

## Original Article

# Do Current Regimes of Hormone Replacement Therapy Protect Against Subsequent Fractures?

T. D. Spector<sup>1</sup>, P. Brennan<sup>2</sup>, P. A. Harris<sup>1</sup>, J. W. W. Studd<sup>3</sup> and A. J. Silman<sup>2</sup>

<sup>1</sup>Departments of Environmental and Preventive Medicine and Rheumatology, St. Bartholomew's Hospital Medical College, London; <sup>2</sup>ARC Epidemiology Unit, Manchester; and <sup>3</sup>Dulwich Menopause Clinic, London, UK

**Abstract.** It is now accepted that unopposed oestrogen therapy reduces osteoporotic fractures by about 50%. Although current regimes with added progestogens are thought to act similarly to unopposed oestrogens, no study has yet demonstrated an effect on fractures with the former. Using a retrospective cohort design we studied fracture rates in women attending a menopause clinic for hormone replacement therapy (HRT) and compared them with women derived from the general population. Data were analysed from 1075 women exposed to HRT and 1741 non-exposed postmenopausal women. In all 226 fractures were reported between 1977 and 1986, the commonest site being the distal radius, occurring in 28 of the HRT women and in 37 of the non-exposed women. The incidence density rate for fracture of the distal radius is 3.5/1000 woman-years (wy) in non-exposed women. This was similar to the rate in the HRT women *prior* to HRT use, the rate falling by 30% after exposure from 3.2 to 2.2/1000 wy. The protective effect on osteoporotic fractures increased progressively with duration of use. After 5 years of use the relative risk fell to 0.5 (95% confidence interval, 0.2–1.2) for all osteoporotic fractures and for the distal radius to 0.18 (95% confidence interval, 0.05–1.3). No similar changes were seen for non-osteoporotic fractures. There were 6 (0.6/1000 wy) reported fractures of the hip in the non-exposed group compared with none in the HRT group (when 1.7 were expected based on non-exposed rates) ( $p=0.15$ ). Although based on observational data, this study suggests that modern

HRT regimes are effective in preventing distal radius fractures and potentially other osteoporotic fractures.

**Keywords:** Bone density; Fractures; Hormone replacement; Oestrogens; Progestogens

## Introduction

Osteoporosis is a major public health problem. It is estimated that the cumulative lifetime risks of a 50-year-old white woman sustaining a distal radius fracture is 15%, a vertebral fracture 32% and a hip fracture 16%, the last being associated with considerable morbidity and mortality [1]. The number of osteoporotic fractures has increased steadily in most countries over the last 30 years [2], and although incidence rates may be levelling off in some populations [3] the number of fracture cases will continue to rise due to the increased numbers of elderly in the population.

Unopposed oestrogen therapy has been shown to reduce subsequent hip fractures by about 50%. These estimates are mainly based on retrospective case-control studies which employed doses of oestrogens generally higher than currently prescribed and without concomitant administration of progestogens [4–7]. Data are lacking on the protective effect of either low-dose oral or implanted oestrogens on fractures. In most countries these are currently prescribed in combination with progestogens. The objective of the present study was to examine whether women treated with newer preparations of oestrogens (orally or by implant) were protected against osteoporosis-related fractures within the first 10 years of treatment. In particular the effect on

distal radius (Colles') fractures was examined, this being the commonest site for an osteoporosis-related fracture in this age group.

## Patients and Methods

A retrospective cohort design was adopted based on the identification of two groups of women. An HRT, or exposed, group consisted of 1703 consecutive attenders at a menopause clinic in Dulwich, South London, from January 1976 to 31 December 1986. Postal questionnaires established that 155 had died or moved. A total of 1225 (79%) of the remaining 1548 responded, of whom 1075 were postmenopausal and had taken HRT for more than 1 month. These women constituted the exposed group for this analysis. The characteristics of these women have been described in detail elsewhere but comprised a high proportion of social class I and II women from all over the UK [8]. The average duration of therapy was 51 months, with the majority (64.7%) having received a subcutaneous oestrogen implant (50–100 mg estradiol), usually 6-monthly, often in combination with testosterone 100 mg (79%). If the uterus was intact (65%) cyclical progestogens were given for 12 days of each cycle. Hysterectomized women were given unopposed oestrogens. Oral oestrogens were taken by 35% and a number received both oral and implants at some time. Accurate recording of the time of menopause was not generally available.

The control, or non-exposed, group was identified from the age–sex registers of four large general practices in Greater London. Postal questionnaires were sent to 5021 women aged 35–60. Of these 494 had moved or died and 3251 (72%) of the remaining 4527 responded. Women who reported still having periods were excluded from subsequent analysis. The 1741 who were postmenopausal and had not taken HRT for more than 1 month constituted the non-exposed group.

Women in both groups were sent an identical postal questionnaire requesting information on all fractures occurring during the period January 1977 to December 1986, including their site, predisposing circumstances and date. All self-reported fractures were checked against the individual's general practitioner records, which were often more precise concerning the site or date of fractures. Fractures resulting from road-traffic accidents were excluded from analysis. In addition, as a check for completeness of ascertainment the general practitioner medical records of a randomly selected series of 200 controls were examined and no missed cases of fracture were detected.

## Analysis

Fractures were divided into categories depending on site and defined as being osteoporotic or non-osteoporotic according to the sites related to low bone density reported by Seeley et al. [9]. Fractures sites classified as

osteoporotic included humerus, clavicle, hand, wrist, pelvis, rib, leg, hip and toe. Incidence density rates were calculated for each fracture site expressed as a rate per 1000 woman-years (wy) of observation either on HRT or postmenopausally. For the exposed cohort, rates were calculated separately for the periods before and whilst on HRT. The period prior to HRT was generally that before referral to the clinic. The number of woman-years of observation in ex-HRT users was small (349 wy) and they were not included in the analysis. Once a woman sustained a fracture she was no longer considered 'at risk'. Ninety-six women (5%) of the non-exposed group had taken HRT, and rates were calculated up to this time point. Incidence rates in the exposed and non-exposed groups were compared by calculating risk ratios. Differences in the cohorts at baseline were examined by standard chi-squared and *t*-tests. The role of potential confounders was assessed by Cox's proportional hazards model using SAS software [10,11]. To examine for the presence of a dose–duration effect Kaplan–Meir survival analysis was used. In addition HRT users were divided into tertiles of duration of use and the relative risks calculated using non-exposed controls for an equivalent period after the menopause. The duration effect was examined using an exact two-sided chi-squared test for trend using EGRET software (SERC, Seattle).

## Results

The response rates adjusted for deaths and incorrect addresses are given in Table 1. Demographic and reproductive variables between the groups are compared in Table 2. As might be expected due to the earlier and often artificial menopause, the exposed group were younger (51.8 years vs. 56.0) with a higher rate of prior hysterectomy (38.6%) than controls (21.8%). They also had higher usage rates of oral contraceptive pills and included a higher proportion of parous women.

The number of fractures reported to have occurred between 1977 and 1986 was 101 in the HRT group, with 55 fractures occurring prior to HRT and 46 subse-

**Table 1.** Response rates

	Exposed group	Non-exposed group
Letters sent	1703	5021
No. died/moved	155	494
No. receiving letter	1548	4527
No. completed questionnaire	1225	3251
Response rate for those receiving questionnaire	79.4%	71.8%
No. eligible for analysis <sup>a</sup>	1075	1741

<sup>a</sup> See text.

**Table 2.** Characteristics of women in the exposed and non-exposed groups

	Exposed (n=1075)	Non-exposed (n=1741)
Age (SD)	51.8 (6.6)*	56.0 (6.9)
Nulliparity (%)	108 (10.0)*	279 (16.0)
Ever OCP <sup>a</sup> users (%)	317 (29.5)*	312 (17.9)
Hysterectomy (%)	415 (38.6)*	380 (21.8)

<sup>a</sup> OCP, oral contraceptive pill.

\* $p < 0.001$ .

quently. There were 125 fractures in the non-exposed women. The frequency of fracture at specific sites is given in Table 3. The commonest site of fracture reported was the distal radius, there being 28 in the HRT group and 37 in the controls. Women with this fracture were older (53.2 vs. 49.5 years,  $p < 0.01$ ), but no other significant differences were noted. Hip fracture was reported in one HRT woman (prior to treatment) and 6 controls. No hip fractures were seen in HRT women on therapy, although 1.7 cases were expected based on rates in non-exposed women ( $p = 0.15$ ).

Before HRT use and referral to the clinic, 944 women in the HRT group were observed for 5929 wy within the study period (excluding women on HRT before 1977), producing an incidence rate of 9.27/1000 wy for all fractures, 5.40/1000 wy for all osteoporotic fractures,

and 3.20/1000 wy for distal radius fractures. The rates were similar to those for the non-exposed group, who had 10 530 wy of observation with incidence rates of 11.8/1000 wy for all fractures, 7.31/1000 wy for osteoporotic fractures and 3.5/1000 wy for distal radius fractures.

The rates for fractures after HRT use were based on 1021 women exposed for a total of 4035 years. The overall incidence rate for women who were currently on HRT was 11.38/1000 wy for all sites and 2.23/1000 wy for the distal radius. This produced an overall relative risk of distal radius fractures in HRT users of 0.70 (95% confidence interval, 0.32–1.55) compared with baseline pretreatment rates and 0.63 (0.31–1.31) compared with rates in the non-exposed women. Relative risks for all osteoporotic fracture were 0.96 (0.55–1.68) and 0.71 (0.43–1.16) respectively. To estimate the possible effects of confounding by age, parity, use of the oral contraceptive pill and hysterectomy, Cox's proportional hazards model was used. For the analysis 1500 women from the non-exposed and 890 from the exposed group were used for whom full data was available and who first used HRT after 1977 (to reduce the effects of 'left censoring'). No significant alterations in the beta coefficients for the effect of HRT was noted when the potential confounding variables were entered into the model either singly or in combination. Parity after adjustment for the other variables had a modest protective effect on wrist fractures (relative risk = 0.84; 95% confidence interval, 0.68–1.05). Adjusting for

**Table 3.** Frequency and sites of fracture in the exposed and non-exposed groups

Fracture site	Exposed		Non-exposed n (rate/1000 wy)
	Pre-HRT n (rate/1000 wy)	Post-HRT n (rate/1000 wy)	
Wrist <sup>a</sup>	19 (3.20)	9 (2.23)	37 (3.51)
Spine <sup>a</sup>	1 (0.17)	2 (0.50)	1 (0.10)
Hip <sup>a</sup>	1 (0.17)	0 —	6 (0.57)
Ankle	7 (1.18)	2 (0.50)	10 (0.90)
Foot	5 (0.84)	6 (1.49)	14 (1.30)
Rib <sup>a</sup>	5 (0.84)	2 (0.50)	6 (0.57)
Finger/hand <sup>a</sup> /thumb	4 (0.67)	4 (0.99)	9 (0.85)
Tibia/fibula <sup>a</sup>	3 (0.51)	1 (0.25)	5 (0.47)
Toe <sup>a</sup>	3 (0.51)	7 (1.42)	11 (1.04)
Skull	0 —	2 (0.50)	1 (0.10)
Shoulder/sternum/clavicle <sup>a</sup>	0 —	2 (0.50)	0 —
Pelvis <sup>a</sup>	0 —	1 (0.25)	2 (0.20)
Humerus <sup>a</sup>	1 (0.17)	1 (0.25)	6 (0.57)
Knee	1 (0.17)	0 —	4 (0.38)
Elbow	2 (0.34)	4 (0.99)	8 (0.76)
Coccyx	1 (0.17)	1 (0.25)	1 (0.10)
Multiple	2 (0.34)	2 (0.50)	4 (0.38)
Woman-years of observation (wy)	5929	4034	10 530
<i>Totals</i>			
Osteoporotic	37 (6.24)	27 (6.69)	83 (7.87)
Non-osteoporotic	18 (3.03)	19 (4.71)	42 (3.98)
All fractures	55 (9.27)	46 (11.38)	125 (11.88)

<sup>a</sup> Classified as osteoporotic according to data from [9].

parity did not, however, alter the crude estimate of relative risk.

To establish the effect of different durations of HRT use, rates of fracture after different durations of use were compared with those in non-exposed women at equivalent times after the menopause. The greatest difference in survival curves was seen for wrist fractures (Fig. 1). There was an increasingly protective effect with duration which was not very strong due to the similarity in the curves in the first 36 months ( $p = 0.1$ ). Relative risks were calculated by dividing subjects into tertiles of exposure to HRT or postmenopausal years (Fig. 2). Three types of fracture were compared: wrist, 'osteoporotic' and non-osteoporotic. The relative risks decreased for all osteoporotic and wrist fractures (chi-squared test for trend  $p = 0.06$ ,  $p = 0.03$  respectively), relative risks after 5 years of treatment being 0.18 (0.01–1.3) and 0.5 (0.2–1.2). Some of the protective effect on osteoporotic fractures was lost when wrist fractures were excluded from the osteoporotic fracture group, although the relative risk dropped from 0.98 (0.36–2.66) at baseline to 0.70 (0.23–2.10) after 5 years.

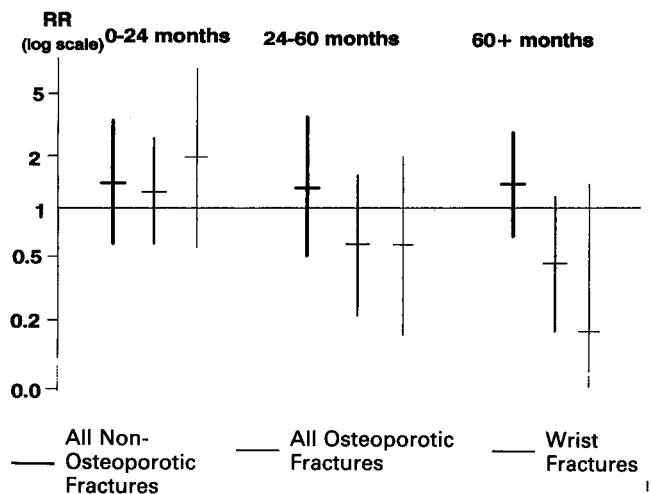


Fig. 1. Survival curves for wrist fracture, comparing the onset of HRT with time since menopause, up to 1987.

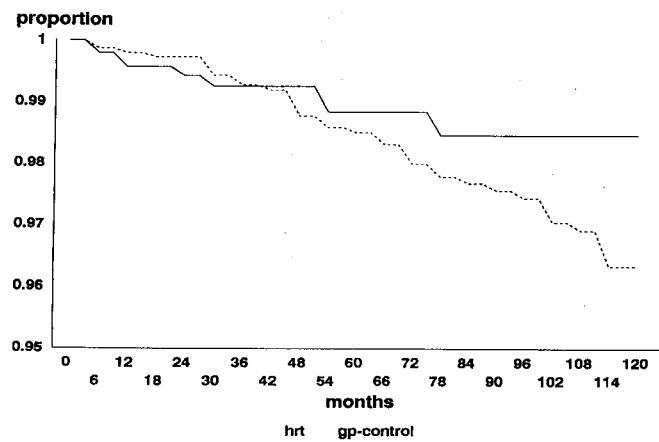


Fig. 2. Relative risks and 95% confidence intervals relating to fractures while exposed to HRT.

No significant change was noted for 'non-osteoporotic' fractures over the same period. The highest duration tertile was examined and the characteristics of the exposed and non-exposed women compared. Differences in age, use of oral contraceptive pills, hysterectomy and parity were noted; these were similar to those found between the groups overall.

With the combination of oestrogens used and changes between routes of administration it was difficult to sort out the effects of individual preparations. Where possible further subdivision of the fracture cases on the basis of different HRT regimes (i.e. opposed vs. unopposed or implants vs. oral) was performed. This did not show any obvious differences, although analysis was limited by the relatively small numbers of fracture cases.

### Discussion

These results suggest that current regimes using implants and progestogens are protective against distal radius fractures. A real effect was supported by evidence of an increasing duration–response effect. A similar but weaker effect was also seen when 'non-osteoporotic' fractures were combined.

A number of potential biases and problems need to be addressed. First the validity of using self-reported fractures requires estimating. All self-reports were checked with the general practitioner records to look for inconsistencies or overreporting. Although inconsistencies between records and self-reporting occurred with regard to exact site and date, no falsely reported cases were found. The survey method was also found to be sufficiently sensitive, as no cases were missed in the 200 medical records sampled. Similar fracture questionnaires have been validated in other studies [12]. We therefore believe the data to be generally reliable. Although women attending the menopause clinic were generally of higher social class than those in the non-exposed group, fracture rates have not been demonstrated to vary markedly by social class in the UK. Moreover, no differences in rates appeared between general practices with predominantly social class II patients and those with predominantly classes III–V. As the two groups of women were likely to differ in a number of other ways, differences in reporting or recall might have occurred. However the two groups received identical questionnaires and were not informed of the hypothesis being tested. Moreover reported fracture rates in both groups prior to HRT were similar. Justification of the accuracy of the fracture data comes from the similarity of the distal radius fracture rate in the non-exposed women (3.5/1000 wy) to those found in other population surveys, allowing for geographical differences and changes in secular trends. In the Oxford–Dundee hospital record study a rate of 2.4/1000 was found in women aged 45 [13], in Newcastle 3.2/1000 for women aged 45–49 [14], in Malmo 1.9/1000 for women aged 40–49 [15], and in Rochester, Minnesota 3.8/1000 for those aged 45–54 [16]. More recently a postal

questionnaire in Oxford found a rate in women aged 45–54 of 2.1/1000 [17], while a population-based study using a fracture register in Leicester detected a rate of 1.9/1000 in women of the same age group [18].

As in most studies, baseline differences existed between exposed and non-exposed women, particularly in age and timing of menopause; confounding was therefore a potential problem. However no significant difference in results was seen when using non-exposed or pre-HRT women for comparison. Furthermore adjustment for potential confounders which included duration of HRT, using the proportional hazards model, did not alter the risk ratios. Prior to the study, power calculations predicted that the sample size would be adequate to detect a 50% difference in distal radius fractures at the 5% level of significance. Unfortunately the numbers of woman-years after HRT use in the cohort were overestimated, and thus despite over 40 000 woman-years of observation the study lacked sufficient power to answer the original objective unequivocally.

The categorization of fractures into 'osteoporotic' and 'non-osteoporotic' is somewhat arbitrary. Hip, vertebral and distal forearm fractures are traditionally regarded as osteoporotic as their incidence increases with age and rates are greater in women. The aetiology of fractures at other sites is probably also related to low bone mass although less strongly than at the distal radius [19]. This has also been demonstrated in a recent study of the elderly [9].

The precise relationship of distal radius fractures to subsequent vertebral and hip fractures remains unclear. Distal radius fractures are the commonest fractures in the postmenopausal period and generally precede hip fractures by 15 years. Studies have shown relative risks which vary between showing no increase to up to an 8-fold increased risk of subsequent hip fracture [20–22]. The association in several studies appears greater when the distal radius fracture is sustained before the age of 60. Although in the present study numbers of hip fractures were insufficient for detailed analysis, we would have expected nearly 2 fractures in the HRT group whereas none was reported, suggesting a similar protective effect. Although caution is needed in extrapolating these data, the current results suggest that modern therapeutic regimes of HRT are also likely to be protective against hip fracture even after short duration of use.

The contribution of the menopause to distal radius fractures is unclear and it is generally believed that these fractures are less closely associated with low bone density than are fractures of the spine and hip. Falling is believed to be a particularly important cause of fractures especially around the menopause, and this might obscure the beneficial effect of HRT [16]. The increased numbers of all fractures observed in the first 24 months after HRT was unexpected. Most of these occurred in the first 12 months of treatment. It is unlikely that this was a direct result of hormonal therapy. One explanation might be an early increase in physical activity in HRT users due to possible psychological and physiolo-

gical benefits. This increased activity might place women at a greater risk of fracture due to falls and minor trauma before any protective effect on bones is seen.

In conclusion, despite the problems of self-selection and interpretation of observational data [23], these data suggest that modern HRT regimes reduce rates of subsequent distal radius fractures and that this protective effect increases with duration of therapy. This implies that the benefits in terms of fractures may be seen relatively early, and modern doses and regimes appear to be as effective as older preparations using higher doses of oestrogens. The data are reassuring as they show that oestrogen-progestogen combinations also appear to protect against 'osteoporotic' fractures – which is the main rationale for their use in prophylaxis. The major public health concern is that of hip fracture, and further follow-up of these women would be needed to ascertain the precise protective benefit. It is becoming obvious, however, that to reduce the numbers of hip fractures significantly HRT may be necessary for at least 20 years and long-term compliance will be crucial [24]. Factors affecting compliance (such as different therapeutic approaches) will require more research in the future.

*Acknowledgements.* This study was funded by a joint grant from Research in Ageing and the Oliver Bird Fund. We would like to thank the staff and patients of the Dulwich Menopause Clinic, Handsworth Avenue Health Centre, Steeles Lane Health Centre, Wapping Health Centre and Parkhurst Road Surgery for their cooperation and help. In addition we are grateful to Lynn Wells, Pat Harris and Roy Ide for clerical and computing assistance and to Dr Malcolm Law for advice and comments on the manuscript.

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*Received for publication 9 August 1991  
Accepted in revised form 19 February 1992*

## Correspondence

### *Acronyms in Bone Densitometry*

SIR,—In response to our publication [1], Wilson and colleagues [2] proposed the adoption of a uniform terminology and corresponding abbreviation for dual X-ray absorptiometry (DXA). We supported this suggestion [3]. Since then, the number of papers on DXA has increased considerably. However, different abbreviations (DEXA, DER, DRA, QDR and DPX) for this technique for bone densitometry are still used, several of which are proprietary in origin. DXA is the upgraded version of DPA (dual photon absorptiometry). The nuclear source has been replaced by an X-ray source and this improved technology has gained widespread acceptance and distribution. Recently, similar developments in SPA (single photon absorptiometry) have occurred, which will add yet another abbreviation to densitometry terminology: SXA for single X-ray absorptiometry, not the alternative 'SEXA'.

Users and manufacturers of these techniques have now joined efforts in an International Standards Committee [4]. In this committee, standards of calibration, measurement units and terminology are discussed. There is general agreement that standard abbreviations for bone densitometry techniques are necessary and the term DXA has been accepted.

Therefore, we would again like to emphasize the use of the acronym DXA for dual X-ray absorptiometry and add the acronym SXA for single X-ray absorptiometry. When we confine ourselves to SPA, SXA, DPA and DXA – next to QCT for quantitative computed tomography – both researchers and clinicians will understand what we mean.

Good acronyms for ultrasound and velocity measurements and for magnetic resonance measurements, both for quantifying bone mineral density and structure, have yet to be agreed upon. Acronyms such as QUS for quantitative ultrasound and QMR for quantitative magnetic resonance would be consistent with QCT. Such terminology, however, would clearly need consensus development.

H. K. Genant  
C. C. Glüer  
K. G. Faulkner  
S. Majumdar  
S. T. Harris  
K. Engelke  
C. van Kuijk

*Osteoporosis Research Group,  
Department of Radiology,  
University of California San Francisco*

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