PAPERS AND SHORT REPORTS

Prevention of rickets in Asian children: assessment of the Glasgow campaign

M G DUNNIGAN, B M GLEKIN, J B HENDERSON, W B McINTOSH, D SUMNER, G R SUTHERLAND

Abstract

In March 1979 the Greater Glasgow Health Board launched a campaign to reduce the high prevalence of rickets in Asian children in the city. A precampaign survey had shown that voluntary low dose vitamin D supplementation would reduce the prevalence of rickets in Asian children. A survey carried out two and three years after the launch of the official campaign also showed a reduction in the prevalence of rickets in children taking low dose supplements equivalent to about 2·5 μg (100 IU) vitamin D daily. There was a considerable reduction in the total prevalence of rickets in this survey compared with the precampaign survey. Hospital discharges of Asian children with rickets declined rapidly after the start of the campaign.

Introduction

Vitamin D deficiency leading to infantile rickets, late rickets in schoolchildren, and osteomalacia in women has been described in the British Asian community on many occasions.[1-4] Prophylactic measures have been lacking or poorly organised. In March 1979 the Greater Glasgow Health Board launched a campaign against rickets in Asian children based on the issue of free vitamin D supplements on demand to children up to 18 years of age. The organisation of the campaign has been described.[5] This report assesses its success in reducing the prevalence of rickets in Asian children in the city.

Departments of Medicine, Biochemistry, and Radiology, Stobhill General Hospital, Glasgow G21

M G DUNNIGAN, MD, FRCP, consultant physician
JANET B HENDERSON, SRD, research dietitian
W B McINTOSH, FIMLS, senior chief medical laboratory scientific officer
G R SUTHERLAND, FRCP, FRCR, consultant radiologist

Department of Clinical Physics and Bioengineering, West of Scotland Health Boards, Glasgow G4

D SUMNER, BSC, DPHIL, principal physicist

Woodside Health Centre, Glasgow G20

B M GLEKIN, MB, MRCGP, general practitioner

Correspondence to: Dr M G Dunnigan, Division of General Medicine, Unit B, Stobhill General Hospital, Glasgow G21 3UW.

Subjects and methods

Precampaign Survey[5]—In 1978 health visitors and school nurses promoted the use of vitamin D supplementation by Asian schoolchildren living in the area served by one health board clinic. Between January and May 1979 children aged 5-17 years on the age and sex register of a general practice in the clinic area were surveyed. A venous blood sample was taken from 189 children (all but four of the children on the register). Children with biochemical evidence of rickets were recalled for *x* ray examination of the knees. Six children with severe and four with mild symptoms of rickets were followed up until *x* ray appearances and biochemical findings had returned to normal. No prophylactic advice about diet, outdoor exposure, or use of supplements was given to the remaining 179 children, since it was intended to resurvey a proportion in the postcampaign survey.

Postcampaign survey—Asian children aged 8-16 years drawn from three general practices in the city were sampled between December and May 1981 (two practices) and December and May 1982 (one practice). These comprised the practice used in the precampaign survey and two previously unsampled practices in a different area of the city. A random sample of families with one or more children in the desired age range was obtained from the age and sex registers of the three practices. A venous blood sample was taken from 255 children; only nine children asked to attend for sampling defaulted. As in the precampaign survey, children with biochemical evidence of rickets were recalled for *x* ray examination of both knees. In both precampaign and postcampaign surveys children were asked about their past and present frequency of consumption of vitamin D supplements. With the exception of older adolescent children the child's statement was checked by interviewing the mother at the initial interview or at a subsequent home visit. Children's stated consumption of vitamin D supplements at the time of survey was classified as "regular" if a supplement was said to be taken at least twice weekly, "intermittent" if taken less frequently, or "none."

Biochemical methods—Serum was separated within half an hour of venepuncture. Serum alkaline phosphatase activity and serum calcium, inorganic phosphorus, and 25-hydroxyvitamin D (25-OHD)

concentrations were measured as described.[5] Based on our further laboratory experience and the results of two recent studies using the same method[6 7] serum alkaline phosphatase activities above 400 IU/l were regarded as definitely abnormal (adult reference range 40-115 IU/l). Activities of 300-399 IU/l were regarded as marginally raised rather than definitely abnormal as previously.[5]

Radiological assessment—X ray films taken in the precampaign and postcampaign studies were reviewed separately on four occasions by a single radiologist (GRS) without knowledge of the biochemical findings and classified as normal, showing borderline or marginal widening of the epiphysial plates ("marginal" rickets), or showing definite abnormal widening and irregularity of the epiphysial plates ("rachitic").

Statistical methods—Differences in the proportions of abnormal biochemical values and radiographs among supplement treatment groups were analysed by the χ^2 method. Differences in group means of biochemical values were analysed by Student's t test. The distributions of serum 25-OHD and alkaline phosphatase values were consistent with a log normal distribution (confirmed by the linearity of plots of normal scores). The data for these values were analysed after logarithmic transformation. Stepwise discriminant analysis was employed to assess the relations between x ray category and serum calcium, inorganic phosphorus, and 25-OHD concentrations, alkaline phosphatase activity, and age in the 56 children examined by radiography in the precampaign survey. The discriminant function derived from this analysis was evaluated prospectively in the 46 children so examined in the postcampaign survey in 1981. Full details of this study have been published elsewhere.[8]

Hospital discharges of children with rickets—A record of children with Asian names discharged from all Glasgow hospitals with the diagnosis of nutritional rickets during 1979-83 inclusive was obtained from the Information Services Division of the Greater Glasgow Health Board. The case record of each child was scrutinised to confirm the diagnosis. Numbers of Asian children discharged from Glasgow hospitals between 1968 and 1978 have been published.[5] A record of children with Asian names discharged with the diagnosis of nutritional rickets from Scottish hospitals outside Glasgow during 1970-81 inclusive was obtained from the Information Services Department of the Scottish Home and Health Department.

Results

RELATIONS BETWEEN RADIOLOGICAL AND BIOCHEMICAL EVIDENCE OF RICKETS

Stepwise discriminant analysis showed that serum alkaline phosphatase activity was the most important single biochemical variable in the prediction of x ray evidence of rickets. Serum 25-OHD, calcium, and inorganic phosphorus concentrations provided poor discrimination. Serum alkaline phosphatase activities above 400 IU/l were therefore used as the best biochemical indicator of the prevalence of rachitic bone disease in the precampaign and postcampaign surveys.[8]

HOSPITAL DISCHARGES OF CHILDREN WITH RICKETS

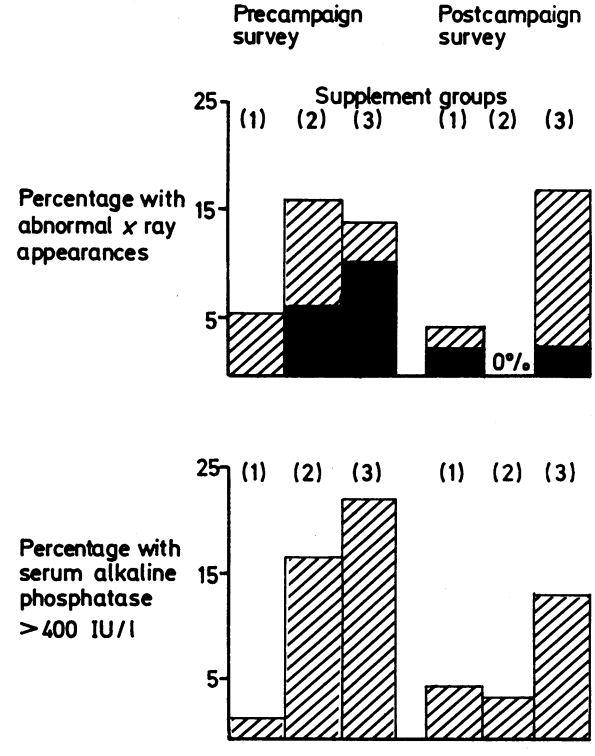
In the five years after the start of the official campaign in 1979 the incidence of children with rickets being discharged from Glasgow hospitals declined steadily; only one child was discharged in 1983 (table I). In the five years before the official campaign (1974-8) 10 Glasgow Asian children required osteotomy for severe rachitic deformity; five children required osteotomy in the five year period 1969-73. In 1979 and 1980 two children required osteotomy for deformity acquired before the start of the official campaign. No children with severe deformity were admitted in the next three years.

The 1981 Census showed that roughly half of Scotland's 16 000 Asians born in the Indian subcontinent lived outside the Glasgow area. In the nine years 1970-8, 111 Asian children aged 0-16 years were discharged from Glasgow hospitals with nutritional rickets compared with only 19 Asian children aged 0-16 years from all other Scottish hospitals. In the first three years of the official campaign (1979-81) 12 Asian children were discharged from Glasgow hospitals with rickets and four from other Scottish hospitals.

TABLE I—*Numbers of Asian children discharged from all Glasgow hospitals with nutritional rickets between 1978 and 1983. (Each case record examined)*

Age (years)	Year					
	1978	1979	1980	1981	1982	1983
0-4	3	0	1	1	0	0
5-16	8	5	2	3	3	1
Incidence (age 5-16)*	2·7	1·7	0·7	1·0	1·0	0·3

*Incidence expressed as number of discharges/1000 Asian schoolchildren aged 5-16 (figures supplied by education authority).



Effect of low dose vitamin D supplementation on percentage of children with abnormal x ray appearances and serum alkaline phosphatase activities in precampaign and postcampaign surveys. Supplement groups=(1) regular; (2) intermittent; (3) none. ▨="Marginal" rickets. ■=Rachitic.

TABLE II—*Precampaign survey of 181 Asian children aged 5-17 years in 1979 classified by stated supplement consumption. Mean serum 25-OHD, calcium, and inorganic phosphorus concentrations and alkaline phosphatase activities*

Supplement consumption group	No (%) of children	25-OHD (nmol/l)		Calcium (mmol/l)		Phosphorus (mmol/l)		Alkaline phosphatase (IU/l)	
		Mean*	95% CI	Mean	95% CI	Mean	95% CI	Mean*	95% CI
(1) Regular	54 (30)	21·5	4·3-105·0	2·41	2·25-2·57	1·55	1·05-1·95	194	174-216
(2) Intermittent	50 (28)	18·3	4·3-77·5	2·39	2·19-2·59	1·43	0·95-1·91	224	189-265
(3) None	77 (43)	17·0	4·0-72·5	2·34	2·01-2·68	1·38	0·86-1·90	250	219-285
p Value (unpaired t test)		1-3	<0·05†	1-3	<0·003	1-3	<0·001	1-3	<0·004

CI = Confidence interval.
*Data log transformed.
†One tailed test.
Conversion: SI to traditional units—25-OHD: 1 nmol/l ≈ 0·4 ng/ml. Calcium: 1 mmol/l ≈ 4·0 mg/100 ml. Phosphorus: 1 mmol/l ≈ 3·1 mg/100 ml.

TABLE III—*Postcampaign survey of 255 Asian children aged 8-16 years in 1981-2 classified by stated supplement consumption. Mean serum 25-OHD, calcium, and inorganic phosphorus concentrations and alkaline phosphatase activities*

Supplement consumption group	No (%) of children	25-OHD (nmol/l)		Calcium (mmol/l)		Phosphorus (mmol/l)		Alkaline phosphatase (IU/l)	
		Mean*	95% CI	Mean	95% CI	Mean	95% CI	Mean*	95% CI
(1) Regular	149 (58)	19·0	6·0-62·5	2·41	2·18-2·64	1·43	1·11-1·75	214	198-232
(2) Intermittent	30 (12)	20·5	5·5-77·5	2·39	2·17-2·61	1·38	1·08-1·68	216	178-261
(3) None	76 (30)	19·5	6·0-65·0	2·40	2·22-2·58	1·44	1·06-1·82	248	219-281
p Value (unpaired *t* test)		NS		NS		NS		1-3	<0·04

CI = Confidence interval.
NS = Not significant.
*Data log transformed.
Conversion: SI to traditional units—25-OHD: 1 nmol/l ≈ 0·4 ng/ml. Calcium: 1 mmol/l ≈ 4·0 mg/100 ml. Phosphorus: 1 mmol/l ≈ 3·1 mg/100 ml.

TABLE IV—*Paired study of 59 Asian children sampled in precampaign and postcampaign surveys, all supplement consumption groups combined. Mean serum 25-OHD calcium, and inorganic phosphorus concentrations and alkaline phosphatase activities*

Survey	25-OHD (nmol/l)		Calcium (mmol/l)		Phosphorus (mmol/l)		Alkaline phosphatase (IU/l)	
	Mean*	95% CI	Mean	95% CI	Mean	95% CI	Mean*	95% CI
Precampaign (1979)	15·5	4·5-55·0	2·37	2·07-2·67	1·50	1·08-1·92	268	125-573
Postcampaign (1981)	21·0	6·8-67·5	2·43	1·11-3·09	1·40	1·08-1·72	208	90-483
p Value (paired *t* test)	<0·007		<0·01		<0·001		<0·002	

CI = Confidence interval.
*Data log transformed.
Conversion: SI to traditional units—25-OHD: 1 nmol/l ≈ 0·4 ng/ml. Calcium: 1 mmol/l ≈ 4·0 mg/100 ml. Phosphorus: 1 mmol/l ≈ 3·1 mg/100 ml.

PRECAMPAIGN SURVEY

In the unsupplemented group six children were found to have severe and four mild clinical symptoms of rickets without deformity. They were treated with 75 μg (3000 IU) vitamin D daily until biochemical and radiological healing had occurred. No child in the "regular" or "intermittent" supplement group had definite rachitic symptoms.

Values of serum calcium, inorganic phosphorus, alkaline phosphatase, and 25-OHD were available from 181 of the 189 children sampled. Seventeen children (22%) had abnormal serum alkaline phosphatase activities in the unsupplemented group compared with 1 (1·8%) in the "regular" group (χ^2 test: $p < 0.05$) (figure). Eleven children (14%) had abnormal *x* ray appearances in the unsupplemented group compared with 3 (5·5%) in the "regular" group. This difference was not significant. Eight children in the unsupplemented group (10%) had radiological appearances in the more severe "rachitic" category compared with none in the "regular" group (χ^2: $p < 0.05$; figure). No child in the unsupplemented group had received vitamin D supplements in the previous five years.

Mean serum calcium, inorganic phosphorus, and 25-OHD concentrations were significantly higher and alkaline phosphatase activities significantly lower in children taking regular supplements compared with unsupplemented children (table II). Children taking supplements intermittently had mean values of those variables intermediate between the "regular" and unsupplemented groups.

POSTCAMPAIGN SURVEY

No child with biochemical and *x* ray evidence of rickets in the postcampaign survey had definite rachitic symptoms or deformity. Values of serum calcium, inorganic phosphorus, alkaline phosphatase, and 25-OHD were available from all 255 children sampled. Ten children (13%) had abnormal serum alkaline phosphatase activities in the unsupplemented group compared with seven children (4·6%) in the "regular" group (χ^2 test: $p < 0.05$; figure). Thirteen children (17%) had abnormal *x* ray appearances in the unsupplemented group compared with 6 (4·0%) in the "regular" group (χ^2: $p < 0.01$; figure). This reduction occurred in the milder, "marginal" rickets category. The prevalence of biochemical and *x* ray evidence of rickets among the small number of "intermittent" supplement takers was low and similar to that in the "regular" group (figure).

Mean serum alkaline phosphatase activities were significantly lower in "regular" supplement takers than in unsupplemented children but there were no significant differences in mean concentrations of serum calcium, inorganic phosphorus, or 25-OHD among the supplement treatment groups (table III).

The prevalence of rickets was less in unsupplemented children in the postcampaign survey than in the precampaign survey. Of the 76 children classed as unsupplemented in the postcampaign survey, 49 (64%) had taken supplements for some weeks or months at the launch of the official campaign and discontinued them. This prior supplementation with vitamin D seems likely to have reduced the prevalence of biochemical and *x* ray abnormalities in this group.

When all three groups were combined the prevalence of abnormal serum alkaline phosphatase activities fell from 24 (15%) in the precampaign survey to 18 (7%) in the postcampaign survey (χ^2: $p < 0.05$). The prevalence of abnormal *x* ray appearances fell from 22 (12%) in the precampaign survey to 19 (7·5%) in the postcampaign survey (NS) and of the more severe "rachitic" category from 11 (6%) to 5 (2%) (χ^2: $p < 0.05$).

PAIRED STUDY

Fifty nine children from the precampaign survey were randomly reselected for the postcampaign survey in 1981. The 10 children with clinical symptoms of rickets who had required treatment were excluded from the reselection. Mean serum calcium and serum 25-OHD concentrations rose significantly and mean serum inorganic phosphorus concentration and alkaline phosphatase activity fell significantly between sampling (table IV).

CONSUMPTION OF SUPPLEMENT

A total of 3230 Asian children attended Glasgow schools in September 1983. The volume of vitamin D supplements dispensed mainly to Asian schoolchildren through clinics, health centres, and selected schools from the central pharmacy of the Greater Glasgow Health Board rose from an average of 2200 10 μg (400 IU) doses daily in the first year of the campaign (March 1979 to February 1980) to 3300 doses daily in 1984 (April to September). By contrast, only 400 10 μg doses of vitamin D had been dispensed daily in the year preceding the official campaign (1978).[5] This rise was reflected in the increased proportion of children taking supplements between the precampaign and postcampaign surveys (tables II and III).

Discussion

There was a gradual long term decline in the incidence of hospital admissions of Asian children with rickets in England

between 1962 and 1978.[9] In Glasgow any such decline had not eliminated vitamin D deficiency before 1979.[5] Two children required osteotomy for severe rachitic deformity in the year before the start of the official campaign. The 88 schoolchildren (15 of whom required osteotomy) aged 5-16 years discharged with severe rickets from Glasgow hospitals between 1970 and 1978 were drawn from an average school population of only 2470 over this period. The incidence of hospital discharges of children with rickets has declined steadily since the start of the official campaign (table I); no child has been admitted with rachitic deformity acquired since it began.

As with previous generations of white children, Glasgow Asian children suffered more than their counterparts in the rest of Scotland from severe rickets, presumably because of reduced exposure to ultraviolet light due to unfavourable housing in central Glasgow. This imbalance was corrected in the three years after the start of the official campaign.

Evidence that severe vitamin D deficiency remained prevalent in Glasgow Asian children immediately before the start of the official campaign was also provided by the unsupplemented children in the precampaign survey. These provided a baseline against which the success of both voluntary vitamin D supplementation and of the subsequent official campaign could be measured. One in five unsupplemented children showed biochemical evidence of rickets and one in seven *x* ray evidence of rickets (figure). Ten had clinical, biochemical, and *x* ray evidence of rickets. The survey also showed that a well organised local initiative based on voluntary vitamin D supplementation would reduce this high prevalence of *x* ray and biochemical evidence of rickets despite only a small rise in mean serum 25-OHD concentrations (figure; table II).

The postcampaign survey measured the success of the official campaign two and three years after its inception. Unsupplemented children showed almost three times the incidence of biochemical evidence of rickets and four times the incidence of *x* ray evidence of rickets as "regular" supplement takers (figure). The proportion of "regular" supplement takers was higher and the prevalence of *x* ray and biochemical evidence of rickets in the sample as a whole lower than in the precampaign survey.

In the precampaign survey (table I) mean serum 25-OHD concentrations rose slightly but significantly in "regular" supplement takers compared with unsupplemented children. There were no significant differences in mean serum 25-OHD concentrations between supplement treatment groups in the postcampaign survey, probably because of prior but discontinued vitamin D supplementation by many unsupplemented children. The paired study provided a more sensitive measure of the effect of low dose vitamin D supplementation in the postcampaign survey (table IV). The significant rise in the mean serum 25-OHD concentration of 5·5 nmol/l (2·2 ng/ml) in the paired study compares with a fourfold greater mean rise of 21·8 nmol/l (8·7 ng/ml) produced by the supervised administration of a 75 μg capsule of vitamin D weekly (roughly 10 μg (400 IU) daily) to Asian adults and children for six months.[5] This suggests that most of the "regular" supplement takers in both the precampaign and postcampaign surveys consumed on average not more than 10 μg vitamin D twice weekly, equivalent to an additional daily intake of the order of 2·5 μg (100 IU). Direct questioning of the children and their mothers confirmed this interpretation.

A supplement of 10 μg vitamin D daily will correct the low serum 25-OHD concentrations which are endemic in the British Asian population.[5] The median dietary vitamin D intake of Glasgow Asian schoolchildren who completed a seven day weighed dietary survey in 1980 was 1·6 μg (64 IU) daily.[10] The precampaign and postcampaign surveys suggest that suboptimal vitamin D supplementation added about 2·5 μg vitamin D to this intake and greatly reduced the prevalence of clinical rickets and rachitic bone disease (as judged by biochemical and *x* ray criteria) despite only small improvements in group vitamin D state. Nevertheless, rickets and osteomalacia (but not hypovitaminosis D) in the British Asian population, and probably in the elderly at risk, would be prevented more efficiently by increasing the national average vitamin D intake from its present level of about 2·5 μg (100 IU) to about 5·0 μg daily by appropriate additional fortification of food. Such average intakes are found in Norway, Sweden, Holland, and West Germany and are about one third of the average intakes in the United States (13·7 μg (548 IU); J Ablett, personal communication).

The lack of objective assessment of the success of prophylactic measures against Asian rickets in Britain has been criticised.[11] The Stop Rickets Campaign, funded by the Department of Health and Social Security, disseminated information on the prevention of rickets to Asian communities in England. It did not coordinate or implement detailed preventive measures and its report does not include an assessment of its success in reducing the incidence of rickets in the areas visited.[12] Goel *et al* found a reduced prevalence of rickets in Glasgow Asian children examined in the first year of the board's official campaign in 1979 compared with a previous survey carried out in 1974 and attributed this to increased consumption of vitamin D supplements.[13] The prevalence of rickets was not related to individual supplement consumption in either sample and the fall in prevalence between surveys might also be explained by adaptation to a more Western diet and lifestyle.

The Glasgow campaign against Asian rickets has not abolished vitamin D deficiency in Glasgow Asian children. Nevertheless, the campaign has shown that suboptimal vitamin D supplementation will greatly reduce the prevalence of severe rickets in Asian children. In the absence of a modest increase in national vitamin D intake through food fortification a well organised programme of supplementation offers the best means of reducing the prevalence of rickets and osteomalacia in the vulnerable Asian and elderly populations.[14] In Glasgow the campaign against rickets has been extended to Asian women who continue to show evidence of vitamin D deficiency leading to osteomalacia. Vitamin D supplements are available to Asian women at cost price in health board clinics and health centres. The success of this extension to the campaign is being monitored.

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References

1 Dunnigan MG, Paton JPJ, Haase S, McNicol GM, Gardner MD, Smith CM. Late rickets and osteomalacia in the Pakistani community in Glasgow. *Scott Med J* 1962;**7**:159-67.
2 Ford JA, Colhoun EM, McIntosh WB, Dunnigan MG. Rickets and osteomalacia in the Glasgow Pakistani community, 1961-71. *Br Med J* 1972;ii:677-80.
3 Ford JA, Colhoun EM, McIntosh WB, *et al*. Clinical and sub-clinical vitamin D deficiency in Bradford children. *Arch Dis Child* 1976;**51**:838-43.
4 Holmes AM, Enoch BA, Taylor JL, Jones ME. Occult rickets and osteomalacia amongst the Asian immigrant population. *Q J Med* 1973;**42**:125-49.
5 Dunnigan MG, McIntosh WB, Sutherland GR, *et al*. Policy for prevention of Asian rickets in Britain: a preliminary assessment of the Glasgow rickets campaign. *Br Med J* 1981;**282**:357-60.
6 Schiele F, Henny J, Hitz J, *et al*. Total bone and liver alkaline phosphatases in plasma: biological variations and reference limits. *Clin Chem* 1983;**29**:634-41.
7 Gomez P, Coca C, Vargas C, *et al*. Normal reference intervals for 10 biochemical variables in healthy infants, children and adolescents. *Clin Chem* 1984;**30**:407-12.
8 Abdul-Motaal AA, Gettinby G, Dunnigan MG, *et al*. Relationships between radiological and biochemical evidence of rickets in Asian schoolchildren. *Postgrad Med J* 1985;**61**:307-12.
9 Department of Health and Social Security. Rickets and osteomalacia. Report of the Working Party on Fortification of Food with Vitamin D, Committee on Medical Aspects of Food Policy. *Rep Health Soc Subj (Lond)* 1980;No 19.
10 Robertson I, Glekin BM, Henderson JB, *et al*. Nutritional deficiencies among ethnic minorities in the United Kingdom. *Proc Nutr Soc* 1982;**41**:243-56.
11 Peach H. A review of aetiological and intervention studies on rickets and osteomalacia in the United Kingdom. *Community Med* 1984;**6**:119-26.
12 Save the Children Fund. *Stop Rickets Campaign*. London: Save the Children Fund, 1983.
13 Goel KM, Sweet EM, Campbell S, *et al*. Reduced prevalence of rickets in Asian children in Glasgow. *Lancet* 1976;ii:405-7.
14 Dunnigan MG, Fraser SA, McIntosh WB, *et al*. The prevention of vitamin D deficiency in the elderly. *Scott Med J* (in press).

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