciferol in birds and monkeys.¹ Recent studies using more precise measurements have raised some doubts as to the absolute equivalency of ergo- and cholecalciferol, but the differences are marginal² and not universally found.³ There are few recent data on relevant biological endpoints. Serum concentrations of the active hormone, 1,25-dihydroxyvitamin D, were not different after administration of ergo- or cholecalciferol,² and increases in bone mineral density were greater after ergocalciferol therapy than after cholecalciferol in patients taking anticonvulsants.¹ In short, on current evidence, differences in biological activity between ergocalciferol and cholecalciferol are likely to be relatively minor.

Are there problems monitoring therapy?

25-Hydroxyvitamin D values may be used for monitoring treatment. The possibilities of impaired detection of the 25-hydroxy metabolite of ergocalciferol by some assays,² and perhaps a smaller rise in total 25-hydroxyvitamin D concentrations after low doses of ergocalciferol, should be borne in mind when monitoring therapy.

What is the current recommendation for vitamin D supplementation?

While the availability of larger dose sizes and/or cholecalciferol preparations would be helpful, 800 IU of ergocalciferol and 1 g of calcium for six months was shown to reduce secondary hyperparathyroidism in older patients,¹ and 600 IU/day (same for

ergo- and cholecalciferol) is the new recommended adequate intake for older patients with limited sun exposure.¹

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Confronting conflict of interest in research organisations: time for national action

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TO THE EDITOR: A recent editorial in the *Journal* focused on the "blurring of research ideals and corporate interests".¹ But there are other funding and commissioning bodies, including government, whose wants or needs also have the potential to blur research ideals and exert control over what can be published. In recent times, those who pay the piper increasingly want to call the tune. Understandably, this is also an issue for research into Aboriginal ill health.^{2,3} Van Der Weyden's plea for the development of national guidelines on institutional conflict of interest should therefore be broadened to include all funding bodies.

One suggestion for inclusion in these guidelines, to enhance public interest in research, is an obligation for authors to state not only their sources of funding, but "the origin of the research question they are attempting to answer"⁴ and the person or group who initiated the funding of the project.

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Recent appearance of clindamycin resistance in community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) in south-east Queensland

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TO THE EDITOR: We report the appearance of erythromycin and inducible clindamycin resistance in the south-west Pacific strain of non-multiresistant methicillin-resistant *Staphylococcus aureus*, which has recently appeared in eastern Australia. Infections occur predominantly in Polynesian people and are usually community-acquired. Most strains belong to Western Samoan phage patterns (WSPP1 or WSPP2) and pulso-type A when typed by pulsed-field gel electrophoresis.^{1,2} These strains are resistant to all β -lactams, but are usually susceptible to erythromycin, clindamycin, gentamicin, tetracycline, trimethoprim-sulfamethoxazole and ciprofloxacin. Although most of these antibiotics would not be recommended for therapy,³ clindamycin has been recommended for non-parenteral treatment of soft-tissue and bone infections, as it is efficacious in treating similar infections caused by methicillin-susceptible *S. aureus*.⁴

Twenty isolates of community-acquired, non-multiresistant pulso-type A MRSA were collected from patients from southern Brisbane and Logan in 1997 and 1998.² A further 16 isolates were obtained from Ipswich patients between December 1998 and February 2001. We found that all 36



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