

Multifaceted Educational Program Increases Prescribing of Preventive Medication for Corticosteroid Induced Osteoporosis

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ABSTRACT. Objective. Despite evidence that oral corticosteroids increase fracture risk and the existence of guidelines for the prevention of corticosteroid induced osteoporosis, few patients prescribed longterm corticosteroids receive osteoporosis prevention. We performed a controlled trial of a comprehensive educational program aimed at increasing the use of osteoporosis preventive therapy in patients prescribed longterm oral corticosteroids.

Methods. The intervention was conducted in Southern Tasmania, Australia, using Northern Tasmania as a control area. All general practitioners and community pharmacies were sent educational material and locally produced guidelines on the prevention of corticosteroid induced osteoporosis. This was followed by academic detailing visits and reminders. Pharmacists were provided with supplies of an educational refrigerator magnet, intended for patients. Outcomes were measured using evaluation feedback from the general practitioners and pharmacists, and drug utilization data provided by (1) a series of patients presenting to hospital and taking oral corticosteroids for at least 3 consecutive months; and (2) dispensing of osteoporosis preventive therapy under the Australian Pharmaceutical Benefits Scheme.

Results. The prevalence of osteoporosis preventive therapy increased from 31% of admitted hospital patients taking longterm oral corticosteroids to 57% postintervention ($p < 0.0001$). The use of bisphosphonates (6% to 24% of patients), calcium (5% to 19%), and vitamin D (3% to 11%) all increased significantly. Prescription data also indicated a significant ($p < 0.01$) increase in the use of osteoporosis preventive therapy in the intervention region.

Conclusion. A multifaceted education program, incorporating academic detailing of general practitioners and community pharmacists, increased the use of osteoporosis prevention strategies in longterm oral corticosteroid users. (J Rheumatol 2004;31:550-6)

Key Indexing Terms:

CORTICOSTEROIDS OSTEOPOROSIS PREVENTION EDUCATION PRESCRIBING

Osteoporosis is a major public health problem and its prevalence is increasing¹. In Australia, roughly one in 2 women and one in 4 men over the age of 60 years will develop an osteoporotic fracture².

While corticosteroid drugs can produce striking improvement in a number of diseases, a major limiting factor is the risk of serious complications with longterm treatment, including osteoporosis. A number of studies have reported decreases in bone mineral density and/or an increase in frac-

ture risk during oral corticosteroid treatment³⁻⁷. Corticosteroid induced osteoporosis is the most common type of secondary osteoporosis⁸. Epidemiological data suggest that corticosteroid treatment doubles the risk of fractures of the hip and distal radius, and at least quadruples the risk of vertebral fracture⁶.

There are effective treatments for corticosteroid induced osteoporosis^{9,10}. Guidelines have been disseminated for the prevention of corticosteroid induced osteoporosis in different settings^{9,11}. However, many users of longterm corticosteroids still do not receive adequate, if any, preventive therapy¹²⁻²⁰. A study from our institution¹⁴ showed that only 21% of patients taking oral corticosteroids for more than 4 weeks received prophylaxis against osteoporosis. We evaluated an education campaign directed at doctors, pharmacists, and patients to increase the use of osteoporosis preventive therapy in patients prescribed longterm oral corticosteroids.

MATERIALS AND METHODS

The study was approved by the Research and Ethics Committees of the

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Royal Hobart Hospital. The Division of General Practice (Southern Tasmanian Division), the Pharmacy Guild of Australia, and Osteoporosis Australia also supported the project.

A composite version of the guidelines produced by the American College of Rheumatology (ACR)⁹, a UK Consensus Group on the management of glucocorticoid induced osteoporosis¹¹, and Osteoporosis Australia²¹ was developed for local use in consultation with medical specialists (Figure 1). The intervention study was conducted in Southern Tasmania (population 230,000), using the northern region of the state as the control area (population 130,000). The guidelines, with an explanatory covering letter, were sent to each general practitioner (total about 270) practicing within Southern Tasmania, using the Medical Council of Tasmania's *Register of Legally Qualified Medical Practitioners*, in October 2001. The results of the recent local study showing evidence of underutilization of both monitoring of bone mineral density and prescribing of recommended osteoporosis-preventing agents¹⁴ were emphasized.

During January to May 2002 all general practitioners in Southern Tasmania were contacted to discuss corticosteroid induced osteoporosis. The research pharmacist (MN) visited each general practitioner and

discussed the rationale of prescribing osteoporosis preventive therapies and treatment to patients receiving longterm oral corticosteroids. Additional reference material was provided on request.

The guidelines were also sent to all community pharmacies in Southern Tasmania (n = 69). All the pharmacies were subsequently visited and the pharmacist on duty was detailed on the guidelines. Each pharmacy was also given a supply of refrigerator magnets (Figure 2) intended for patients presenting with a prescription for an oral corticosteroid drug (and who had been taking it for longer than 3 months or who were undergoing treatment expected to last for longer than 3 months). While the academic detailer was in the pharmacy, shelf markers (labelled "Is patient on longterm corticosteroids? Consider osteoporosis prevention") were also placed below the different brands of oral corticosteroids to serve as a reminder for pharmacists when they selected the products from the shelf for dispensing.

Participating general practitioners and pharmacists were surveyed anonymously (using a visual analog scale) to assess the usefulness of the mailed information and academic detailing visit. In particular, general practitioners were asked if they were more likely to consider preventive therapy in their patients using corticosteroids following the educational program.

GUIDELINES FOR THE PREVENTION AND MANAGEMENT OF CORTICOSTEROID-INDUCED OSTEOPOROSIS

For adult patients prescribed an oral dose of ≥ 7.5 mg/day prednisolone (or equivalent) or ≥ 800 mcg/day of inhaled budesonide (or equivalent) for ≥ 3 months

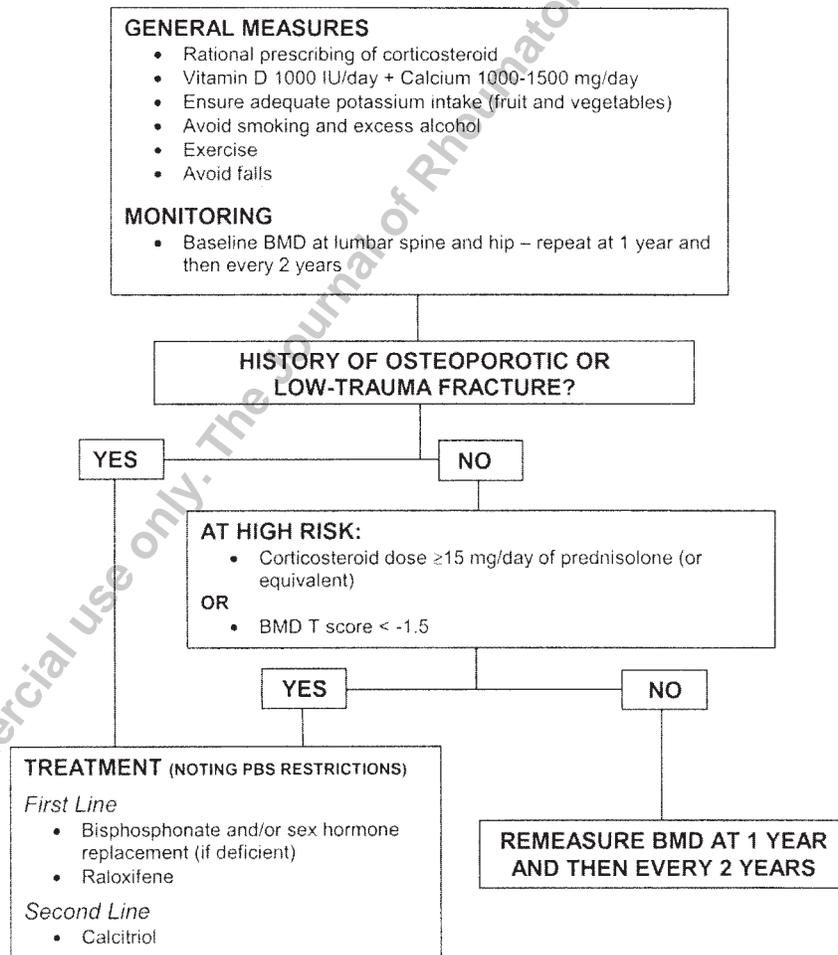


Figure 1. Guidelines distributed to doctors and pharmacists. Note that in Australia, vitamin D is only commonly available alone as 1000 IU.

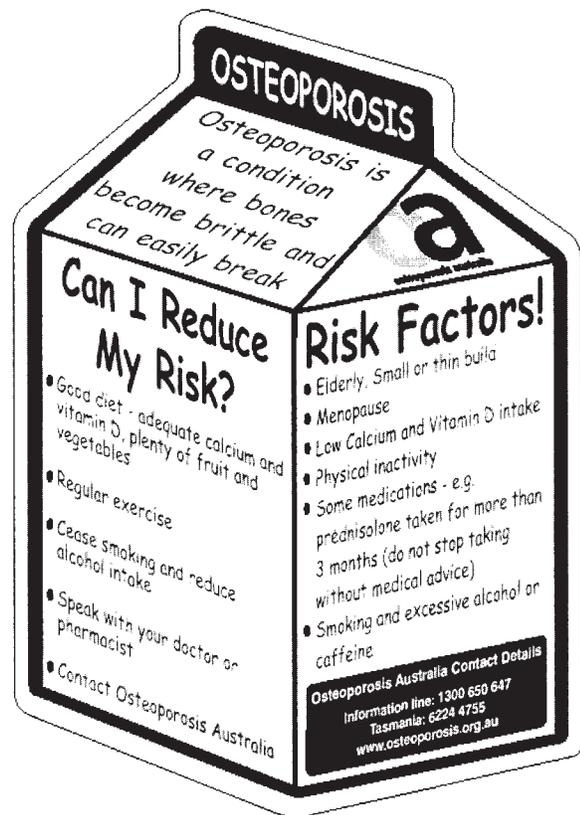


Figure 2. Refrigerator magnet supplied to community pharmacists for distribution to patients prescribed longterm corticosteroids.

Pharmacists were asked if they were more likely to discuss preventive measures with patients taking oral corticosteroids and to refer patients to a general practitioner when appropriate. Completed evaluation forms were returned via reply-paid envelopes.

Two sources of prescribing data were utilized to measure the outcome of the educational intervention. As in our previous study¹⁴, data were collected on patients admitted to the Royal Hobart Hospital, a 400-bed acute care academic hospital and the only major public hospital in the southern region of Tasmania. The baseline sample consisted of all adult patients who had been taking oral corticosteroids for at least 3 months (as identified from medical records and drug charts) and who were admitted to the medical wards of the hospital during the period April 1, 2001, to September 30, 2001. The only exclusions were patients whose medical records were incomplete, those who were receiving oral corticosteroids for palliative care, or those who were unable to answer questions about their therapy.

Variables recorded for each patient included demographic information, reason for admission to hospital, smoking and alcohol intake, medical history, medication history on admission, bone densitometry data when available, and drug therapy on discharge from hospital. Concurrent medications that either increased the risk of osteoporosis (e.g., loop diuretics, anti-convulsants) or increased the risk of falls (e.g., benzodiazepines, tricyclic antidepressants) were recorded. Patients were also asked a short series of questions during their hospitalization to extract some of the relevant information (e.g., level of sun exposure per week, family history of osteoporosis, whether they had ever sustained a low trauma fracture). One month after completion of the academic detailing phase of the project, followup data were again collected on consecutive adults admitted to the Royal Hobart Hospital who had been taking oral corticosteroids for at least 3

months. This procedure was identical to that for the baseline data collection.

Pharmaceutical Benefit Scheme (PBS) and Repatriation Pharmaceutical Benefits Scheme (RPBS) dispensing data were also obtained from the Department of Health and Ageing for alendronate, risendronate, etidronate, raloxifene, calcitriol, calcium carbonate, and prednisolone for North Tasmania (postcodes 7200-7299; control region) and South Tasmania (7000-7199; intervention region) for March–August 2001 and March–August 2002. We did not include the use of hormone replacement therapy or testosterone therapy in dispensing data analysis because these medications are primarily used for indications other than osteoporosis prevention or treatment in Australia. Data were unavailable on the use of vitamin D, which is not listed on the PBS or RPBS.

The unit quantities of each drug dispensed were converted to defined daily doses (DDD)²². The key variable examined was the total DDD of osteoporosis preventive therapy, expressed as a ratio to the total DDD of prednisolone between North and South Tasmania. Nonparametric techniques were principally used to describe patient characteristics and examine differences in variables between subgroups of patients. Statistical comparisons for drug dispensing data were made between the different areas of the state (i.e., South/intervention region vs North/control region), both before and after the intervention, and within each study area (before vs after intervention) using a normal approximation to the binomial distribution. A p value less than 0.05 was considered statistically significant. Statistical analysis was performed using Statview® 5.01 (Abacus Concepts Inc., Berkeley, CA, USA).

RESULTS

During the academic detailing period 200 general practitioners (74% of those in Southern Tasmania) and 69 pharmacists (81 pharmacists) were visited. Each general practitioner visit was about 15 minutes in duration. There was an evaluation survey response of 83% for general practitioners and 47% for pharmacists. The sessions were favorably accepted by general practitioners, with a median score of 8 on the visual analog scale (range 1–10; 0 = not useful, 10 = very useful) when they were asked whether the detailing had been useful. Pharmacists also found the session useful, with a median score of 8 on the scale (range 5.5–10).

General practitioners and pharmacists were also asked to indicate whether they had routinely considered osteoporosis prevention in patients prescribed longterm corticosteroids (where 0 = strongly disagree and 10 = strongly agree). The general practitioners' median score was 6.5 (range 0.5–10) and the pharmacists' median score was 2.5 (0–8). Both the general practitioners and pharmacists were asked whether they agreed or disagreed that they were more likely to consider preventive therapy in patients prescribed longterm corticosteroids after being exposed to the educational program. The median score (0 = strongly disagree, 10 = strongly agree) for general practitioners and pharmacists was 8.5 (range 0–10) and 9 (6–10), respectively.

In the hospital based study, a total of 233 patients were included — 113 in the preintervention and 120 in the postintervention group. The 2 groups were very similar in key sociodemographic and clinical variables (Table 1). Patients in the postintervention group were significantly more likely to have been prescribed some form of antiresorptive therapy

Table 1. Hospital patient characteristics before and after the intervention.

	Baseline, n = 113	Postintervention, n = 120
Female, %	61	62.5
Median age, years (range)	71 (20–93)	70 (20–92)
Aged > 65 years, %	63	63
Smoker, %	23	19
Ex-smoker, %	52	54
Median corticosteroid daily dose on admission, mg (range)*	10 (2–50)	7.5 (3–100)
Usual median daily dose, mg (range)*	7.5 (2–50)	7.5 (3–100)
Usual median dose > 7.5 mg daily, %*	56	52
Median time of continuous oral corticosteroid use, mo (range)	60 (3–480)	60 (3–372)
Median number of chronic medical conditions (range)	4 (1–10)	4 (1–12)
Median number of medications on admission (range)	8 (2–19)	8.5 (1–26)
Documented osteoporosis, %	24	33
Hypogonadism, %		
Men	7	9
Women (menopause < 45 yrs)	38	31
Indication for corticosteroid use, %**		
Asthma	11	8
Chronic obstructive pulmonary disease	27	21
Rheumatoid arthritis	27	27
Inflammatory bowel disease	4	5
Muscular/connective tissue disease	23	23
Temporal arteritis	5	4
Other	17	20
Previous fracture, %	41	43
If yes, while on corticosteroids, %	80	83
Low sun exposure [†]	38	43
Family history of osteoporosis	10	13
Taking other medications that increase the risk of osteoporosis, %	41	47
Taking other medications that increase the risk of falls, %	83	79
Receiving osteoporosis preventive therapy, %	31	57

* Prednisolone or equivalent. ** Some patients had more than one indication. † Patients who were not exposed to at least 1 hour of sunlight a week³⁶.

than the preintervention group on presentation to hospital (57% vs 31%; $p < 0.0001$). In particular, patients were more likely to be prescribed a bisphosphonate following the intervention (6% in the preintervention vs 24% in the postintervention group; $p < 0.001$; Table 2). There were smaller, but statistically significant, increases in the use of calcium (5% vs 19%; $p < 0.01$), vitamin D (3% vs 11%; $p < 0.05$), and hormone replacement therapy (5% vs 14%; $p < 0.05$) after the intervention. There was no change in the use of calcitriol after the intervention (17% vs 15%; $p = 0.7$).

About 29% of patients who were prescribed at least 7.5 mg of prednisolone (or equivalent) daily were receiving osteoporosis preventive therapy in the baseline group, and this significantly increased to 51% of the postintervention group ($p < 0.01$). Roughly 34% of patients who had been taking oral corticosteroids continuously for at least 12 months in the preintervention group were receiving preventive medication, compared to 61% in the postintervention group ($p < 0.001$). The use of preventive therapy was more common in women in both groups of patients. In the base-

line group, 39% of women were receiving preventive therapy compared with 18% of men ($p < 0.05$), while the matching figures in the postintervention group were 64% and 44% ($p < 0.05$).

PBS and RPBS data were obtained (Table 3). The prescribing of osteoporosis preventive agents relative to prednisolone was very similar in the 2 regions during the baseline period ($z = 0.34$, $p = 0.7$). At followup, however, the prescribing of osteoporosis preventive agents relative to prednisolone was significantly higher in the South/intervention region ($z = 2.17$, $p < 0.05$). Prescription of osteoporosis preventive agents relative to prednisolone increased in both regions over the course of the study, but particularly in the intervention region (control: $z = 1.87$, $p = 0.06$ and intervention: $z = 4.42$, $p < 0.0001$). The largest relative increases occurred with calcium (increased by 22% in the intervention region, with no change in the control region) and raloxifene (increased by 38% in the intervention and 6% in the control region). The use of bisphosphonates (alendronate, risedronate, and etidronate) increased markedly in both regions

Table 2. Use of osteoporosis preventive therapy in hospital patients before and after intervention.

Preventive Therapy*	Baseline, %		Postintervention, %	
	Admission, n = 113	Discharge, n = 108	Admission, n = 120	Discharge, n = 111
Bisphosphonates	6	11	24	27
Alendronate	4	6	13	11
Risedronate	0	0	1	1
Etidronate	0	0	2	2
Pamidronate	3	5	5	7
Zoledronate	0	0	3	5
Calcitriol	17	19	15	15
Calcium	5	12	19	22
Hormone replacement therapy	5	8	14	14
Vitamin D	3	6	11	17
Raloxifene	2	2	1	1
Anabolic steroids	1	2	2	2

* Some patients were receiving more than one preventive medication.

Table 3. Dispensing of osteoporosis preventive therapy and prednisolone under the PBS and RPBS in Tasmania for March-August 2001 and 2002. The intervention region was Southern Tasmania; the control region comprised the north part of the state.

Period	Region	Osteoporosis Preventive Agents*	Prednisolone	Ratio Osteoporosis Preventive Agents: Prednisolone
		DDD (*000)		
March-August 2001	Intervention	231	380	0.61
	Control	141	243	0.58
March-August 2002	Intervention	396	402	0.99
	Control	186	245	0.76

* Alendronate (Defined daily dose, DDD 10 mg), risedronate (DDD 5 mg), etidronate (DDD 0.4 mg), raloxifene (DDD 60 mg), calcium carbonate (DDD 3 g), calcitriol (DDD 1 µg), prednisolone (DDD 10 mg).

(by 119% in the intervention and 123% in the control region).

DISCUSSION

In this project, written educational material and the technique of academic detailing were employed to promote the use of osteoporosis preventive therapy in patients taking oral corticosteroids. Success of the education program was indicated by a statistically significant increase in prescription of osteoporosis preventive drugs, relative to prednisolone, in the intervention region compared with the control region. In addition, there was a significant increase in the use of osteoporosis preventive drugs in 2 well-matched series of patients admitted to hospital and taking longterm oral corticosteroids.

It was apparent that there was a trend to an increase in the use of osteoporosis preventive drugs relative to prednisolone in the control region of the state over the course of the study. This result was not unexpected. The issue of osteoporosis has received considerable government attention and coverage in professional journals and the media over the past 2 years. There has also been very active

promotion of the bisphosphonates by the pharmaceutical industry to doctors, although currently there is no direct-to-consumer advertising in Australia for prescription medications. Finally, contamination of the 2 groups of prescribers via professional contact is possible, particularly on an island like Tasmania. Nevertheless, our program was able to achieve significant changes in prescriber behavior despite this background change. An improvement in prescribing practices within the control region has been reported by others and ourselves in academic detailing studies²³⁻²⁶.

It was evident that the use of bisphosphonates, calcium, and vitamin D increased significantly following the program. Current recommendations suggest that bisphosphonates are first-line therapy in the treatment and prevention of corticosteroid induced osteoporosis^{9,11,21}. The increase in use of calcium and vitamin D was pleasing, as this was one of the key messages of the program, and these agents were not being actively promoted by the pharmaceutical industry and they have consistently been shown to be underutilized in studies investigating the use of preventive therapy in patients prescribed corticosteroids^{14,18}.

This program employed key strategies for successful

implementation of evidence-based guidelines, as suggested by Gibson²⁷: the use of guidelines that are well supported by evidence (e.g., from National Osteoporosis Society UK, Osteoporosis Australia, and the ACR); local adaptation and consensus, with guidelines being reviewed by local experts; wide dissemination (with the academic detailing program targeting both general practitioners and pharmacists); and multifaceted intervention (e.g., with the academic detailing program supported by the use of the refrigerator magnet to target patients). The sources of greatest practical importance when attempting to modify prescribing practices are those involving the transfer of information through the medium of personal contact^{28,29}. While academic detailing has been shown to be an effective method to modify prescribing behavior, especially when combined with other approaches^{30,31}, this is the first project to our knowledge that has shown that academic detailing can increase the use of osteoporosis preventive therapy in patients prescribed longterm oral corticosteroids.

Investigators have suggested that broad educational effort directed to physicians of varied specialties is needed to ensure that osteoporosis prevention becomes the standard of care for patients receiving longterm corticosteroid treatment¹⁹. However, it is not only physicians who have contact with patients who take longterm corticosteroids. Hart and Green concluded¹⁸ that osteoporosis prophylaxis during corticosteroid treatment should be promoted by local hospital guidelines, hospital and community pharmacists, audit, and general practitioners. Our study was not able to distinguish between the relative contributions of the educational efforts to doctors and pharmacists in producing the observed outcomes, but the results support the belief that it is imperative to involve other health professionals, such as pharmacists, from the beginning when disseminating clinical guidelines and attempting to change practice³². Pharmacists are ideally placed to offer advice to patients, as they have high levels of patient contact and are a well respected and highly regarded source of information.

We targeted general practitioners, rather than specialists, as most doctors in Australia are family general practitioners and they would prescribe most of the corticosteroid therapy, especially on a continuing basis. These doctors see patients more frequently than specialists and are in a position to implement preventive therapy more readily than specialists.

This comprehensive multifaceted educational intervention had a significant influence on prescription of agents that reduce bone loss and subsequent risk of osteoporosis in patients prescribed longterm corticosteroids. However, there clearly is room for improvement. For instance, according to our hospital based data, almost one-half of patients taking longterm oral corticosteroids were still not receiving preventive therapy against osteoporosis following the program. The reasons for this are probably complex, including patient, physician, and health care system related

barriers. Proven pharmacological treatments, such as the bisphosphonates and raloxifene, have only been available recently. Because of their relatively high cost, the use of bisphosphonates at a subsidized rate in Australia is restricted to patients who have sustained a low trauma fracture, even if they have documented osteoporosis, while raloxifene has not been approved for the treatment or prevention of corticosteroid induced osteoporosis. Hence, many patients at high risk of low trauma fracture (e.g., a T score < -2.5) cannot access these medications, despite good evidence for their cost-effectiveness^{10,33}, unless the patients pay the full cost of the therapy.

There are limitations to our study. It is uncertain if the effects from the intervention will be of longterm utility and it would be useful to collect repeat followup data after a longer time. We relied on a before versus after (historical controls) data collection procedure for the hospital patients. It is also unknown whether the hospital patients who were not receiving preventive therapy were considered for such possible therapy by their doctor, or indeed, whether discussions occurred with the patient or if the patient refused preventive therapy. Our results also do not indicate whether patients were receiving any of the medications for osteoporosis prevention or other indication (e.g., hormone replacement therapy for menopausal symptoms, bisphosphonate for Paget's disease), although the use of a control region in the analysis of the prescription data would diminish this possibility. To minimize any bias from the PBS and RPBS data, we used ratio indicators, as these have been suggested to be more robust, and may describe more valid prescribing measures than prescribing rates³⁴.

In the future, corticosteroids may be available that suppress immune function without causing bone loss³⁵. Until then, continuing promotion of corticosteroid induced osteoporosis is imperative for patients receiving these medications if there is to be an effect on reducing the burden of osteoporotic fractures.

A multifaceted education program, incorporating academic detailing of general practitioners and community pharmacists, increased the use of effective osteoporosis prevention strategies in longterm oral corticosteroid users.

REFERENCES

1. Anon. The burden of brittle bones: costing osteoporosis in Australia. Canberra: Access Economics Pty Limited; 2001:1-36.
2. Sambrook PN, Seeman E, Phillips SR, et al. Preventing osteoporosis: Outcomes of the Australian Fracture Prevention Summit — Cosponsored by Osteoporosis Australia and the National Prescribing Service. *Med J Aust* 2002;176:S1-S16.
3. Sambrook P, Birmingham J, Kempler S, et al. Corticosteroid effects on proximal femur bone loss. *J Bone Miner Res* 1990;5:1211-6.
4. van Staa TP, Leufkens HG, Abenhaim L, Zhang B, Cooper C. Oral corticosteroids and fracture risk: relationship to daily and cumulative doses. *Rheumatology Oxford* 2000;39:1383-9.
5. Naganathan V, Jones G, Nash P, Nicholson G, Eisman J, Sambrook PN. Vertebral fracture risk with long-term corticosteroid

- therapy — Prevalence and relation to age, bone density, and corticosteroid use. *Arch Intern Med* 2000;160:2917-22.
6. Lips P. Prevention of corticosteroid induced osteoporosis. *BMJ* 1999;318:1366-7.
 7. van Staa TP, Leufkens HG, Cooper C. The epidemiology of corticosteroid-induced osteoporosis: a meta-analysis. *Osteoporos Int* 2002;13:777-87.
 8. Gennari C, Martini G, Nuti R. Secondary osteoporosis. *Aging-Clin Exp Res* 1998;10:214-24.
 9. American College of Rheumatology Ad Hoc Committee on Glucocorticosteroid-induced Osteoporosis. Recommendations for the prevention and treatment of glucocorticosteroid-induced osteoporosis. *Arthritis Rheum* 2001;44:1496-503.
 10. Saag K, Pisu M. Balancing bones and bucks among new glucocorticoid users. *J Rheumatol* 2003;30:1-3.
 11. Eastell R, Reid DM, Compston J, et al. A UK Consensus Group on management of glucocorticoid-induced osteoporosis: an update. *J Intern Med* 1998;244:271-92.
 12. Peat ID, Healy S, Reid DM, Ralston SH. Steroid induced osteoporosis: an opportunity for prevention? *Ann Rheum Dis* 1995;54:66-8.
 13. Aagaard EM, Lin P, Modin GW, Lane NE. Prevention of glucocorticoid-induced osteoporosis: provider practice at an urban county hospital. *Am J Med* 1999;107:456-60.
 14. Hougardy DMC, Peterson GM, Bleasel MD, Randall CTC. Is enough attention being given to the adverse effects of corticosteroid therapy? *J Clin Pharm Ther* 2000;25:227-34.
 15. Smith MD, Cheah SP, Taylor K, Ahern MJ. Prevention of corticosteroid induced osteoporosis in inpatients recently discharged from a tertiary teaching hospital. *J Rheumatol* 2001;28:566-70.
 16. Walsh LJ, Wong CA, Pringle M, Tattersfield AE. Use of oral corticosteroids in the community and the prevention of secondary osteoporosis: a cross sectional study. *BMJ* 1996;313:344-6.
 17. Yood RA, Harrold LR, Fish L, et al. Prevention of glucocorticoid-induced osteoporosis — Experience in a managed care setting. *Arch Intern Med* 2001;161:1322-7.
 18. Hart SR, Green B. Osteoporosis prophylaxis during corticosteroid treatment: failure to prescribe. *Postgrad Med J* 2002;78:242-3.
 19. Buckley LM, Marquez M, Feezor R, Ruffin DM, Benson LL. Prevention of corticosteroid-induced osteoporosis — Results of a patient survey. *Arthritis Rheum* 1999;42:1736-9.
 20. Gudbjornsson B, Juliusson UI, Gudjonsson FV. Prevalence of long term steroid treatment and the frequency of decision making to prevent steroid induced osteoporosis in daily clinical practice. *Ann Rheum Dis* 2002;61:32-6.
 21. Sambrook P, Diamond T, Fiatarone-Singh M, et al. Corticosteroid induced osteoporosis. *Aust Fam Physician* 2001;30:793-6.
 22. WHO Collaborating Centre for Drug Statistics Methodology. Anatomical therapeutic chemical classification index including defined daily doses for plain substances. Oslo: WHO; 2003.
 23. Landgren FT, Harvey KJ, Mashford ML, Moulds RFW, Guthrie B, Hemming M. Changing antibiotic prescribing by educational marketing. *Med J Aust* 1988;149:595-9.
 24. Peterson GM, Sugden JE. Educational program to improve the dosage prescribing of allopurinol. *Med J Aust* 1995;162:74-7.
 25. Peterson GM, Stanton LA, Bergin JK, Chapman GA. Improving the prescribing of antibiotics for urinary tract infection. *J Clin Pharm Ther* 1997;22:147-53.
 26. Peterson GM, Bergin JK, Nelson BJ, Stanton LA. Improving drug use in rheumatic disorders. *J Clin Pharm Ther* 1996;21:215-20.
 27. Gibson P. Implementing evidence-based guidelines. *Med J Aust* 2001;174:377-8.
 28. McGettigan P, Golden J, Fryer JL, Chan R, Feely J. Prescribers prefer people: The sources of information used by doctors for prescribing suggest that the medium is more important than the message. *Br J Clin Pharmacol* 2001;51:184-9.
 29. Prosser H, Almond S, Walley T. Influences on GPs' decision to prescribe new drugs — the importance of who says what. *Fam Pract* 2003;20:61-8.
 30. Gross PA, Pujat D. Implementing practice guidelines for appropriate antimicrobial usage: a systematic review. *Med Care* 2001;39:55-69.
 31. Thomson O'Brien MA, Oxman AD, Davis DA, Haynes RB. Educational outreach visits: effects on professional practice and health care outcomes (Cochrane Review). In: *The Cochrane Library*. Vol. Issue 1, 2003. Oxford: Update Software; 2003.
 32. Phillips PA, Rubin GL, Morey PS. Evidence for evidence-based medicine at the coalface. *Med J Aust* 2000;172:259-60.
 33. Buckley LM, Hillner BE. A cost effectiveness analysis of calcium and vitamin D supplementation, etidronate, and alendronate in the prevention of vertebral fractures in women treated with glucocorticoids. *J Rheumatol* 2003;30:132-8.
 34. Robertson J, Fryer J, O'Connell D, Smith A, Henry D. Limitations of Health Insurance Commission data for deriving prescribing indicators. *Med J Aust* 2002;176:419-24.
 35. Riggs BL, Hartmann LC. Selective estrogen-receptor modulators — mechanisms of action and application to clinical practice. *N Engl J Med* 2003;348:618-29.
 36. Nowson CA, Margerison C. Vitamin D intake and vitamin D status of Australians. *Med J Aust* 2002;177:149-52.