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Thickness of the subchondral mineralised tissue zone (SMZ) in normal male and female and pathological human patellae

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ABSTRACT

The objective of this paper was to analyse sex differences of the thickness of the subchondral mineralised tissue zone (SMZ), and to find out whether systematic changes of SMZ thickness are associated with naturally occurring, non-full-thickness cartilage lesions of human patellae. In 32 methyl-methacrylate-embedded specimens (16 normal, 8 with focal medial, and 8 with lateral lesions) the SMZ thickness was determined, using a binocular macroscope and an image analysing system. In each case, the thickness distribution was reconstructed throughout the entire joint surface. The maximal and mean SMZ thicknesses were significantly higher in males than in females ($P < 0.01$). In normal patellae and those with lateral lesions, the thickness was significantly thicker laterally than medially ($P < 0.05$), but it was not in specimens with medial damage. Patellae with medial damage exhibited a significantly lower total mean and lateral mean ($P < 0.05$). A lower SMZ thickness was found directly beneath medial lesions than beneath lateral ones, but the local thickness was always in the range of that observed in normal specimens. We conclude that differences of patellar SMZ thickness exist between males and females. Naturally occurring cartilage lesions appear, however, not to be associated with local changes of SMZ thickness, but they may be associated with an altered regional distribution pattern within the joint surface.

Key words: Subchondral bone; cartilage lesions; femoropatellar joint; chondromalacia patellae.

INTRODUCTION

Articular cartilage and subchondral bone act in concert in transmitting loads through joints, and the integrity of both tissues is required for appropriate functioning. The linkage of the hyaline cartilage and the subchondral bone is provided by a thin layer of calcified cartilage (Redler et al. 1975; Milz & Putz, 1994) which, together with the subchondral bone layer, may be called the subchondral mineralised tissue zone (SMZ, Fig. 1). This zone may be regarded as a morphological and mechanical unit and has been recognised to play an important role in attenuating the axial impact forces typically encountered during dynamic joint loading (Radin & Paul, 1970; Radin et al. 1970; Hoshino & Wallace, 1978; Radin & Rose, 1986).

It has been suggested that (1) subchondral bone adapts to the mechanical demands made upon it during normal and abnormal joint function, thus reflecting the long-term distribution of stress in the joint surfaces (Pauwels, 1963, 1980; Tillmann & Brade, 1980; Müller-Gerbl et al. 1989, 1994) and that (2) subchondral bone plays an important role in the

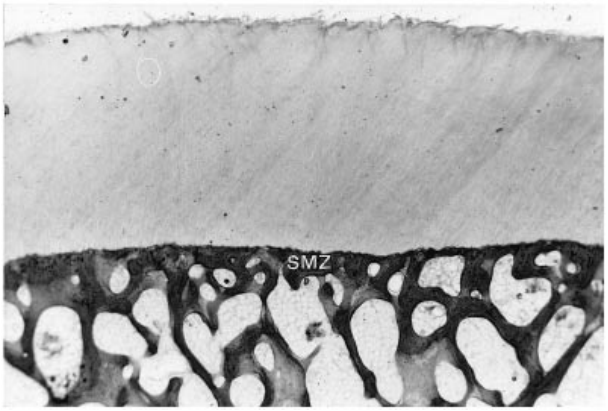

Fig. 1. Histological section of the subchondral mineralised tissue zone (SMZ) covered by uncalcified cartilage (top) and supported by epiphyseal trabeculae (bottom).

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pathogenesis of cartilage degeneration. A thickening of the SMZ has been suggested to increase the internal cartilage stresses by either bringing about a higher stiffness of the subchondral bone or by progressive thinning of the cartilage layer (Radin et al. 1970, 1972, 1978, 1991; Simon & Radin, 1972; Brown et al. 1984; Wu et al. 1990; Shimizu et al. 1993).

Whereas numerous investigations have previously dealt with the radiological density of the subchondral bone in man under physiological and pathological conditions (Pauwels, 1963, 1980; Tillmann & Brade, 1980, Müller-Gerbl et al. 1989, 1994; Eckstein et al. 1992, 1993, 1995), only a few have focused on its actual thickness. Studies on the relationship of cartilage damage and the thickness of the underlying subchondral bone have so far yielded contradictory results. Both an increased and decreased SMZ thickness have been reported in animal models of osteoarthrosis (Simon & Radin, 1972; Benske et al. 1988; Layton et al. 1988; Dedrick et al. 1991, 1993, 1995; Armstrong et al. 1994) and in human joints (Darracott & Vernon-Roberts, 1971; Christensen et al. 1982; Chai et al. 1991; Grynpas et al. 1991; Shimizu et al. 1993). However, in these studies the SMZ thickness was usually measured at certain (defined) points, but not systematically throughout the entire joint surface.

Only recently has the regional distribution of SMZ thickness been reported in human tibiae (Milz & Putz, 1994) as well as in normal patellae and proximal ulnae (Milz et al. 1995, 1997), in which a relatively low correlation with the thickness of the uncalcified cartilage was demonstrated. However, it is open to question whether there exist systematic differences between males and females in general and/or whether the local distribution of SMZ thickness is altered in the case of naturally occurring cartilage lesions. Being the place of earliest and most severe cartilage damage in the body (Ficat & Hungerford, 1977) and showing more progressive cartilage damage at its lateral than at its medial facet (Meachim & Emery, 1974), the patella is ideally suited for such an analysis, and it has even been termed an 'ideal observatory' for cartilage degeneration (Ficat, 1973).

The objective of the current study was therefore to examine the topographical thickness distribution of the subchondral mineralised tissue zone (SMZ) in male and female human patellae, and in specimens with naturally occurring, focal, non-full-thickness cartilage lesions.

The specific questions to be addressed were: (1) Are there systematic differences in SMZ thickness between normal men and women? (2) Do specimens with different sites of focal cartilage lesions (medial or lateral) yield different distribution patterns of SMZ thickness throughout the joint, possibly indicating an alteration of the long-term mechanical loading responsible for these lesions? (3) Are focal, non-full-thickness cartilage lesions associated with locally increased or decreased SMZ thickness at these specific locations?

MATERIALS AND METHODS

32 cadaveric human patellae (aged 54–92 y, mean 78 y; 14 males, 18 females), fixed in a solution of 4% formalin, were chosen from about 200 dissecting-room subjects which consisted of 45% males and 55% females. The criteria by which they were selected were the absence of macroscopically visible cartilage damage or the presence of a circumscribed, focal non-full-thickness cartilage lesion, either in the medial or lateral patellar facet. In 16 specimens (8 male, 8 female) the cartilage was intact and showed no sign of damage on naked eye inspection; 16 cadavers exhibited focal cartilage lesions grade 2 or 3 (classification of Outerbridge, 1961), the damage being located in 8 cases in the medial, and in 8 in the lateral facet of the patella. The lesions had a diameter between 0.2 and 2 cm and comprised 1–2-thirds of the thickness of the cartilage layer; specimens with generalised cartilage damage or exposure of the bone–cartilage interface were discarded from the study. No data were available about the past medical and social history of the individuals from whom the specimens were obtained.

The patellae were dehydrated and embedded in methylmethacrylate (Milz & Putz, 1994; Milz et al. 1995). The ventral part of the specimens was cut open to accelerate the embedding process. Because the articular surface of the patella is mainly curved within the transverse plane, transverse 500 μm sections were obtained at intervals of 3400 μm with a saw microtome (Leitz, Wetzlar, Germany), in order to keep angular distortion of the thickness measurements minimal. Figure 2*a* shows a contact radiograph of a section from a patella without a cartilage lesion. Additional sections, 100 μm thick, were stained with van Gieson (Fig. 2*b*). The thickness of the SMZ was assessed from the 500 μm transverse sections at intervals of 4 mm from the main patellar ridge to the periphery on either side, and additional measurements were made directly beneath the lesions. The distances from the tidemark (the border between the uncalcified and the calcified cartilage) to the transitional line of the SMZ and the subarticular trabecular bone were

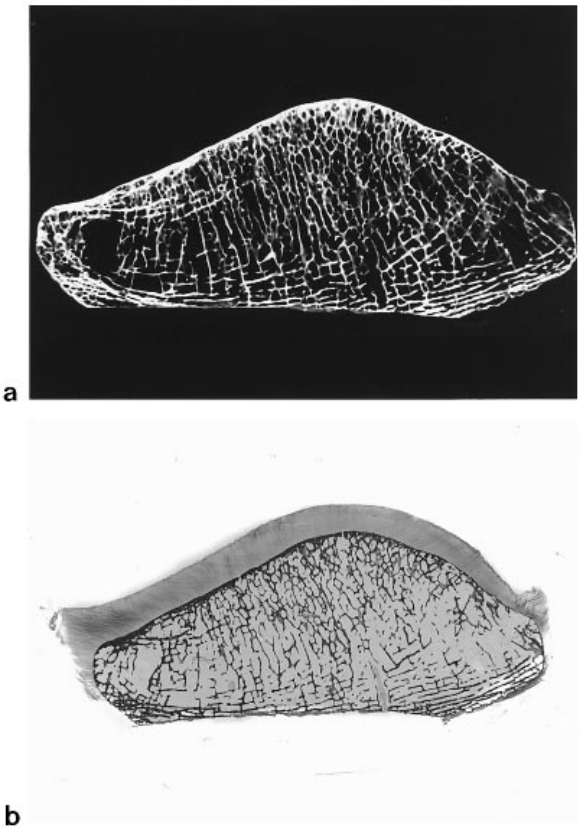

Fig. 2. (*a*) Contact radiograph of a transverse 500 µm MMA-embedded section obtained from a normal patella (the ventral aspect of the patella has been cut to accelerate the methylmethacrylate embedding); (*b*) neighbouring section stained with van Gieson.

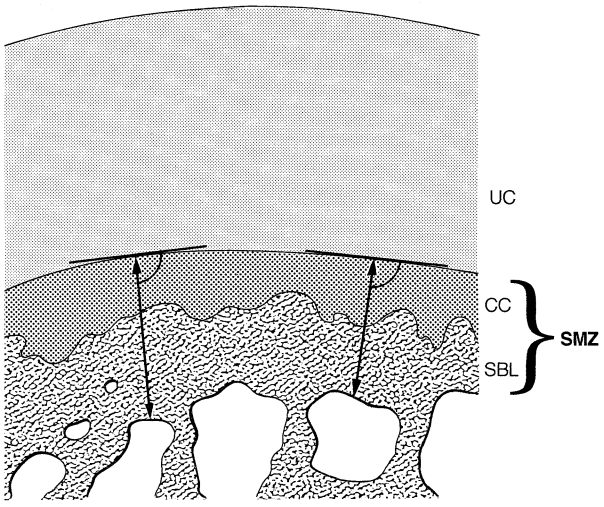

Fig. 3. Schematic drawing of the subchondral mineralised tissue zone (SMZ) and 2 representative measurements (UC, uncalcified cartilage; CC, calcified cartilage; SBL, subchondral bone layer).

measured perpendicular to the bone–cartilage interface at a magnification of $\times 15$ (Fig. 3), using a Wild M 420 binocular macroscope (Wild Leitz, Heerbrugg, Switzerland—numerical aperture 0.25) in connection

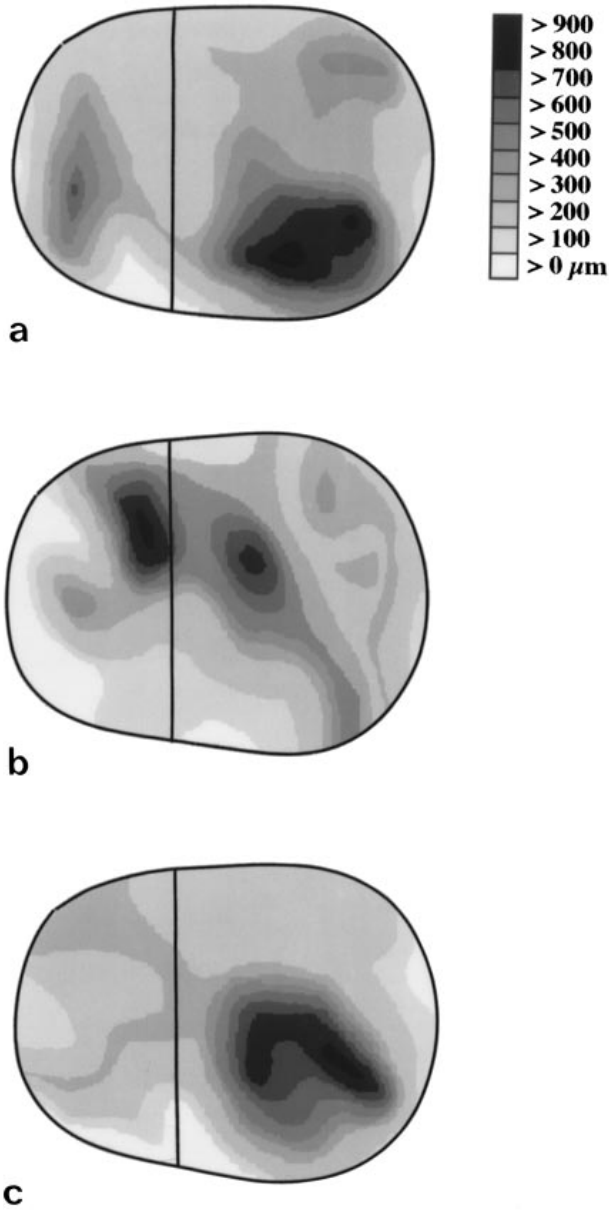

Fig. 4. Regional distribution pattern of SMZ thickness in the patella; reconstruction from the rectangular measurement grid using b-spline interpolation (dorsal view). The medial patellar facet is on the left and the lateral facet on the right, the vertical line indicating the principal patellar ridge space. (*a*) Single specimen with normal undamaged cartilage. (*b*) Single specimen with medial cartilage lesion. (*c*) Single specimen with lateral cartilage lesion.

with an IPS 10 image analysing system (Kontron, Eching, Germany). In a previous study (Milz & Putz, 1994), the precision of the thickness measurements was confirmed to be in the range of 5–10 µm. To obtain a comprehensive visual display, topographical thickness maps were prepared from the measuring points by Gnuplot software (Computer Solutions, Grafing, Germany), with b-spline interpolation and isolines delineating thickness intervals of 100 µm (Fig. 4).

The following parameters were evaluated: (1) the maximal thickness of the SMZ, (2) the position of the maximum and the distribution pattern of SMZ thickness, (3) the total mean thickness of the SMZ (all measuring points), (4) the mean SMZ thickness of the medial and lateral patellar facets, and (5) the local thickness of the SMZ beneath focal (medial or lateral) cartilage lesions. The mean values of the medial and lateral facets were compared statistically using the Wilcoxon test for matched pairs; differences between males and females and between specimens with and without lesions were evaluated with the Mann–Whitney U test.

RESULTS

SMZ thickness in normal male and female specimens

The SMZ was found to exhibit a high degree of variation of thickness within each patella, and there were also important differences between individual specimens. In normal male patellae (Table 1) the maximal thickness varied between 1000 μm and 2110 μm (mean = 1530 ± 380 μm) and in females (Table 2) between 770 and 1390 μm (mean = 1070 ± 260 μm). With the exception of 1 specimen, the maxima were always localised in the lateral patellar facet. From this maximum, the SMZ thickness generally decreased to values of less than 100 μm at the patellar margin (Fig. 4). In some cases, a secondary maximum was observed between the paramedian segment and the odd facet, just beneath the medial patellar ridge (Fig. 4*a*). The distribution pattern did not show systematic variations between males and females. The total mean of SMZ thickness ranged from 430 to 730 μm (mean = 620 ± 120 μm) in normal male specimens (Table 1) and from 350 to 570 μm (mean = 440 ± 70 μm) in the females (Table 2). In both sexes the mean lateral thickness was significantly greater than that of the medial facet ($P < 0.05$). In the male group the maximal thickness ($P < 0.01$), the total mean ($P < 0.01$), the lateral mean ($P < 0.01$) and also the medial mean SMZ thickness ($P < 0.05$) were greater than in females (Fig. 7). Within the male group and within the female group no significant correlation between these parameters and either the age or the body size was found.

Table 1. *Thickness of the subchondral mineralised tissue zone (SMZ) in normal male patellae*

Age (y)	Body size (cm)	Maximum and location of the SMZ thickness (μm)	Mean of the total patella (μm)	Mean of the medial facet (μm)	Mean of the lateral facet (μm)
54	176	1380 LF	480	**360**	600
58	183	1410 LF	640	540	730
64	166	**1000** LF	**430**	390	**480**
68	170	**2110** LF	**770**	630	**890**
69	153	1350 LF	550	460	650
74	167	1510 LF	730	**640**	800
82	185	2100 LF	730	530	880
87	162	1370 LF	610	590	680

LF, lateral facet; MF, medial facet; PR, principal ridge; minima and maxima are marked bold.

Table 2. *Thickness of the subchondral mineralised tissue zone (SMZ) in normal female patellae*

Age (y)	Body size (cm)	Maximum and location of the SMZ thickness (μm)	Mean of the total patella (μm)	Mean of the medial facet (μm)	Mean of the lateral facet (μm)
75	164	1000 LF	360	**280**	430
76	163	1310 LF	**570**	470	**650**
76	167	**1390** PR	470	290	500
81	158	820 LF	390	400	**380**
83	148	780 LF	500	**480**	520
83	153	1140 LF	430	320	510
89	148	**770** LF	440	400	470
91	145	1330 LF	**350**	**280**	410

LF, lateral facet; MF, medial facet; PR, principal ridge; minima and maxima are marked bold.

Table 3. *Thickness of the SMZ in patellae with cartilage lesions at the medial facet*

Age (y)	Body size (cm)	Maximum and location of the SMZ thickness (μm)	Mean of the total patella (μm)	Mean of the medial facet (μm)	Mean of the lateral facet (μm)
62 f	154	1310 LF	510	470	560
70 m	175	**810** MF	**270**	340	**230**
78 f	169	850 MF	360	350	350
81 f	149	1170 LF	350	300	380
85 f	138	1500 PR	480	330	500
86 f	160	**1850** LF	**550**	**590**	**570**
88 f	150	1230 LF	390	310	480
92 f	155	920 PR	290	**230**	320

LF, lateral facet; MF, medial facet; PR, principal ridge; minima and maxima are marked bold.

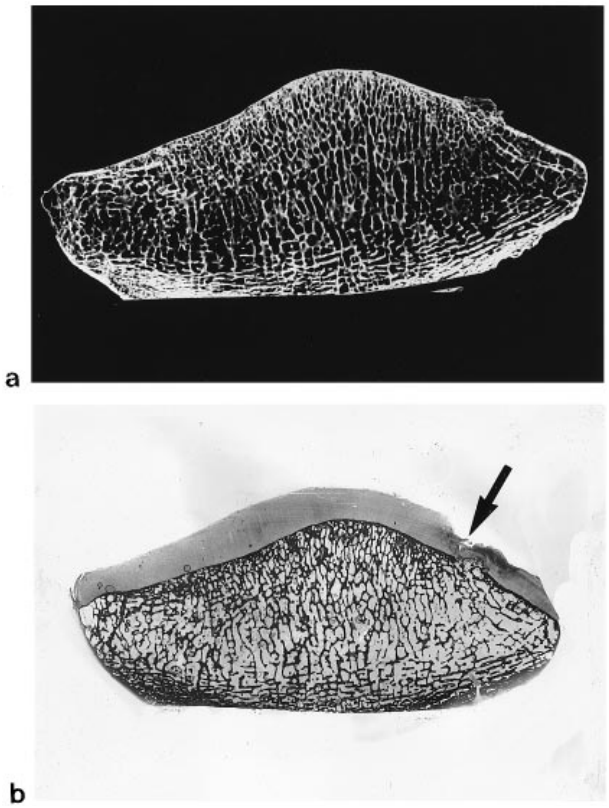

Fig. 5. Transverse sections through a patella with a focal medial cartilage lesion (arrow): (*a*) contact radiograph (500 μm section); (*b*) histological section (100 μm) stained with van Gieson.

SMZ thickness in specimens with medial and lateral cartilage lesions

In patellae with medial lesions (Table 3) maxima of 810–1850 μm (mean = 1210 ± 350 μm) were found, and in those with lateral lesions (Table 4) peak values of 930–2450 μm (mean = 1710 ± 610 μm). In the latter, the maxima were always localised in the lateral facet (Fig. 4*c*), but in the specimens with medial lesions 2 cases were observed in which they were localised at the principal ridge and in 2 at the medial facet (Fig. 4*b*). In the specimens with medial lesions (Table 3, Fig. 5) the mean thickness varied between 270 and 550 μm (mean = 400 ± 100 μm) and in those with lateral lesions (Table 4, Fig. 6) between 310 and 730 μm (mean = 560 ± 160 μm). The mean lateral thickness was significantly greater than that of the medial facet in the patellae with lateral cartilage lesions ($P < 0.05$), but not in the group with medial cartilage damage. The comparison between normal and pathological specimens showed that whereas those with lateral cartilage damage did not differ significantly from normals, those with medial damage exhibited a significantly lower total and lateral SMZ thickness than the normal specimens ($P < 0.05$) (Fig. 8). The group with medial damage did not, however, show a significantly lower SMZ thickness of the medial patellar facet.

The thickness of the SMZ measured directly beneath the medial cartilage lesions ranged from 200 to 600 μm (mean 390 ± 140 μm), that beneath the lateral cartilage lesions from 470 to 940 μm (mean 670 ± 210 μm). In some cases the medial lesions were accompanied by a local advancement of ossification (Fig. 5), but this was not a consistent finding. The SMZ beneath the lateral cartilage lesions was significantly thicker than that beneath the medial lesions ($P < 0.05$), but at both sites the values were well within the range of those measured in normal patellae (Tables 1, 2). The lateral cartilage lesions were not usually found at the sites of maximal SMZ thickness.

DISCUSSION

The questions raised in this study were, whether there exist differences in normal patellar SMZ thickness of males and females, and whether alterations of the local SMZ thickness or of its distribution pattern throughout the joint surface are observed in naturally

Table 4. *Thickness of the SMZ in patellae with cartilage lesions at the lateral facet*

Age (y)	Body size (cm)	Maximum and location of the SMZ thickness (μm)	Mean of the total patella (μm)	Mean of the medial facet (μm)	Mean of the lateral facet (μm)
76 m	170	1010 LF	**310**	**240**	**330**
77 m	175	2270 LF	690	520	990
77 m	177	**2450** LF	**730**	470	**1020**
81 m	166	1760 LF	570	490	650
83 f	155	1100 LF	490	640	490
83 f	155	2180 LF	**730**	**680**	770
90 f	145	**930** LF	370	330	440
92 *m*	163	2000 LF	620	450	760

LF, lateral facet; MF, medial facet; PR, principal ridge; minima and maxima are marked bold.

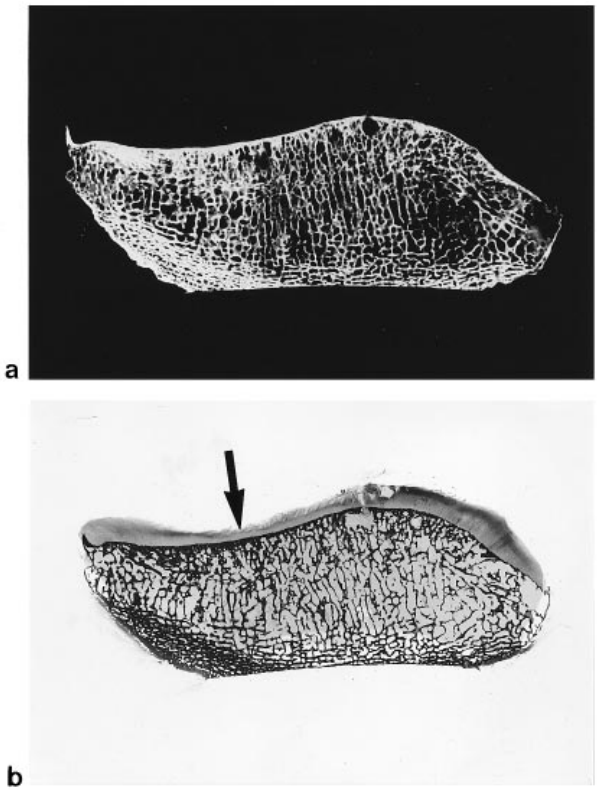

Fig. 6. Transverse sections through a patella with a focal lateral cartilage lesion (arrow): (*a*) contact radiograph (500 μm section); (*b*) histological section (100 μm) stained with van Gieson.

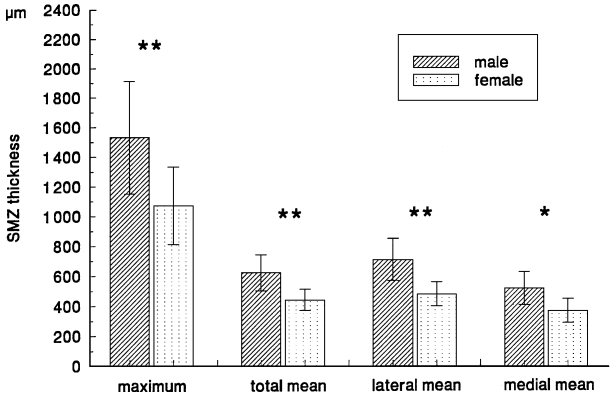

Fig. 7. Patellar SMZ thickness in males and females (* $P < 0.05$; ** $P < 0.01$). The bars show mean values and standard deviations (in μm).

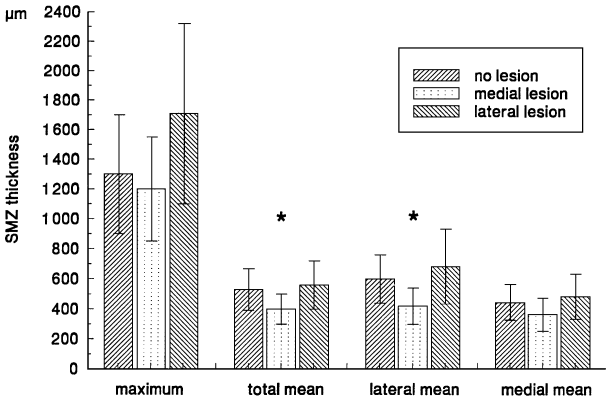

Fig. 8. Patellar SMZ thickness in specimens with normal cartilage (group 1), medial cartilage lesions (group 2) and lateral cartilage lesions (group 3). The bars show mean values and standard deviations (in μm). The level of statistical significance applies to the differences of group 2 with the groups 1 and 3 (* $P < 0.05$; ** $P < 0.01$).

occurring, focal, non-full-thickness cartilage lesions. Since the boundary between the calcified cartilage and the subchondral bone layer is highly irregular, and because the calcified cartilage and the subchondral bone layer may be regarded as a morphological, radiographic, and mechanical unit, both layers were measured together rather than separately.

Comparison with previous morphological and biomechanical studies

Roux (1896) was probably the first to observe variations in the thickness of what he called the 'pressure-receiving plate' ('Druckaufnahmeplatte').

He reported a thickness of 1 mm in patellae of normal subjects, and values around 0.1 mm in an individual who had not used his lower limb for a period of 3 y. Our results show that the patellar SMZ exhibits an inhomogeneous thickness distribution, with maxima of > 2 mm in the lateral facet and values as low as 100 μm at the periphery of the joint. These data complement previous studies in which the trabecular bone underlying the medial patellar facet has been shown to be less dense and to yield a different structural organisation from that of the lateral side (Raux et al. 1975). Even though we find a fairly constant distribution pattern of SMZ thickness, there is considerable variation between individuals with regard to the maximal and mean values. The thickness patterns observed in this study agree in principle with those of the apparent (Pedley & Meachim, 1979) and radiological subchondral bone density (Müller-Gerbl et al. 1989; Eckstein et al. 1992, 1993), and also with those of subchondral bone stiffness (Townsend et al. 1975). They exhibit, however, a greater diversity than the density patterns, probably because in radiological studies the partial volume effect smooths topographic variations of the SMZ.

Pauwels (1963, 1980) suggested that the density of the subchondral bone may be regarded as an 'embodiment' of the stress diagram and his view has been supported by x-ray densitometric (Tillmann & Brade, 1980) and CT osteoabsorptiometric studies (Müller-Gerbl et al. 1989, 1994; Eckstein et al. 1992, 1993, 1995). This hypothesis is based on the idea that bone functionally adapts to the long-term stress encountered during normal daily activity (Wolff, 1892), and this has been confirmed in animal experiments (Goodship et al. 1979; Rubin & Lanyon, 1987), photoelastic models (Kummer, 1966; Pauwels, 1990), and finite element analyses (Huiskes et al. 1987; Beaupre et al. 1990). Variations in subchondral bone density and thickness of the femorotibial joint have been shown to be associated with variations in joint loading (Christensen et al. 1982; Shimizu et al. 1993; Milz & Putz, 1994; Armstrong et al. 1995), particularly in genu valgum and varum (Christensen et al. 1982; Müller-Gerbl et al. 1994). It has also been demonstrated that subchondral density patterns are subject to change in the adult following a high tibial osteotomy (Müller-Gerbl et al. 1994), and that strenuous running exercise can increase the subchondral bone thickness in dogs (Oettmeier et al. 1992). For these reasons it appears tempting to derive the long-term loading conditions of joints from the quantitative distribution of the subchondral bone tissue.

Patellar contact-pressure changes dramatically during knee flexion and extension (Ahmed et al. 1983; Hille et al. 1985; Hehne, 1990), in particular in the medial facet, but the proximal lateral facet has been shown always to remain in contact from 60° to 140° of flexion. The lateral facet thus supports the load over a wider range of motion and is used more frequently during normal activity. Also, in a computer model of passive knee flexion (Heegaard et al. 1995), greater stresses were calculated in the lateral patellar facet. These findings are consistent with higher lateral SMZ thickness and it is important to note that the thickness of the SMZ may account for the unique loading pattern of an individual joint and its specific neuromuscular control mechanisms (which is difficult to determine experimentally), and it may therefore be qualified to assess the long-term 'loading history' of joints during normal activity.

SMZ thickness in male and female specimens

Our study shows that there exist significant differences of SMZ thickness between males and females. Although the ratio between medial and lateral thickness is similar in both sexes, women exhibit a strikingly lower maximal and mean SMZ thickness than men. This contrasts with the findings on patellar subchondral bone density, in which no such differences could be detected (Eckstein et al. 1992). The reasons for the lower SMZ thickness in the women may be their lower body weight, and hence the less severe mechanical loading, but hormonal factors and, in particular, osteoporosis may also play an important role in this issue. Future clinical studies, based on noninvasive imaging techniques, may cast some light on these issues, since they have the advantage that more information about the history of the patient can be obtained.

SMZ thickness in specimens with medial and lateral cartilage lesions

Patellar cartilage lesions have been shown to occur very early in life (Aleman, 1928) and to affect a vast proportion of the elderly population (Mitrovic et al. 1987). Nevertheless, there has been much (as yet unresolved) controversy as to the origin of these lesions. Goodfellow et al. (1976) have attributed cartilage degeneration of the medial 'odd facet' to its 'habitual non-contact' during joint function, and Ficat & Hungerford (1977) lateral cartilage lesions to a so-called 'excessive-pressure syndrome'. In this

context it should be remembered that the thickness of the SMZ may reflect the dominant loading pattern of the joint, but that it does not account for single (infrequent) loading events that may lead to cartilage degeneration.

In the specimens with lateral cartilage lesions we observed some cases of elevated mean lateral thickness, but this group was not significantly different from normal patellae. At least as far as the given age range is concerned, our data therefore do not support the hypothesis that a greater average pressure acts on the lateral facet of these individuals and that lateral cartilage damage is brought about by a long-term alteration of load distribution between both facets. The progression of the cartilage damage could not be evaluated in this study, but the higher tendency of lateral patellar cartilage lesions to progress with time that has been reported in the literature (Meachim & Emery, 1974) may have to do with the higher thickness of the SMZ in the lateral patellar facet (Abernethy et al. 1978).

Our findings indicate that the patellae with medial damage may have been exposed to an other mechanical environment than normals, since their total mean and lateral mean SMZ thickness (but not their mean medial thickness) is significantly less. This result is consistent with previous findings of decreased overall subchondral bone density and thickness in patients with medial chondromalacia patellae (Darracott & Vernon-Roberts, 1971; Eckstein et al. 1993) and indicates that insufficient joint use may be a promoting factor for medial patellar cartilage damage. Animal experiments have shown that osteoarthrotic changes can follow a reduction in intermittent joint loading and that these changes are preceded by a decrease in bone density (Smith et al. 1992). With mechanical disuse the proteoglycans may decrease (Smith et al. 1992; Kiviranta et al. 1994), the cartilage having an impaired ability to withstand normal levels of physiological loading. The significantly lower mean lateral (but not medial) SMZ thickness in this group indicates that the long-term stress distribution between the medial and lateral patellar facet may be different from normal subjects, and the imbalance may result from an unusual neuromuscular innervation pattern of the quadriceps femoris (Weh & Eickhoff, 1983). One may therefore speculate that medial patellar cartilage lesions are promoted by infrequent, high-magnitude stress peaks during deep knee flexion (Hehne, 1990), and these may be particularly critical in mechanical disuse, in which the cartilage is more vulnerable (Kiviranta et al. 1994).

It has previously been suggested that a thickening of the subchondral bone plate may be an important initial step towards cartilage degeneration, before changes occur in the cartilage itself (Radin et al. 1970, 1972, 1978, 1991; Simon & Radin, 1972; Brown et al. 1984; Shimizu et al. 1993). Although we could directly assess neither the mechanical behaviour of the subchondral bone nor the time sequence of events at the beginning of cartilage lesions, our results suggest that focal, non-full-thickness cartilage damage is found in areas of both high and low subchondral bone thickness, and that the values recorded there are in the range of those beneath healthy, undamaged cartilage. In this context, it should also be remembered that the stiffness of subchondral bone (Choi et al. 1990; Mente & Lewis, 1994) is several orders of magnitude higher than that of the uncalcified cartilage and that therefore subtle changes in its mechanical properties will probably not affect the mechanical stresses within the cartilage matrix.

Animal models of osteoarthrosis (Simon & Radin, 1972; Benske et al. 1988; Layton et al. 1988; Dedrick et al. 1991, 1993, 1995; Armstrong et al. 1994) have yielded contradictory results regarding local changes of the subchondral bone thickness in cartilage degeneration, probably because different species, different joints, and different models of osteoarthrosis were used. However, in contrast to previous studies of the human hip (Chai et al. 1991; Grynpas et al. 1991) we can show that naturally occurring, focal, non-full-thickness lesions of the patellar cartilage may appear in areas of high as well as those of low SMZ thickness and are not necessarily associated with systematic thickness changes of subchondral bone.

Conclusions

Our findings suggest that males and females show a similar distribution pattern of SMZ thickness, but that the maximal and mean values are significantly lower in women than in men. In elderly subjects, naturally occurring, focal, non-full-thickness cartilage lesions of the patella appear not to be associated with a systematically higher or lower local SMZ thickness. However, specimens with medial cartilage lesions show some differences in the regional distribution pattern of SMZ thickness, indicating a potential alteration in the long-term mechanical loading. Valuable insights into joint function and pathology may be gathered from an analysis of the SMZ thickness distribution throughout the entire joint, and these should be extended in the future to individuals with a well-documented medical and social history.

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REFERENCES

Abernethy PJ, Townsend PR, Rose RM, Radin EL (1978) Is chondromalacia patellae a separate clinical entity? *Journal of Bone and Joint Surgery* **60B**, 205–210.

Ahmed AM, Burke DL, Yu A (1983) In-vitro measurement of static pressure distribution in synovial joints. Part II. Retropatellar surface. *Journal of Biomechanical Engineering* **105**, 226–236.

Aleman O (1928) Chondromalacia posttraumatica patellae. *Acta Chirurgica Scandinavica* **63**, 149–190.

Armstrong S, Read R, Ghosh P (1994) The effects of intraarticular hyaluronan on cartilage and subchondral bone changes in an ovine model of early osteoarthritis. *Journal of Rheumatology* **21**, 680–688.

Armstrong SJ, Read RA, Price R (1995) Topographical variation within the articular cartilage and subchondral bone of the normal ovine knee joint: a histological approach. *Osteoarthritis and Cartilage* **3**, 25–33.

Beaupre GS, Orr TE, Carter DR (1990) An approach for time-dependent bone modeling and remodeling—application: a preliminary remodeling situation. *Journal of Orthopedic Research* **8**, 662–670.

Benske J, Schunke M, Tillmann B (1988) Subchondral bone formation in arthrosis: polychrome labeling studies in mice. *Acta Orthopaedica Scandinavica* **59**, 536–541.

Brown TD, Radin EL, Martin RB, Burr DB (1984) Finite element studies of some juxtaarticular stress changes due to localized subchondral stiffening. *Journal of Biomechanics* **17**, 11–24.

Chai BF, Tang XM, Li H (1991) Scanning electron microscopic study of subchondral bone tissues in osteoarthritic femoral head. *Chinese Medical Journal* **104**, 503–509.

Choi K, Kuhn JL, Ciarelli MJ, Goldstein SA (1990) The elastic moduli of human subchondral, trabecular and cortical bone tissue and size-dependency of cortical bone modulus. *Journal of Biomechanics* **23**, 1103–1113.

Christensen P, Kjaer J, Melsen F, Nielsen HE, Sneppen O, Vang PS (1982) The subchondral bone of the proximal tibial epiphysis in osteoarthritis of the knee. *Acta Orthopaedica Scandinavica* **53**, 889–895.

Darracott J, Vernon-Roberts B (1971) The bony changes in chondromalacia patellae. *Rheumatology and Physical Medicine* **11**, 175–179.

Dedrick DK, Goulet RW, Huston L, Goldstein SA, Bole GG (1991) Early bone changes in experimental osteoarthritis using microscopic computed tomography. *Journal of Rheumatology* **18** (Suppl. 27), 44–45.

Dedrick DK, Goldstein SA, Brandt KD, O'Connor BL, Goulet RW, Albrech M (1993) A longitudinal study of subchondral plate and trabecular bone in cruciate-deficient dogs with osteoarthritis followed up for 54 months. *Arthritis and Rheumatism* **36**, 1460–1467.

Dedrick DK, Goulet RW, O'Connor BL, Brandt KD (1995) Changes in subchondral plate and cancellous bone in an accelerated canine model of osteoarthritis. *Transactions of the Orthopedic Research Society* **20**, 248.

Eckstein F, Müller-Gerbl M, Putz R (1992) Distribution of subchondral bone density and cartilage thickness in the human patella. *Journal of Anatomy* **180**, 425–433.

Eckstein F, Putz R, Müller-Gerbl M, Steinlechner M, Benedetto KP (1993) Cartilage degeneration in the human patella and its relationship to the mineralisation of the underlying bone: a key to the understanding of chondromalacia patellae and femoropatellar arthrosis? *Surgical and Radiologic Anatomy* **15**, 279–286.

Eckstein F, Müller-Gerbl M, Steinlechner M, Kierse R, Putz R (1995) Subchondral bone density of the human elbow assessed by CT osteoabsorptiometry: a reflection of the loading history of the joint surfaces. *Journal of Orthopedic Research* **13**, 268–278.

Ficat P (1973) *Les Déséquilibres Rotuliens de l'Hyperpression à l'Arthrose*. Paris: Masson.

Ficat P, Hungerford DS (1977) *Disorders of the Patellofemoral Joint*. Paris: Masson.

Goodfellow J, Hungerford DS, Zindel M (1976) Patello-femoral joint mechanics and pathology. Functional anatomy of the patello-femoral joint. *Journal of Bone and Joint Surgery* **58B**, 287–290.

Goodship AE, Lanyon LE, McFie H (1979) Functional adaptation of bone to increased stress. An experimental study. *Journal of Bone and Joint Surgery* **61B**, 539–546.

Grynpas MD, Alpert B, Katz I, Leiberman I, Pritzker KP (1991) Subchondral bone in osteoarthrosis. *Calcified Tissue International* **49**, 20–26.

Heegaard JH, Leyvraz PF, Curnier A, Rakotomanana L, Huiskes R (1995) The biomechanics of the human patella during passive knee flexion. *Journal of Biomechanics* **28**, 1265–1279.

Hehne HJ (1990) Biomechanics of the patellofemoral joint and its clinical relevance. *Clinical Orthopaedics and Related Research* **258**, 73–85.

Hille KP, Schulitz KP, Henrichs C, Schneider T (1985) Pressure and contact-surface measurements within the femoropatellar joint and their variations following lateral release. *Archives of Orthopaedic and Trauma Surgery* **104**, 275–282.

Hoshino A, Wallace WA (1978) Impact-absorbing properties of the human knee. *Journal of Bone and Joint Surgery* **69B**, 807–811.

Huiskes R, Weinans H, Grootenboer HJ, Dalstra M, Fudala B, Sloof TJ (1987) Adaptive bone-remodeling theory applied to prosthetic-design analysis. *Journal of Biomechanics* **20**, 1135–1150.

Kiviranta I, Tammi M, Jurvelin J, Arokoski J, Säämänen AM, Helminen HJ (1994) Articular cartilage thickness and glycosaminoglycan distribution in the young canine knee joint after remobilization of immobilized limb. *Journal of Orthopedic Research* **12**, 161–167.

Kummer B (1966) Photoelastic studies on the functional structure of bone. *Folia Biotheoretica* **6**, 31–40.

Layton MW, Goldstein SA, Goulet RW, Feldkamp LA, Kubinski DJ, Bole GG (1988) Examination of subchondral bone architecture in experimental osteoarthritis by microscopic computed axial tomography. *Arthritis and Rheumatism* **31**, 1400–1405.

Meachim G, Emery IH (1974) Quantitative aspects of patellofemoral cartilage fibrillation in Liverpool necropsies. *Annals of the Rheumatic Diseases* **33**, 39–47.

Mente PL, Lewis JL (1994) Elastic modulus of calcified cartilage is an order of magnitude less than that of subchondral bone. *Journal of Orthopedic Research* **12**, 637–647.

Milz S, Putz R (1994) Quantitative morphology of the subchondral plate of the tibial plateau. *Journal of Anatomy* **185**, 103–110.

Milz S, Eckstein F, Putz R (1995) The thickness of the subchondral plate and its correlation with the thickness of the uncalcified articular cartilage in the human patella. *Anatomy and Embryology* **192**, 437–444.

Milz S, Eckstein F, Putz R (1997) Thickness distribution of the subchondral mineralization zone of the trochlear notch and its correlation with the cartilage thickness: an expression of

functional adaptation to mechanical stress acting on the humeroulnar joint. *Anatomical Record* **248**, 189–197.

Mitrovic D, Stankovic A, Borda-Iriarte O, Uzan M, Quintero M, Ryckewaert A (1987) Résultats de l'examen autopsique des cartilages des genoux chez 120 sujets décédés en milieu hospitalier. 1.Articulation fémoro-patellaire. *Revue du Rheumatisme* **54**, 15–21.

Müller-Gerbl M, Putz R, Hodapp N, Schulte E, Wimmer B (1989) Computed tomography-osteoabsorptiometry for assessing the density distribution of subchondral bone as a measure of long term mechanical adaptation in individual joints. *Skeletal Radiology* **18**, 507–512.

Müller-Gerbl M, Griebl R, Putz R, Goldmann A, Kuhr M, Taeger KH (1994) Assessment of subchondral bone density distribution patterns in patients subjected to correction osteotomy. *Transactions of the Orthopaedic Research Society* **19**, 574.

Oettmeier R, Arokoski J, Roth AJ, Helminen HJ, Tammi M, Abendroth K (1992) Quantitative study of articular cartilage and subchondral bone remodeling in the knee joint of dogs after strenuous running training. *Journal of Bone and Mineral Research* **7** (Suppl. 2), 419–424.

Outerbridge RE (1961) The etiology of chondromalacia patellae. *Journal of Bone and Joint Surgery* **43B**, 752–757.

Pauwels F (1963) Die Druckverteilung im Ellbogengelenk, nebst grundsätzlichen Bemerkungen über den Gelenkdruck. 11. Beitrag zur funktionellen Anatomie und kausalen Morphologie des Stützapparates. *Zeitschrift für Anatomie und Entwicklungsgeschichte* **123**, 643–667.

Pauwels F (1980) *Biomechanics of the Locomotor Apparatus*. Berlin, Heidelberg, New York: Springer.

Pedley RB, Meachim G (1979) Topographical variation in patellar subarticular calcified tissue density. *Journal of Anatomy* **128**, 737–745.

Radin EL, Paul IL (1970) Does cartilage compliance reduce skeletal impact loads? The relative force-attenuating properties of articular cartilage, synovial fluid, periarticular soft tissues and bone. *Arthritis and Rheumatism* **13**, 139–144.

Radin EL, Paul IL, Lowy M (1970) A comparison of the dynamic force transmitting properties of subchondral bone and articular cartilage. *Journal of Bone and Joint Surgery* **52A**, 444–456.

Radin EL, Paul IL, Rose RM (1972) Role of mechanical factors in the pathogenesis of primary osteoarthrosis. *Lancet* **1**, 519–522.

Radin EL, Abernethy PJ, Townsend PM, Rose RM (1978) The role of bone changes in the degeneration of articular cartilage in osteoarthrosis. *Acta Orthopaedica Belgica* **44**, 55–63.

Radin EL, Rose RM (1986) Role of subchondral bone in the initiation and progression of cartilage damage. *Clinical Orthopaedics and Related Research* **213**, 34–40.

Radin EL, Burr DB, Caterson B, Fyhrie D, Brown TD, Boyd RD (1991) Mechanical determinants of osteoarthrosis. *Seminars in Arthritis and Rheumatism* **21** (3 Suppl. 2), 12–21.

Raux P, Townsend PR, Miegel R, Rose RM, Radin EL (1975) Trabecular architecture of the human patella. *Journal of Biomechanics* **8**, 1–7.

Redler I, Mow VC, Zimny ML, Mansell J (1975) The ultrastructure and biomechanical significance of the tidemark of articular cartilage. *Clinical Orthopaedics and Related Research* **112**, 357–362.

Roux W (1896) Über die Dicke der statischen Elementarteile und die Maschenweite der Substantia spongiosa der Knochen. *Zeitschrift für orthopädische Chirurgie*. IV. Band, Separatdruck

Rubin CT, Lanyon LE (1987) Osteoregulatory nature of mechanical stimuli: function as a determinant for adaptive remodeling in bone. *Journal of Orthopedic Research* **5**, 300–310.

Shimizu M, Tsuji H, Matsui H, Katoh Y, Sano A (1993) Morphometric analysis of subchondral bone of the tibial condyle in osteoarthrosis. *Clinical Orthopaedics and Related Research* **293**, 229–239.

Simon SR, Radin EL (1972) The response of joints to impact loading. II. In vivo behavior of subchondral bone. *Journal of Biomechanics* **5**, 267–272.

Smith RL, Thomas KD, Schurman DJ, Carter DR, Wong M, Van Der Meulen MC (1992) Rabbit knee immobilization: bone remodeling precedes cartilage degradation. *Journal of Orthopedic Research* **10**, 88–95.

Tillmann B, Brade H (1980) Die Beanspruchung des Femoropatellargelenkes. *Anatomischer Anzeiger* **147**, 477–478.

Townsend PR, Raux P, Rose RM, Miegel RE, Radin EL (1975) The distribution and anisotropy of the stiffness of cancellous bone in the human patella. *Journal of Biomechanics* **8**, 363–367.

Weh L, Eickhoff W (1983) Innervationsstörungen des Musculus quadriceps bei Chondropathia patellae. *Zeitschrift für Orthopädie und ihre Grenzgebiete* **121**, 171–176.

Wolff J (1986) *Das Gesetz der Transformation der Knochen*. Berlin: Hirschwald, 1892 (reprint Stuttgart: Schattauer, 1991); also: translated by Maquet P, Furlong R, *The Law of Bone Remodelling*. Berlin: Springer.

Wu DD, Burr B, Boyd RD, Radin EL (1990) Bone and cartilage changes following experimental varus or valgus tibial angulation. *Journal of Orthopedic Research* **8**, 572–585.