Osteoarthritis and Cartilage xxx (xxxx) xxx–xxx

Osteoarthritis and Cartilage

OARSI OSTEOARTHRITIS RESEARCH SOCIETY INTERNATIONAL

Structural knee MRI findings are already frequent in a general population-based birth cohort at 33 years of age

Antti Kemppainen # † *, Joona Tapio ‡, Miika T. Nieminen # † §, Simo Saarakkala # † §, Mika T. Nevalainen # † § [1]

Research Unit of Health Sciences and Technology, Faculty of Medicine, University of Oulu, P.O. Box 5000, FI-90014 Oulu, Finland
† *Department of Diagnostic Radiology, Oulu University Hospital, P.O. Box 50, FI-90029 Oulu, Finland*
‡ *Biocenter Oulu and Faculty of Biochemistry and Molecular Medicine, Oulu Center for Cell-Matrix Research, University of Oulu, P.O. Box 5400, FIN-90014 Oulu, Finland*
§ *Medical Research Center Oulu, University of Oulu and Oulu University Hospital, Oulu, Finland*

ARTICLE INFO

Article history:
Received 28 November 2024
Accepted 10 April 2025

Keywords:
Cartilage, Articular/diagnostic imaging
Knee Joint/diagnostic imaging
Magnetic Resonance Imaging
Osteoarthritis, Knee/diagnostic imaging
Osteophyte/diagnostic imaging

SUMMARY

Objective: To evaluate the incidence and severity of knee magnetic resonance imaging (MRI) findings and their associated lifestyle and health factors in a relatively healthy subset of a general population-based birth cohort.

Design: The study population (n = 288, 61.1% females, mean age 33.7 years) is a subpopulation of the Northern Finland Birth Cohort 1986 on whom a thorough clinical evaluation, laboratory analyses and knee MRI were conducted at 33 years of age. Knee MRI data was graded using the MRI Osteoarthritis Knee Score system. Descriptive statistics and multivariable regression models were used for data analysis.

Results: Subjects were mostly asymptomatic. Detected articular cartilage lesions were mostly small and identified in 56.2% (n = 162) of patellofemoral and 25.3% (n = 63) of tibiofemoral joints. Full-thickness cartilage lesions and bone marrow lesions were mostly located in the patellofemoral joint. Osteophytes, mostly small or doubtful, were detected in 51.7% (n = 146) of patellofemoral and 17.4% (n = 41) of tibiofemoral joints. In finding-specific regression analyses, higher body mass index (BMI) was most frequently associated with knee MRI findings.

Conclusions: In this relatively young and asymptomatic population, subtle knee MRI findings were already frequent, especially in the patellofemoral joint. Of analyzed background and clinical parameters, higher BMI was most frequently associated with MRI findings. Based on these results, longitudinal studies are warranted to further identify risk factors and proportions of progressing MRI findings.

Introduction

Knee joint is the most common joint affected by osteoarthritis (OA).[1] Degenerative findings in the knee joint are common in older adults,[1,2] but can manifest already in early adulthood. A large cohort study of over 290 000 patients found that the fraction of patients with OA in the 18–44-year age category had increased from 6.2% in 2001 to 22.7% in 2018.[3] The Global Burden of Disease Study 2019 survey[1] reported a consistent rise in the global OA incidence and prevalence in the 30–44-year age groups from 1990 to 2019. In high socio-demographic index regions, the incidence and prevalence of OA in these age groups exceeded the age-standardized world average. As high body mass index (BMI) contributed to only approximately 20% of the total OA burden,[1] the increase is likely attributed to increased health awareness, a lowered threshold to seek medical evaluation and, consequently, diagnostic imaging.[4]

Magnetic resonance imaging (MRI) is the gold standard of modern OA research for identifying structural changes within a joint.[5,6] MRI is increasingly utilized in patient workup, with published reports of all MRI examinations tripling between 1997–2006 in a large nonprofit healthcare organization in the US,[7] and the annual growth in adult MRI scans being approximately 11.4% between 2000–2004 and 1–2% between 2005–2016 in a large study of several

* Corresponding author at: Department of Diagnostic Radiology, Oulu University Hospital, P.O. Box 50, FI-90029 Oulu, Finland.
E-mail address: antti.kemppainen@pohde.fi (A. Kemppainen).
[1] ORCID ID: 0000-0002-9483-7690

https://doi.org/10.1016/j.joca.2025.04.008

North American healthcare systems,[8] and approximately 9.2% between 1995–2021 in Finland.[9] Conventional knee MRI offers superb structural information but has limitations to detect microstructural OA changes; however, quantitative MRI provides an interesting tool to study early cartilage and bone degeneration.[10]

Previous research on knee MRI findings in young adults has focused on athletes and post-injury patients.[11–15] Studies on subjects without obvious OA risk factors or previous injuries are sparse.[16,17] Utilizing the available MRI data and comprehensive background information of the Northern Finland 1986 Birth Cohort (NFBC1986), the objective of this study was to provide a baseline characterization of knee MRI findings and their related health factors in young adults.

Method

Study population

NFBC1986 is a longitudinal general population-based birth cohort consisting of 99% of children who were due to be born in the two northernmost provinces of Finland between July 1985 and June 1986 (n = 9432).[18] The latest data collection was conducted at the age of 33 years. For a subset (n = 297) of the cohort, knee MRI was conducted as a random subsample with no other selection criteria. 9 MRIs were incomplete and subsequently excluded. Participants were asked which knee they experienced more symptoms in (77.1% (n = 222) right knee, 22.9% (n = 66) left knee), and that knee joint was imaged.[19]

Inclusion criteria for the current study were a completed knee MRI and available clinical and postal questionnaire data (n = 288) at 33 years of age. A flow-chart of the study population is presented in Fig. S1.

Background and clinical characteristics

Background information was determined by postal questionnaires. Subjects self-reported their medical history, prior lower limb fractures, family history of knee OA (parents, siblings and grandparents, classed as “no” or “yes”), medications, smoking status (“never” or “ever”), and the frequency and number of alcoholic beverages consumed. “Physical activity score” was based on four questions: How long do you perform light and heavy exercise at a time (none (0) to over 1.5 h (6)) and how often (once a month at most (0) to daily (6)), respectively, and the sum was used in the analyses as a continuous variable.

Before the study visit, the participants were informed to abstain from caffeine and smoking. The examination consisted of blood sampling and clinical measurements. Body height and weight, waist and hip circumference, brachial systolic and diastolic blood pressure (Sbp and Dbp) and heart rate were measured by trained nurses. BMI was calculated as the ratio of weight to height squared.

The Visual Analog (VAS) version of The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC)[20,21] scale was filled prior to knee MRI. The questionnaire was asked to be filled out concerning the knee joint to be imaged. Participants marked answers on a 100-mm VAS scale, which were then measured with a ruler and documented. In the VAS version of WOMAC, each question is scored from 0 to 10. Subscale scores range from 0 to 50 (pain), 0 to 20 (stiffness) and 0 to 170 (physical function). For data presentation, cut-off values for each question were set at 0–3 (none to mild), 4–7 (moderate) and 8–10 (high) on the VAS scale as in[22] and reported (Table S1). For reference, the VAS scale (0 to 240) was transformed to the Likert Scale (0 to 96) and reported (Table S2).

Laboratory analyses

Laboratory analyses were conducted from fresh blood samples after an overnight fast. Fasting plasma glucose, serum total cholesterol and plasma urate (P-Urate) were determined with photometric, enzymatic assays. High- (HDL) and low-density lipoprotein (LDL) were determined by photometric, direct enzymatic measurements. Plasma alanine aminotransferase (P-ALAT) level was determined with a photometric assay. An immune nephelometric assay (BN ProSpec, Siemens Healthcare Diagnostics INC., Newark, DE) was used to define the concentration of high-sensitivity C-reactive protein (hs-CRP).

Magnetic resonance imaging

Knee MRI was performed using two 3 T MRI scanners (“Skyra” and “Vida”, Siemens Healthineers, Erlangen, Germany) with dedicated knee coils. Similar MRI protocol was used for all subjects with identical repetition time (TR), echo time (TE) and flip angle (FA) in different pulse sequences. Turbo spin echo T2 sequence with fat saturation (TR: 1000 ms; TE: 123 ms; FA: 120°, acquisition time 6 min 32 s, 192 slices), turbo spin echo proton density weighted sequence (TR: 900 ms; TE: 76 ms; FA: 120°, acquisition time 6 min 28 s, 176 slices) and double echo steady state sequence (TR: 14.1 ms; TE: 5 ms; FA: 25°, acquisition time 4 min 4 s, 160 slices) were used. All the sequences were isotropic (i.e. 3D), the field-of-view was 160×160 mm^2, the acquired and reconstructed image sizes were 256×256, slice orientation was sagittal, slice gap was 20% and slice thickness and spacing were 0.6 mm, respectively. Subjects without all three MRI sequences were excluded from this study.

MRIs were scored using the semi-quantitative MRI Osteoarthritis Knee Score (MOAKS) system[23,24] by a sixth-year radiology resident with ten years of experience in musculoskeletal research (AK) after training and calibration sessions with a board-certified fellowship-trained musculoskeletal radiologist with eleven years of experience (MTN). Grade 1 osteophytes were defined as either small or doubtful. Small (grade 1) knee effusion was defined as fluid continuous in the retropatellar space or any knee effusion with measurable width of 3 mm or more. Meniscal extrusion and hypertrophy were not assessed.

MOAKS-defined subregions were pooled into seven regions: 1) tibial medial (anterior, central and posterior medial tibia subregions) 2) tibial lateral (anterior, central and posterior lateral tibia subregions) 3) femoral medial (central and posterior medial femur subregions) 4) femoral lateral (central and posterior lateral femur subregions) 5) tibiofemoral (1–4 combined), 6) patellofemoral (patellofemoral medial and lateral, and anterior medial and lateral femur subregions) and 7) any. A single highest grade for cartilage loss, full thickness cartilage loss, bone marrow lesion (BML) size, BML characteristics and osteophyte size was reported for each anatomic region.

Statistical analysis

Subjects with missing data were omitted from individual analyses. Continuous variables were checked for skewness. Normally distributed continuous variables are presented as mean (M) and standard deviation, Non-normally distributed continuous variables are presented as median (Mn) and interquartile range. Count data is presented as number of observations (n) and percentage.

MRI parameters with < 5 cases were not analyzed beyond descriptive statistics. For parameters with ≥ 5 cases, unadjusted regression analyses evaluating individual associations with background and clinical parameters were conducted. In case of a binary outcome, a logistic regression model was used, and a Poisson regression model with robust standard errors was used for multi-classed outcomes. For colinear variables (such as Sbp and Dbp), only one of the parameters is reported.

In multivariable regression models, the first model included family history, sex, P-urate, BMI and Sbp as independent variables. The second model included family history, P-urate and BMI as independent variables. Independent variables were selected as covariates based on the unadjusted associations (BMI, P-urate, Sbp) and/or are well-established to be associated with knee OA and showed large variation in the risk estimates (sex, family history).

To validate results from the regression analyses, we did classifications solely for descriptive purposes. These classes were not used in regression analyses. BMI classification was done according to World Health Organization guidelines.[25] Height tertiles were first done separately for males and females and pooled afterwards. Severity categories based on MOAKS classification were based on the criteria from Whole-Organ MRI Score system as in.[26] The three categories based solely on MRI findings were set as: cartilage loss or osteophyte or both MOAKS < 1, both MOAKS = 1 and both MOAKS ≥ 2. The same categories were used as a basis for the symptomatic categories, that included pain ≥ VAS 3 in any of the pain questions in WOMAC (Table S1) as an additional criterion for the latter two categories.

To approximate the prevalence of OA with Hunter's classification,[27] we defined tibiofemoral OA as: Any tibiofemoral osteophyte MOAKS ≥ 2 + any tibiofemoral full thickness cartilage loss, or either of the previous and two of the following criteria: Any BML, horizontal meniscal tear or meniscal maceration, any cartilage loss MOAKS ≥ 2. Patellofemoral OA was defined as patellofemoral osteophyte MOAKS ≥ 2 + either of the following: Patellofemoral cartilage loss MOAKS ≥ 2 or patellofemoral full-thickness cartilage loss.

Statistical analyses were performed using IBM SPSS statistics, version 29.0.0.0 (IBM Corp, Armonk, NY).

Ethical aspects

This study was conducted adhering to the Declaration of Helsinki and with the approval of the Northern Ostrobothnia Hospital District Ethics Committee and the University of Oulu.

Results

Background and clinical characteristics

The background and clinical characteristics are presented in Table I. The study population consisted of 288 participants (61.1% females). All participants were around 33 years of age. Prior lower limb fractures and alcohol consumption were more common in males. Regular use of anti-inflammatory medication was sparse. Similar family history, smoking status and physical activity were observed for both sexes (Table I). The mean BMI was 25.7 kg/m^2, classified as slightly overweight.[25] Males had a longer waist circumference and higher bp values and higher levels of fasting glucose, serum lipids, P-Urate and P-ALAT and females had higher heart rate and hs-CRP (Table I).

The average total WOMAC scores were low overall (mean 8.4 / 240) (Table II). Most participants were asymptomatic, with 93.0 to 98.9% having no to mild knee pain, 96.5 to 96.8% having no to mild knee stiffness and 97.5 to 99.3% having no to mild loss of physical function (Tables S1 and S2).

	All subjects	Males	Females
	M (SD)/Mn (IQR) or n (%)	M (SD)/Mn (IQR) or n (%)	M (SD)/Mn (IQR) or n (%)
Background characteristics			
Participants	288 (100.0)	112 (100.0)	176 (100.0)
Males, n (%)	112 (38.9)	112 (100.0)	0 (0.0)
Females, n (%)	176 (61.1)	0 (0.0)	176 (100.0)
Age (years)	33.7 (0.4)	33.7 (0.4)	33.7 (0.4)
Prior lower limb fracture, n (%)	37 (12.8)	20 (17.9)	17 (9.7)
Family history of knee OA, n (%)	93 (32.3)	37 (33.0)	56 (31.8)
Anti-inflammatory medication, n (%)	6 (2.1)	3 (2.7)	3 (1.7)
Never smoker, n (%)	110 (38.2)	37 (33.0)	74 (42.0)
Ever smoker, n (%)	178 (61.8)	75 (67.0)	102 (58.0)
Alcohol consumption g/week	16.5 (10.5–34.9)	31.5 (13.5–49.5)	13.5 (7.5–31.5)
Physical activity score	14.9 (3.3)	14.8 (3.7)	14.8 (3.1)
Clinical characteristics			
BMI (kg/m^2)	25.7 (4.6)	25.7 (3.6)	25.8 (5.1)
Height (cm)	171.2 (9.4)	180.4 (5.8)	165.4 (5.8)
Weight (kg)	75.7 (15.8)	83.6 (12.8)	70.7 (15.5)
Waist circumference (cm)	87.0 (13.1)	91.8 (10.0)	83.9 (14.0)
Hip circumference (cm)	99.7 (11.5)	100.2 (6.4)	99.5 (13.8)
Systolic blood pressure (mmHg)	112 (12.2)	120.1 (10.4)	106.8 (10.2)
Diastolic blood pressure (mmHg)	74.2 (8.9)	76.5 (8.3)	72.8 (9.0)
Heart rate (bpm)	72 (12)	69.9 (13.7)	73.4 (11.1)
fP-Glucose (mmol/L)	5.0 (0.6)	5.2 (0.4)	4.9 (0.7)
fP-Total cholesterol (mmol/L)	4.7 (0.9)	4.9 (0.9)	4.5 (0.8)
fP-HDL cholesterol (mmol/L)	1.5 (0.3)	1.4 (0.3)	1.6 (0.3)
fP-LDL cholesterol (mmol/L)	2.7 (0.8)	3.0 (0.8)	2.6 (0.8)
P-Urate (umol/L)	304.4 (70.3)	352.5 (58.2)	273.8 (59.4)
P-Alat (U/l)	21.0 (15.0–30.0)	30.0 (22.5–47.5)	17.0 (13.0–22.0)
hs-CRP (mg/L)	0.8 (0.4–1.6)	0.5 (0.25–1.0)	0.9 (0.5–2.0)

Data is presented as mean (M) and SD for normally-distributed continuous variables, median (Mn) and interquartile range (IQR) for skewed continuous variables and count (n) and percentage (%) for count variables.
M, Mean; SD, Standard Deviation; fP-, fasted plasma; Alat, alanine aminotransferase.

Table I

Osteoarthritis and Cartilage

Background and clinical characteristics of the study population.

	N	Min	Max	Mean	SD
Whole population					
Pain (0–50)	284	0	34.0	2.4	4.1
Stiffness (0–20)	284	0	16.0	1.0	2.0
Physical function (0–170)	284	0	99.5	4.9	10.6
Total (0–240)	284	0	128.5	8.4	15.7
Males					
Pain (0–50)	111	0	28.0	2.0	3.9
Stiffness (0–20)	111	0	16.0	0.9	2.0
Physical function (0–170)	111	0	99.5	4.3	12.2
Total (0–240)	111	0	128.5	7.2	17.1
Females					
Pain (0–50)	173	0	34.0	2.7	4.2
Stiffness (0–20)	173	0	10.5	1.2	2.0
Physical function (0–170)	173	0	69.0	5.3	9.6
Total (0–240)	173	0	113.5	9.1	14.8

Number of study participants (N), minimum (Min), maximum (Max), Mean and standard deviation (SD) of pain (0 to 50), stiffness (0 to 20), physical function (0 to 170) and total WOMAC scores (0 to 240) for whole study population, males and females.

Table II Osteoarthritis and Cartilage

WOMAC characteristics of the study population on VAS scale.

MRI findings

Prevalence and severity of the most severe cartilage lesions, BMLs and osteophytes are presented in Tables III, S3 and S4. In the tibiofemoral joint, the percentage of joint quadrants with no cartilage lesions ranged between 81.3% and 95.5% (Table III). In total, tibiofemoral cartilage lesions were found in 25.3% of the joints (Table S5). Most of the tibiofemoral cartilage lesions were small and full-thickness cartilage lesions and BMLs were rare (Table S5). Most tibiofemoral osteophytes were small or doubtful, whereas medium and large osteophytes were infrequent (Table III).

In the patellofemoral joint, cartilage lesions were found in 56.2% of the joints, with the most severe cartilage lesion being small in 36.5%, moderate in 17.0% and large in 2.8% of subjects, respectively (Tables III and S5). Cartilage lesions were more common in females (59.1%) compared to males (51.8%), mostly accounted for by small lesions (Tables S3 and S4). Patellofemoral osteophytes were common, with the most severe in-subject finding being small or doubtful (44.1%), medium (5.9%) and large (0.7%), respectively, and more common in males (58.9%) compared to females (45.5%), with most of the difference explained by small or doubtful osteophytes (Tables III, S3 and S4). Full-thickness cartilage lesions were found in 11.8% of MRIs, with the majority being small lesions (Tables III and S5).

Almost two thirds of knees had any cartilage lesion (63.9%,), and more than half of the knees had at least a small or doubtful osteophyte (54.2%, Table III).

Other MRI features evaluated are presented in Table IV. 85.8% of medial and 96.9% of lateral menisci were normal in MRI. Meniscal findings were slightly more common in males compared to females, mostly accounted for by intrameniscal signal (Tables S6 and S7). Two reconstructed anterior cruciate ligament (ACL) tears and three posterior cruciate ligament (PCL) tears were observed. Increased patellar tendon signal was identified in 4.2% of MRIs. Ganglion cysts were seen in 21.9% of MRIs, being almost exclusively small extra-articular cysts at gastrocnemius and popliteus tendon insertions (data not shown). Increased T2-signal was seen in the infrapatellar region in 17% and the prepatellar region in 33% of MRIs. Popliteal cysts were present in 38.2% of MRIs, being more common in males. Small knee effusion was seen in 34.4%, moderate in 7.3% and large in 1% of subjects, and joint effusion was more common in males (Tables IV, S6, S7).

	Tibiofemoral	Tibial medial	Tibial lateral	Femoral medial	Femoral lateral	Patellofemoral	Any
Cartilage loss							
0 (none)	215 (74.7)	275 (95.5)	261 (90.6)	234 (81.3)	271 (94.1)	126 (43.8)	104 (36.1)
1 (< 10%)	49 (17.0)	11 (3.8)	23 (8.0)	34 (11.8)	12 (4.2)	105 (36.5)	113 (39.2)
2 (10–75%)	22 (7.6)	2 (0.7)	4 (1.4)	19 (6.6)	4 (1.4)	49 (17.0)	62 (21.5)
3 (> 75%)	2 (0.7)	0 (0.0)	0 (0.0)	1 (0.3)	1 (0.3)	8 (2.8)	9 (3.1)
Full-thickness cartilage loss							
0 (none)	270 (93.8)	286 (99.3)	282 (97.9)	279 (96.9)	281 (97.6)	254 (88.2)	241 (83.7)
1 (< 10%)	11 (3.8)	1 (0.3)	6 (2.1)	4 (1.4)	4 (1.4)	27 (9.4)	34 (11.8)
2 (10–75%)	6 (2.1)	1 (0.3)	0 (0.0)	4 (1.4)	3 (1.0)	6 (2.1)	11 (3.8)
3 (> 75%)	1 (0.3)	0 (0)	0 (0.0)	1 (0.3)	0 (0.0)	1 (0.3)	2 (0.7)
Size of BML							
0 (none)	272 (94.4)	286 (99.3)	282 (97.9)	281 (97.6)	283 (98.3)	262 (91.0)	248 (86.1)
1 (< 33%)	12 (4.2)	1 (0.3)	6 (2.1)	5 (1.7)	4 (1.4)	21 (7.3)	32 (11.1)
2 (33–66%)	3 (1.0)	1 (0.3)	0 (0.0)	1 (0.3)	1 (0.3)	4 (1.4)	6 (2.1)
3 (> 66%)	1 (0.3)	0 (0.0)	0 (0.0)	1 (0.3)	0 (0.0)	1 (0.3)	2 (0.7)
% of lesion that is BML							
0 (none)	272 (94.4)	286 (99.3)	282 (97.9)	281 (97.6)	283 (98.3)	265 (92.0)	251 (87.2)
1 (< 33%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
2 (33–66%)	1 (1.0)	0 (0.0)	0 (0.0)	1 (0.3)	0 (0.0)	1 (0.3)	1 (0.3)
3 (> 66%)	15 (5.2)	2 (0.7)	6 (2.1)	6 (2.1)	5 (1.7)	22 (7.6)	36 (12.5)
Osteophytes							
0 (none)	238 (82.6)	269 (93.4)	266 (92.4)	270 (93.8)	250 (86.8)	142 (49.3)	132 (45.8)
1 (small or doubtful)	41 (14.2)	14 (4.9)	17 (5.9)	13 (4.5)	32 (11.1)	127 (44.1)	135 (46.9)
2 (medium)	7 (2.4)	3 (1.0)	4 (1.4)	3 (1.0)	4 (1.4)	17 (5.9)	19 (6.6)
3 (large)	2 (0.7)	2 (0.7)	1 (0.3)	2 (0.7)	2 (0.7)	2 (0.7)	2 (0.7)

Data is presented as count (n) and percentage (%).

Table III Osteoarthritis and Cartilage

Counts of the most severe in-patient MRI-detected cartilage lesions, BMLs and osteophytes in the tibiofemoral and patellofemoral joint regions.

	Medial			Lateral		
	Anterior	Body	Posterior	Anterior	Body	Posterior
Meniscal morphology						
Normal	281 (97.6)	250 (86.8)	251 (87.2)	284 (98.7)	285 (99.0)	283 (98.3)
Intrameniscal signal	3 (1.0)	21 (7.3)	22 (7.6)	1 (0.3)	0 (0)	3 (1.0)
Vertical tear	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Horizontal tear	1 (0.3)	11 (3.8)	11 (3.3)	2 (0.7)	1 (0.3)	2 (0.7)
Radial tear	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Complex tear	2 (0.7)	4 (1.4)	2 (0.7)	1 (0.3)	2 (0.7)	0 (0)
Partial maceration	1 (0.3)	2 (0.7)	2 (0.7)	0 (0)	0 (0)	0 (0)
Total maceration	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Other parameters of interest	Present					
ACL tear	2 (0.7)					
ACL repair	2 (0.7)					
PCL tear	3 (1.0)					
PCL repair	0 (0)					
Patellar tendon signal	12 (4.2)					
Any ganglion cyst	63 (21.9)					
Pes anserine bursitis	2 (0.7)					
Infrapatellar bursa signal	49 (17.0)					
Prepatellar bursa signal	95 (33.0)					
Popliteal cyst	110 (38.2)					
	None	*Small*	*Medium*	*Large*		
Joint effusion	165 (57.3)	99 (34.4)	21 (7.3)	3 (1.0)		

Data is presented as count (n) and percentage (%).

Table IV

Osteoarthritis and Cartilage

Meniscal morphology, other parameters of interest and prevalence and severity of knee joint effusion in the study population.

Unadjusted associations of background and clinical parameters with MRI findings

Next, we evaluated unadjusted associations for individual background and clinical variables with the MRI findings. The relative risk ratios (RRs) or odds ratios (ORs) of each analyzed parameter in an age- and sex-adjusted regression model are presented in Tables V and S8.

Considering the clinical parameters, BMI was positively associated with 22/40 MRI parameters, including tibiofemoral cartilage lesions, full thickness cartilage lesions, BMLs and osteophytes. Besides BMI, p-Urate was also positively associated with 14/40 and Sbp with 9/40 MRI findings. Other clinical parameters were not as prominently associated with MRI findings (Table V).

Out of background parameters, a family history of knee OA was associated with 5/40 MRI parameters, most notably tibiofemoral

	BMI (kg/m^2)	P-Urate (umol/L)	P-Alat (U/l)	hs-CRP (mg/L)	LDL chol. (umol/L)	fP-glucose (umol/L)	Systolic bp (mmHg)
Cartilage loss							
Tibial medial	1.185 (1.102–1.274)	1.007 (1.001–1.012)	1.011 (0.998–1.024)	1.059 (0.991–1.132)	0.976 (0.468–2.038)	1.316 (0.957–1.809)	1.039 (1.007–1.072)
Tibial lateral	1.091 (1.021–1.165)	1.008 (1.003–1.012)	1.011 (1.002–1.020)	1.002 (0.876 –1.147)	1.359 (0.882–2.092)	1.202 (0.915–1.579)	1.038 (1.017–1.059)
Femoral medial	1.091 (1.044–1.141)	1.003 (1.000–1.006)	1.007 (0.999–1.015)	0.978 (0.868–1.102)	1.278 (0.983–1.660)	1.121 (0.835–1.506)	1.011 (0.992–1.030)
Femoral lateral	1.099 (1.010–1.194)	1.004 (0.999–1.009)	1.006 (0.992–1.020)	0.996 (0.881–1.125)	1.108 (0.594–2.068)	1.282 (0.959–1.713)	1.045 (1.025–1.065)
Tibiofemoral	1.070 (1.029–1.112)	1.003 (1.001–1.006)	1.006 (0.998–1.013)	0.950 (0.830–1.088)	1.152 (0.909–1.461)	1.113 (0.854–1.450)	1.013 (0.998–1.028)
Patellofemoral	1.015 (0.992–1.040)	1.000 (0.999–1.002)	1.000 (0.993–1.008)	0.973 (0.925–1.023)	1.037 (0.899–1.197)	0.900 (0.716–1.132)	1.006 (0.997–1.016)
Any	1.017 (0.996–1.038)	1.001 (1.000–1.002)	1.001 (0.996–1.007)	0.962 (0.914–1.012)	1.009 (0.892–1.142)	0.962 (0.788–1.174)	1.007 (0.999–1.015)
FT cartilage loss							
Tibial lateral*	0.970 (0.803–1.172)	1.012 (1.001–1.023)	1.017 (0.994–1.040)	0.498 (0.128–1.944)	1.165 (0.443–3.064)	1.199 (0.474–3.035)	1.046 (0.988–1.107)
Femoral medial	1.158 (1.037–1.293)	1.002 (0.995–1.008)	1.004 (0.973–1.035)	1.055 (0.933–1.192)	1.145 (0.649–2.019)	1.228 (0.813–1.855)	0.992 (0.940–1.048)
Femoral lateral	1.126 (0.997–1.272)	1.006 (0.999–1.014)	1.009 (0.994–1.024)	0.911 (0.658–1.261)	1.161 (0.572–2.359)	1.237 (0.835–1.833)	1.011 (0.976–1.047)
Tibiofemoral	1.105 (1.017–1.201)	1.005 (0.999–1.010)	1.011 (0.998–1.024)	1.011 (0.878–1.165)	1.193 (0.748–1.903)	1.189 (0.841–1.681)	1.001 (0.967–1.037)

(continued on next page)

Table V (*continued*)

	BMI (kg/m^2)	P-Urate (umol/L)	P-Alat (U/l)	hs-CRP (mg/L)	LDL chol. (umol/L)	fP-glucose (umol/L)	Systolic bp (mmHg)
Patellofemoral	1.075 (0.999–1.157)	1.005 (1.002–1.009)	0.988 (0.969–1.006)	1.022 (0.917–1.140)	0.840 (0.546–1.293)	0.831 (0.360–1.919)	1.018 (0.994–1.043)
Any	1.072 (1.014–1.133)	1.005 (1.000–1.008)	1.002 (0.991–1.013)	0.997 (0.892–1.114)	0.975 (0.700–1.358)	0.945 (0.582–1.534)	1.010 (0.989–1.032)
Size of BML							
Tibial lateral*	1.057 (0.905–1.236)	1.019 (1.007–1.031)	1.015 (0.991–1.039)	0.772 (0.353–1.693)	1.967 (0.819–4.719)	1.313 (0.617–2.793)	1.064 (1.007–1.125)
Femoral medial	1.208 (1.065–1.369)	1.005 (0.996–1.014)	1.011 (0.985–1.038)	1.113 (1.004–1.233)	1.280 (0.630–2.601)	1.426 (1.059–1.918)	1.017 (0.964–1.074)
Femoral lateral	1.067 (0.987–1.154)	1.000 (0.992– 1.008)	1.007 (0.987–1.028)	0.809 (0.365–1.793)	1.002 (0.431–2.330)	1.237 (0.841–1.819)	1.010 (0.967–1.055)
Tibiofemoral	1.157 (1.061–1.262)	1.006 (1.000–1.012)	1.012 (0.998–1.026)	1.065 (0.958–1.185)	1.539 (1.011–2.343)	1.286 (0.952–1.736)	1.029 (1.000–1.058)
Patellofemoral	1.036 (0.944–1.137)	1.004 (1.001–1.008)	0.978 (0.963–0.994)	1.031 (0.936–1.136)	0.780 (0.450–1.352)	0.890 (0.425–1.863)	1.022 (0.989–1.056)
Any	1.071 (1.000–1.148)	1.005 (1.002–1.008)	1.001 (0.988–1.014)	1.025 (0.933–1.126)	1.048 (0.720–1.526)	1.041 (0.708–1.529)	1.022 (0.998–1.047)
% that is BML							
Tibial lateral*	1.057 (0.905–1.236)	1.019 (1.007–1.031)	1.015 (0.991–1.039)	0.772 (0.353–1.693)	1.967 (0.819–4.719)	1.313 (0.617–2.793)	1.064 (1.007–1.125)
Femoral medial	1.156 (1.046–1.277)	1.002 (0.992–1.012)	1.005 (0.974–1.037)	1.034 (0.903–1.184)	0.861 (0.374–1.985)	1.326 (0.965–1.823)	0.998 (0.952–1.047)
Femoral lateral	1.081 (1.003–1.166)	1.001 (0.992–1.010)	1.010 (0.991–1.029)	0.871 (0.499–1.520)	0.812 (0.360–1.830)	1.181 (0.717–1.944)	1.001 (0.956–1.049)
Tibiofemoral	1.112 (1.047–1.181)	1.006 (0.999–1.013)	1.012 (1.000–1.024)	0.987 (0.870–1.119)	1.342 (0.797–2.260)	1.190 (0.881–1.608)	1.021 (0.992–1.051)
Patellofemoral	1.063 (0.991–1.140)	1.004 (0.999–1.009)	0.980 (0.961–0.998)	0.972 (0.832–1.134)	0.554 (0.333–0.922)	0.871 (0.427–1.775)	1.011 (0.982–1.040)
Any	1.074 (1.024–1.127)	1.005 (1.002–1.009)	1.001 (0.990–1.013)	0.950 (0.832–1.086)	0.849 (0.577–1.247)	0.994 (0.684–1.444)	1.014 (0.992–1.036)
Osteophytes							
Tibial medial	1.176 (1.109–1.247)	1.008 (1.002–1.014)	0.986 (0.967–1.004)	1.068 (0.998–1.142)	1.402 (0.890–2.208)	1.308 (0.986–1.735)	1.023 (0.996–1.050)
Tibial lateral	1.133 (1.061–1.211)	1.005 (0.998–1.012)	0.988 (0.972– 1.005)	1.066 (0.996–1.141)	1.173 (0.658–2.092)	1.182 (0.847–1.650)	1.013 (0.985–1.042)
Femoral medial	1.172 (1.096–1.254)	1.008 (1.002–1.014)	0.988 (0.971–1.005)	1.067 (0.995–1.144)	1.978 (1.322–2.962)	1.303 (0.983–1.728)	1.030 (1.002–1.058)
Femoral lateral	1.109 (1.054–1.167)	1.003 (0.998–1.008)	0.988 (0.973–1.003)	1.039 (0.973–1.109)	1.049 (0.680–1.619)	1.016 (0.681–1.514)	1.011 (0.988–1.036)
Tibiofemoral	1.100 (1.053–1.149)	1.003 (0.999–1.007)	0.989 (0.977–1.002)	1.032 (0.974–1.093)	1.104 (0.784–1.553)	1.122 (0.845–1.490)	1.004 (0.984–1.025)
Patellofemoral	1.037 (1.010–1.066)	1.002 (1.001–1.004)	1.007 (1.004–1.010)	0.954 (0.891–1.022)	1.118 (0.964–1.298)	1.199 (1.051–1.368)	1.013 (1.005–1.022)
Any	1.041 (1.016–1.066)	1.002 (1.000–1.004)	1.007 (1.004–1.010)	0.981 (0.935–1.028)	1.110 (0.964–1.279)	1.191 (1.044–1.359)	1.011 (1.003–1.020)
Meniscal Tear							
Med. body horizontal*	1.126 (1.013–1.253)	1.004 (0.996–1.012)	1.001 (0.974–1.029)	0.970 (0.769–1.223)	1.906 (0.978–3.712)	1.185 (0.570–2.466)	1.012 (0.965–1.061)
Med. posterior horizontal*	1.042 (0.923–1.175)	0.999 (0.990–1.008)	0.997 (0.967–1.028)	0.981 (0.791–1.216)	1.359 (0.675–2.736)	1.246 (0.642–2.419)	1.007 (0.959–1.057)
Other							
Patellar tend. signal*	1.023 (0.907–1.154)	1.003 (0.995–1.011)	1.007 (0.986–1.029)	1.023 (0.876–1.194)	1.098 (0.544–2.214)	0.974 (0.347–2.734)	1.007 (0.961–1.055)
Any ganglion cyst*	0.996 (0.936–1.059)	0.998 (0.994–1.002)	0.982 (0.964–1.001)	1.006 (0.924–1.096)	0.901 (0.634–1.281)	1.224 (0.797–1.881)	1.001 (0.978–1.024)
Inf.pat. bursa signal*	0.993 (0.928–1.063)	1.002 (0.998–1.007)	1.002 (0.989–1.016)	0.991 (0.896–1.096)	1.118 (0.768–1.626)	0.515 (0.231–1.150)	1.018 (0.993–1.043)
Prepat. bursa signal*	1.036 (0.982–1.092)	1.001 (0.998–1.005)	0.990 (0.977–1.003)	1.047 (0.973–1.127)	0.873 (0.641–1.189)	0.910 (0.577–1.434)	1.002 (0.982–1.022)
Joint effusion	1.062 (1.035–1.089)	1.004 (1.003–1.006)	1.005 (0.999–1.010)	0.958 (0.898–1.023)	1.267 (1.069–1.503)	1.115 (0.894–1.391)	1.014 (1.003–1.025)
Popliteal cyst*	1.031 (0.979–1.086)	1.002 (0.998–1.005)	1.006 (0.995–1.017)	0.914 (0.823–1.014)	1.278 (0.952–1.717)	1.019 (0.680–1.526)	1.015 (0.996–1.036)

FT, Full thickness; fP-, fasted plasma; Alat, alanine aminotransferase.

* Indicates a logistic regression model and OR. Unmarked parameters were analyzed with Poisson regression and the result is given as RR.

Table V

Osteoarthritis and Cartilage

Unadjusted Relative Risks or Odds Ratios with 95% Confidence Intervals of individual clinical parameters for knee MRI findings.

cartilage loss and BMLs. Male sex and especially family history had RRs and ORs with large confidence intervals. Of meniscal tears, anti-inflammatory medication (n = 6) drastically increased the risk of medial horizontal tears. Other background parameters were not as prominently associated with MRI findings (Table S8).

MRI findings according to BMI classes

As BMI was most notably associated with MRI findings in the unadjusted regression models, descriptive statistics on knee MRI findings according to BMI classes[25] were conducted (Tables S9 and S10).

For all tibiofemoral cartilage lesions, subjects with BMI < 25 had consistently higher counts of healthy cartilage or grade 1 cartilage loss compared to those with BMI > 25. Furthermore, the percentage of larger cartilage lesions generally increased in line with BMI. For the patellofemoral joint, the prevalence of grade 2–3 cartilage loss increased according to BMI classes (Table S9).

For all osteophytes, the increase in incidence and severity according to BMI classes was most notable. Subjects with BMI < 25 had generally none-to-small or doubtful osteophytes at most, while subjects with BMI > 25 had higher counts of osteophytes in general, with clear incremental increases in the severity and incidence according to BMI classes (Table S9).

Similar findings were observed according to height tertiles (Tables S11 and S12). However, the prevalence of meniscal findings, popliteal cysts and joint effusion increased according to BMI but not according to height.

Multivariable regression models for MRI findings

Next, we conducted multivariable regression models for each MRI parameter with ≥ 5 cases. Independent variables for the models were selected based on individual associations in the unadjusted models. The first model included family history of OA, P-Urate and BMI as explanatory variables (Table S13). The second model included additionally Sbp and male sex as explanatory variables (Table VI).

Of cartilage lesions, BMI was an explanatory variable for 3/4 tibiofemoral joint quadrants with RRs ranging from 1.085 (1.029–1.143) to 1.143 (1.014–1.287) in the second model (Table VI).

For full-thickness cartilage loss, family history and P-urate were associated with tibial lateral cartilage loss, male sex with femoral lateral cartilage loss and BMI with both femoral medial and femoral lateral cartilage loss (Table VI).

Of BML size, family history and P-urate were associated with tibial lateral BMLs, while BMI was associated with femoral medial BML size. Similar results were observed for actual BMLs (Table VI).

Of tibiofemoral osteophytes, BMI was associated with osteophytes in all tibiofemoral quadrants with RRs ranging from 1.123 (1.054 – 1.198) to 1.187 (1.061 – 1.328). BMI was also associated with patellofemoral osteophytes 1.034 (1.003 – 1.066) (Table VI).

For other MRI findings of interest, family history was associated with infrapatellar bursa signal, while male sex and BMI were associated with joint effusion (Table VI).

Background and clinical characteristics in categories based on MOAKS classification

Finally, we analyzed the background and clinical characteristics in three derived severity classes, solely based on the knee MRI findings (Table VII).

There were more female participants with more severe MOAKS scores in the tibiofemoral compartment. The largest differences between the categories were observed in body composition measures, as these increased drastically according to MRI findings severity. Out of other parameters, those participants with more severe MRI findings consumed more alcohol and had higher bp values, p-Urate, hs-CRP and cholesterol values (Table VII). When pain was included as an additional criterion, most differences observed using just the MRI criteria became even more drastic, although the number of participants in the more advanced categories also decreased dramatically (Table S14).

Perhaps coincidentally, all 10 participants that had the most severe MRI findings in the tibiofemoral compartment also had the most severe findings in the patellofemoral compartment. The differences observed in the categories based on tibiofemoral findings were similar but not as incremental as in the tibiofemoral compartment, although most values drastically increased in the participants with the more severe findings (Table VII). Similar findings were observed when including pain as a criterion, although the number of participants in the more advanced categories again decreased dramatically (Table S14).

Discussion

This study describes the incidence and severity of knee MRI findings in mostly asymptomatic 33-year-old participants from the local birth cohort (NFBC1986). We report associations between background and clinical factors with imaging findings, which were surprisingly frequent, especially in the patellofemoral compartment. Of health factors, higher BMI was most frequently associated with MRI findings, with greater incidence and severity corresponding to higher BMI classes.

Few studies have previously described knee MRI findings in young adults without obvious knee OA risk factors, such as previous knee injuries,[28–31] athletic background[28–30,32,33] or physically demanding occupation.[34] In 2003, Sowers et al. studied women aged 35–55 years with and without knee pain and knee OA visible in radiographs. In their MRI dataset of 231 knee joints, only 25% of subjects had no cartilage defects.[17] In 2024, Singh et al. studied knee MRIs of 329 participants aged 35 years: 31.6% had patellofemoral and 23.1% had tibiofemoral cartilage defects.[16] Our results align with a study of 993 consecutive knee arthroscopies (median age 35 years), where cartilage lesions were found in 66% of patients.[35] Here, most cartilage lesions identified were in the patellofemoral joint and the medial tibiofemoral region, the most common sites for chondral lesions.[35–37] This finding is also consistent with histology, where retropatellar cartilage differs significantly in its composition, with, for example, a wider zone of transition. This possibly relates to the inherent role of patellar cartilage to resist shear forces.[38] Our results indicate that chondral lesions start accumulating in the knee joint at approximately 30 years of age as van der Heijden et al. reported only a few cartilage lesions in MRIs of patients with patellofemoral pain and healthy controls (mean age 23 years).[39] Small osteophytes, however, were prevalent in these study groups and in another MRI study of 154 ACL rupture patients between 18–45 years of age.[40,41]

Obesity is a known risk factor for knee OA in older subjects.[1,42–44] In multivariable regression models, higher BMI was associated with 20/40 analyzed MRI parameters. The incidence and severity of knee MRI findings increased incrementally with both BMI classes and height tertiles. However, meniscal findings, popliteal cysts and joint effusion severity increased only with BMI classes, and bodyweight increased more drastically than height according to MRI finding severity. Some results could be driven by a handful of participants with advanced MRI findings and higher BMI. However, BMI was

	Family history of OA (yes)	Male sex	P-Urate (umol/L)	BMI (kg/m^2)	Systolic bp (mmHg)
Cartilage loss					
Tibial medial	0.431 (0.144–1.292)	0.737 (0.268–2.022)	1.001 (0.990–1.012)	1.143 (1.014–1.287)	1.022 (0.975–1.073)
Tibial lateral	0.461 (0.209–1.019)	0.811 (0.341–1.930)	1.006 (1.000–1.012)	1.026 (0.961–1.095)	1.027 (0.998–1.058)
Femoral medial	0.529 (0.314–0.891)	1.121 (0.538–2.335)	1.001 (0.996–1.005)	1.085 (1.029–1.143)	0.998 (0.972–1.025)
Femoral lateral	0.941 (0.330–2.681)	2.611 (0.968–7.046)	0.996 (0.988–1.004)	1.115 (1.002–1.240)	1.025 (0.999–1.051)
Tibiofemoral	0.600 (0.387–0.930)	1.145 (0.634–2.069)	1.001 (0.998–1.005)	1.058 (1.011–1.107)	1.002 (0.981–1.023)
Patellofemoral	0.955 (0.735–1.241)	0.821 (0.589–1.143)	1.000 (0.998–1.002)	1.007 (0.980–1.034)	1.009 (0.998–1.020)
Any	0.902 (0.722–1.125)	0.917 (0.689–1.220)	1.001 (0.999–1.002)	1.009 (0.985–1.033)	1.007 (0.997–1.016)
FT cartilage loss					
Tibial lateral*	9.876 (1.242–78.547)	2.601 (0.214–31.571)	1.017 (1.001–1.034)	0.819 (0.641–1.046)	1.065 (0.972–1.166)
Femoral medial	0.428 (0.094–1.947)	3.186 (0.287–35.403)	0.995 (0.982–1.008)	1.271 (1.021–1.582)	0.953 (0.892–1.018)
Femoral lateral	1.473 (0.263–8.243)	4.837 (1.201–19.486)	1.000 (0.986–1.013)	1.201 (1.018–1.416)	0.961 (0.910–1.015)
Tibiofemoral	0.482 (0.169–1.378)	2.160 (0.537–8.687)	1.002 (0.993–1.011)	1.130 (0.998–1.280)	0.968 (0.923–1.015)
Patellofemoral	1.071 (0.501–2.291)	0.846 (0.334–2.142)	1.004 (1.000–1.009)	1.045 (0.967–1.131)	1.004 (0.975–1.035)
Any	0.737 (0.406–1.337)	1.161 (0.516–2.615)	1.004 (1.000–1.009)	1.054 (0.984–1.130)	0.989 (0.961–1.019)
Size of BML					
Tibial lateral*	12.602 (1.283–123.819)	1.122 (0.062–20.361)	1.023 (1.005–1.040)	0.907 (0.719–1.146)	1.060 (0.961–1.169)
Femoral medial	0.276 (0.057–1.351)	2.981 (0.402–22.088)	0.996 (0.984–1.009)	1.321 (1.121–1.556)	0.969 (0.908–1.034)
Femoral lateral	2.554 (0.304–21.451)	N/A	0.997 (0.988–1.007)	1.083 (0.972–1.208)	1.006 (0.956–1.058)
Tibiofemoral	0.349 (0.119–1.025)	2.408 (0.649–8.936)	0.999 (0.990–1.009)	1.184 (1.063–1.320)	0.997 (0.959–1.038)
Patellofemoral	1.003 (0.458–2.195)	0.873 (0.335–2.279)	1.004 (0.998–1.009)	1.005 (0.914–1.105)	1.015 (0.972–1.060)
Any	0.679 (0.372–1.242)	1.355 (0.599–3.069)	1.003 (0.998–1.007)	1.053 (0.975–1.138)	1.005 (0.972–1.040)
% that is BML					
Tibial lateral*	12.602 (1.283–123.819)	1.122 (0.062–20.361)	1.023 (1.005–1.040)	0.907 (0.719–1.146)	1.060 (0.961–1.169)
Femoral medial	0.540 (0.110–2.652)	4.173 (0.464–37.491)	0.994 (0.978–1.011)	1.277 (1.042–1.565)	0.957 (0.889–1.032)
Femoral lateral	2.706 (0.234–31.326)	9.835 (0.619–156.364)	0.991 (0.976–1.007)	1.200 (1.081–1.333)	0.958 (0.905–1.013)
Tibiofemoral	0.446 (0.154–1.290)	2.450 (0.673–8.916)	1.001 (0.990–1.012)	1.126 (1.006–1.261)	0.991 (0.949–1.035)
Patellofemoral	0.727 (0.324–1.632)	0.769 (0.257–2.298)	1.004 (0.997–1.011)	1.035 (0.947–1.130)	1.003 (0.965–1.043)
Any	0.577 (0.312–1.069)	1.263 (0.552–2.889)	1.004 (0.998–1.009)	1.056 (0.986–1.131)	0.994 (0.965–1.025)
Osteophytes					
Tibial medial	0.555 (0.208–1.477)	1.530 (0.618–3.787)	1.003 (0.994–1.013)	1.186 (1.067–1.319)	0.981 (0.953–1.010)
Tibial lateral	0.553 (0.235–1.302)	1.500 (0.676–3.325)	1.001 (0.991–1.011)	1.146 (1.052–1.249)	0.986 (0.953–1.020)
Femoral medial	0.709 (0.258–1.951)	1.932 (0.861–4.336)	1.003 (0.993–1.012)	1.187 (1.061–1.328)	0.988 (0.957–1.019)
Femoral lateral	0.830 (0.423–1.630)	1.195 (0.617–2.314)	1.000 (0.994–1.006)	1.116 (1.042–1.195)	0.995 (0.969–1.020)
Tibiofemoral	0.909 (0.511–1.620)	1.620 (0.908–2.891)	1.000 (0.994–1.006)	1.123 (1.054–1.198)	0.981 (0.960–1.003)
Patellofemoral	0.830 (0.643–1.070)	1.331 (0.967–1.833)	1.000 (0.998–1.002)	1.034 (1.003–1.066)	1.005 (0.994–1.015)
Any	0.851 (0.671–1.080)	1.382 (1.032–1.851)	1.000 (0.997–1.002)	1.042 (1.014–1.072)	1.002 (0.992–1.012)
Meniscal Tear					
Med. body horizontal*	1.008 (0.278–3.654)	0.497 (0.094–2.621)	0.999 (0.988–1.010)	1.159 (1.003–1.338)	0.984 (0.924–1.047)
Med. posterior horizontal*	1.669 (0.485–5.751)	0.506 (0.094–2.733)	0.995 (0.983–1.007)	1.067 (0.921–1.236)	1.001 (0.942–1.065)
Other					
Patellar tend. signal*	0.727 (0.189–2.802)	2.112 (0.410–10.879)	1.005 (0.994–1.015)	0.990 (0.864–1.135)	1.010 (0.954–1.070)
Any ganglion cyst*	0.654 (0.345–1.239)	1.102 (0.495–2.452)	0.997 (0.992–1.002)	1.008 (0.941–1.081)	1.008 (0.980–1.037)
Inf.pat. bursa signal*	2.692 (1.407–5.151)	1.513 (0.637–3.596)	1.003 (0.998–1.009)	0.949 (0.877–1.026)	1.027 (0.995–1.059)
Prepat. bursa signal*	1.027 (0.602–1.753)	1.642 (0.816–3.307)	1.002 (0.998–1.007)	1.020 (0.960–1.083)	1.005 (0.980–1.031)
Joint effusion	1.190 (0.875–1.619)	1.529 (1.080–2.162)	1.002 (0.999–1.004)	1.061 (1.027–1.097)	0.994 (0.981–1.007)
Popliteal cyst*	1.028 (0.613–1.725)	0.892 (0.458–1.740)	1.000 (0.996–1.005)	1.024 (0.965–1.086)	1.011 (0.986–1.036)

FH, Family History.

* Indicates a logistic regression model and OR. Unmarked parameters were analyzed with Poisson regression and the result is given as RR. Each line represents the model for the given MRI parameter.

Table VI

Osteoarthritis and Cartilage

Relative Risk or Odds Ratios with 95% Confidence Intervals from multivariable regression model for knee MRI findings including all parameters presented.

significantly associated, especially with the MRI findings that were most frequently observed in the data set (cartilage loss and osteophytes), supporting our interpretation that BMI is an overall important factor for knee OA in young and healthy adults.

In unadjusted regression models, Sbp was positively associated with MRI findings, but these associations were likely largely mediated by higher BMI, as Sbp was not associated with MRI findings in the multivariable model. Obesity is a major component of metabolic syndrome characterized by higher blood pressure, insulin resistance, glucose intolerance, dyslipidemia, and systemic inflammation.[45] However, higher P-Urate, an indicator of gout, and associated with metabolic syndrome,[46] was also associated with multiple MRI findings in the multivariable models adjusted for BMI, suggesting a BMI-independent role for P-Urate in structural knee MRI findings.

Of the background factors, a family history of knee OA was associated with tibial lateral full thickness cartilage lesions and increased infrapatellar T2 signal, aligning with current knowledge of family history and genetics being risk factors of OA in general.[47] Interestingly, only male sex was associated with femoral lateral full thickness cartilage lesions, joint effusion and osteophytes in this study population. Female sex is a well-determined risk factor of OA, especially in aging populations.[48] Therefore, although body composition is also subject to genetics, these results suggest that lifestyle factors, such as higher BMI, might outweigh the role of family history in knee OA development in young adulthood.

	Tibiofemoral compartment			Patellofemoral compartment		
	Cartilage loss or Osteophyte or both MOAKS < 1	Cartilage loss and Osteophyte both MOAKS 1	Cartilage loss and Osteophyte both MOAKS ≥ 2	Cartilage loss or Osteophyte or both MOAKS < 1	Cartilage loss and Osteophyte both MOAKS 1	Cartilage loss and Osteophyte both MOAKS ≥ 2
Background characteristics						
Participants n	249	29	10	234	44	10
Males, n (%)	100 (40.2)	8 (27.6)	4 (40.0)	93 (39.7)	15 (34.1)	4 (40.0)
Females, n (%)	149 (59.8)	21 (72.4)	6 (60)	141 (60.3)	29 (65.9)	6 (60)
Age (years)	33.7 (0.4)	33.7 (0.4)	33.8 (0.4)	33.7 (0.4)	33.6 (0.3)	33.8 (0.4)
Prior lower limb fracture, n (%)	32 (12.9)	5 (17.2)	0 (0.0)	34 (14.5)	3 (6.8)	0 (0.0)
Family history of knee OA, n (%)	78 (31.3)	10 (34.5)	5 (50.0)	76 (32.5)	12 (27.3)	5 (50.0)
Anti-inflammatory medication, n (%)	6 (2.4)	0 (0.0)	0 (0.0)	6 (100.0)	0 (0.0)	0 (0.0)
Never smoker, n (%)	92 (36.9)	14 (48.3)	5 (50.0)	87 (37.2)	19 (43.2)	5 (50.0)
Ever smoker, n (%)	157 (63.1)	15 (51.7)	5 (50.0)	147 (62.8)	25 (56.8)	5 (50.0)
Alcohol consumption g/week	31.5 (40.1)	21.4 (21.2)	61.3 (70.2)	30.2 (36.5)	31.6 (49.6)	61.3 (70.2)
Physical activity score	14.7 (3.4)	15.9 (3.1)	15.2 (2.5)	14.7 (3.4)	15.4 (3.0)	15.2 (2.5)
Clinical characteristics						
BMI (kg/m^2)	25.4 (4.5)	27.1 (3.6)	30.6 (5.9)	25.5 (4.3)	25.9 (5.0)	30.6 (5.9)
Height (cm)	170.9 (9.6)	169.9 (7.2)	175.1 (8.4)	171.0 (9.3)	169.3 (9.7)	175.1 (8.4)
Weight (kg)	74.9 (15.2)	78.2 (13.1)	92.7 (15.8)	75.4 (15.1)	74.6 (14.5)	92.7 (15.8)
Waist circumference (cm)	86.7 (15.4)	87.2 (21.6)	97.4 (15.9)	87.7 (14.5)	81.7 (22.6)	97.4 (15.9)
Hip circumference (cm)	97.6 (14.3)	97.4 (22.7)	107.7 (12.3)	98.1 (12.9)	95.1 (25.0)	107.7 (12.3)
Systolic blood pressure (mmHg)	111.8 (12.0)	111.9 (14.8)	118.0 (6.5)	111.9 (12.2)	110.8 (12.9)	118.0 (6.5)
Diastolic blood pressure (mmHg)	74.0 (8.9)	74.2 (8.9)	80.3 (6.0)	74.0 (8.7)	74.5 (10.0)	80.3 (6.0)
Heart rate (bpm)	72.3 (12.5)	70.4 (11.7)	71.4 (8.2)	72.1 (12.7)	72.1 (10.8)	71.4 (8.2)
fP-Glucose (mmol/l)	5.0 (0.6)	5.0 (0.4)	5.1 (0.5)	5.0 (0.6)	5.0 (0.4)	5.1 (0.5)
fP-Total cholesterol (mmol/L)	4.7 (0.9)	4.5 (1.0)	5.0 (0.7)	4.7 (0.9)	4.6 (1.0)	5.0 (0.7)
fP-HDL cholesterol (mmol/L)	1.5 (0.3)	1.5 (0.3)	1.6 (0.4)	1.5 (0.3)	1.4 (0.3)	1.6 (0.4)
fP-LDL cholesterol (mmol/L)	2.8 (0.8)	2.6 (0.9)	3.0 (0.8)	2.7 (0.8)	2.7 (0.9)	3.0 (0.8)
P-Urate (umol/L)	303.2 (67.9)	297.8 (85.0)	353.5 (72.0)	304.8 (69.6)	291.2 (69.8)	353.5 (72.0)
P-Alat (U/l)	26.9 (22.3)	28.2 (18.5)	25.3 (11.0)	27.0 (22.4)	27.3 (19.3)	25.3 (11.0)
hs-CRP (mg/L)	1.7 (3.3)	0.9 (0.7)	2.4 (3.7)	1.7 (3.4)	1.1 (1.6)	2.4 (3.7)

Data is presented as mean (M) and SD for normally-distributed continuous variables, median (Mn) and interquartile range (IQR) for skewed continuous variables and count (n) and percentage (%) for count variables.
M, Mean; SD, Standard Deviation; fP-, fasted plasma; Alat, alanine aminotransferase.

Table VII Osteoarthritis and Cartilage

Background and clinical characteristics of the study population in categories based on MOAKS classification.

The association between minor MRI findings and patellofemoral pain is uncertain, as their incidence is reportedly similar in symptomatic and asymptomatic subjects.[39] Compositional quantitative MRI differences have not been reported either.[49] Although not the focus of this study, we plan to evaluate the imaging and background associations for knee symptoms in this study population.

There are limitations to this study, primarily the infrequency of advanced MRI findings. The prevalence of OA was 2.4% (n =7) for tibiofemoral and 3.5% for patellofemoral (n = 10) joints, based on Hunter's classification.[27] Full-length standing radiographs for assessing axial alignment were unavailable for this study. Although parameters were first analyzed individually and then with multivariable regression models, some residual confounding could exist. The possibility of results being based on chance is higher with a limited sample size. Socioeconomic status was not analyzed, though related lifestyle factors such as smoking and alcohol consumption were investigated. Lower limb injuries other than fractures were not analyzed, though only two ACL tears were observed. Some outcome data was highly skewed and zero-inflated, being suboptimal for Poisson regression. We ran the models with robust estimates and different regression models were tested, including negative binomial regression, and Poisson regression was chosen for multi-classed outcomes due to stable estimates and good correspondence to descriptive data. The key findings of the regression models were verified with further descriptive statistics.

Small osteophytes are reportedly common especially in young adults,[39,40] and may result from physiological bone remodeling rather than early knee OA.[41] A recent study found that progressive cartilage lesions were associated with knee pain development, while stable lesions were not.[50] Without longitudinal studies, the exact role of grade 1 osteophytes and small cartilage lesions in our study population remains unclear, warranting future investigation.

In conclusion, structural knee MRI findings, particularly in patellofemoral joints, were common in our study population of young and asymptomatic adults. Of analyzed parameters, higher BMI was most frequently associated with imaging findings. Although causality isn't demonstrated, our results suggest that lifestyle factors significantly contribute to OA pathogenesis from early adulthood. Longitudinal studies are needed to identify which MRI findings remain stable and which progress into debilitating knee OA.

Role of the funding source

This work was supported by the Research Council of Finland (Flagship of Advanced Mathematics for Sensing Imaging and Modelling grant #359186 and grant #354692). NFBC1986 33–35y follow-up study received financial support from the University of Oulu (Strategic funding from donations) and Oulu University Hospital (K65760).

Author contributions

A.K.: Conceptualization, data curation, writing – original draft, writing – review & editing. J.T.: Conceptualization, formal analysis,

visualization, writing – original draft, writing – review & editing. M.N.: Funding acquisition, resources, writing – review & editing. S.S.: Funding acquisition, resources, writing – review & editing. M.T.N.: Conceptualization, project administration, resources, supervision, writing – review & editing. The corresponding author (A.K.) takes responsibility for the integrity of the research article as a whole.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgments

We wish to thank all cohort members, researchers and NFBC project center personnel who participated in the NFBC data collections.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.joca.2025.04.008.

References

1. GBD 2021 Osteoarthritis Collaborators. Global, regional, and national burden of osteoarthritis, 1990-2020 and projections to 2050: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Rheumatol 2023;5(9):e508–22. https://doi.org/10.1016/S2665-9913(23)00163-7
2. Vina ER, Kwoh CK. Epidemiology of osteoarthritis: literature update. Curr Opin Rheumatol 2018;30(2):160–7. https://doi.org/10.1097/BOR.0000000000000479
3. Graham J, Novosat T, Sun H, Piper BJ, Boscarino JA, Kern MS, *et al*. Medication use and comorbidities in an increasingly younger osteoarthritis population: an 18-year retrospective open-cohort study. BMJ Open 2023;13(5), e067211. https://doi.org/10.1136/bmjopen-2022-067211
4. He Y, Jiang W, Wang W. Global burden of osteoarthritis in adults aged 30 to 44 years, 1990 to 2019: results from the Global Burden of Disease Study 2019. BMC Musculoskelet Disord 2024;25(1):303. https://doi.org/10.1186/s12891-024-07442-w
5. Roemer FW, Eckstein F, Hayashi D, Guermazi A. The role of imaging in osteoarthritis. Best Pract Res Clin Rheumatol 2014;28(1):31–60. https://doi.org/10.1016/j.berh.2014.02.002
6. Guermazi A, Roemer FW, Jarraya M, Hayashi D. A call for screening MRI as a tool for osteoarthritis clinical trials. Skeletal Radiol 2023;52(11):2011–9. https://doi.org/10.1007/s00256-023-04354-z
7. Smith-Bindman R, Miglioretti DL, Larson EB. Rising use of diagnostic medical imaging in a large integrated health system. Health Aff ((Millwood)) 2008;27(6):1491–502. https://doi.org/10.1377/hlthaff.27.6.1491
8. Smith-Bindman R, Kwan ML, Marlow EC, Theis MK, Bolch W, Cheng SY, *et al*. Trends in use of medical imaging in US Health Care Systems and in Ontario, Canada, 2000-2016. JAMA 2019;322(9):843–56. https://doi.org/10.1001/jama.2019.11456
9. Ruonala V., Radiologisten tutkimusten ja toimenpiteiden määrät vuonna 2021: Terveydenhuollon valvontaraportti, STUK, B-295, 2022. http://urn.fi/urn:nbn:fi:att:f5c10eef-3d25-4bd0-beb8-f2d59df95b8e.
10. Eckstein F, Wluka AE, Wirth W, Cicuttini F. 30 Years of MRI-based cartilage & bone morphometry in knee osteoarthritis: From correlation to clinical trials. Osteoarthr Cartil 2024;32(4):439–51. https://doi.org/10.1016/j.joca.2024.02.002
11. Aitken D, Balogun S, Foong YC, Humphries D, Laslett L, Pitchford N, *et al*. Clinical relevance of MRI knee abnormalities in Australian rules football players: a longitudinal study. BMJ Open Sport Exerc Med 2021;7(3), e001097. https://doi.org/10.1136/bmjsem-2021-001097
12. Cristiani R, van de Bunt F, Kvist J, Stålman A. High prevalence of associated injuries in anterior cruciate ligament tears: A detailed magnetic resonance imaging analysis of 254 patients. Skeletal Radiol 2024;53(11):2417–27. https://doi.org/10.1007/s00256-024-04665-9
13. Danieli MV, Guerreiro JP, Queiroz Ad, Pereira Hd, Tagima S, Marini MG, *et al*. Diagnosis and classification of chondral knee injuries: comparison between magnetic resonance imaging and arthroscopy. Knee Surg Sports Traumatol Arthrosc 2016;24(5):1627–33. https://doi.org/10.1007/s00167-015-3622-8
14. Friel NA, Chu CR. The role of ACL injury in the development of posttraumatic knee osteoarthritis. Clin Sports Med 2013;32(1):1–12. https://doi.org/10.1016/j.csm.2012.08.017
15. Hart HF, Crossley KM, Patterson BE, Guermazi A, Birmingham TB, Koskoletos C, *et al*. Adiposity and cartilage lesions following ACL reconstruction. Osteoarthr Cartil 2024;32(7):931–6. https://doi.org/10.1016/j.joca.2024.04.004
16. Singh A, Venn A, Blizzard L, March L, Eckstein F, Jones G, *et al*. Association between knee magnetic resonance imaging markers and knee symptoms over 6-9 years in young adults. Rheumatology ((Oxford)) 2024;63(2):436–45. https://doi.org/10.1093/rheumatology/kead227
17. Sowers MF, Hayes C, Jamadar D, Capul D, Lachance L, Jannausch M, *et al*. Magnetic resonance-detected subchondral bone marrow and cartilage defect characteristics associated with pain and X-ray-defined knee osteoarthritis. Osteoarthr Cartil 2003;11(6):387–93. https://doi.org/10.1016/s1063-4584(03)00080-3
18. Järvelin MR, Elliott P, Kleinschmidt I, Martuzzi M, Grundy C, Hartikainen AL, *et al*. Ecological and individual predictors of birthweight in a northern Finland birth cohort 1986. Paediatr Perinat Epidemiol 1997;11(3):298–312. https://doi.org/10.1111/j.1365-3016.1997.tb00007.x
19. University of Oulu. Northern Finland Birth Cohort. Oulu, Finland: University of Oulu; 1986. http://urn.fi/urn:nbn:fi:att:bc1e5408-980e-4a62-b899-43bec3755243.
20. Soininen JV, Paavolainen PO, Gronblad MA, Kaapa EH. Validation study of a Finnish version of the Western Ontario and McMasters University osteoarthritis index. Hip Int 2008;18(2):108–11. https://doi.org/10.1177/112070000801800207
21. Roos EM, Klässbo M, Lohmander LS. WOMAC Osteoarthritis Index: Reliability, validity, and responsiveness in patients with arthroscopically assessed osteoarthritis. Western Ontario and MacMaster Universities. Scand J Rheumatol 1999;28(4):210–5. https://doi.org/10.1080/03009749950155562
22. Kapstad H, Hanestad BR, Langeland N, Rustøen T, Stavem K. Cutpoints for mild, moderate and severe pain in patients with osteoarthritis of the hip or knee ready for joint replacement surgery. BMC Musculoskelet Disord 2008;9(1):55. https://doi.org/10.1186/1471-2474-9-55
23. Hunter DJ, Guermazi A, Lo GH, Grainger AJ, Conaghan PG, Boudreau RM, *et al*. Evolution of semi-quantitative whole joint assessment of knee OA: MOAKS (MRI Osteoarthritis Knee Score). Osteoarthr Cartil 2011;19(8):990–1002. https://doi.org/10.1016/j.joca.2011.05.004
24. Roemer FW, Kwoh CK, Hannon MJ, Hunter DJ, Eckstein F, Wang Z, *et al*. Can structural joint damage measured with MR imaging be used to predict knee replacement in the following year? Radiology 2015;274(3):810–20. https://doi.org/10.1148/radiol.14140991

25. WHO. Obesity and overweight fact sheet. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight. [Accessed August 10, 2024].
26. Liew JW, Rabasa G, LaValley M, Collins J, Stefanik J, Roemer FW, *et al*. Development of a magnetic resonance imaging-based definition of knee osteoarthritis: Data from the Multicenter Osteoarthritis Study. Arthritis Rheumatol 2023;75(7):1132–8. https://doi.org/10.1002/art.42454
27. Hunter DJ, Arden N, Conaghan PG, Eckstein F, Gold G, Grainger A, *et al*. Definition of osteoarthritis on MRI: results of a Delphi exercise. Osteoarthr Cartil 2011;19(8):963–9. https://doi.org/10.1016/j.joca.2011.04.017
28. Krajnc Z, Vogrin M, Recnik G, Crnjac A, Drobnic M, Antolic V. Increased risk of knee injuries and osteoarthritis in the non-dominant leg of former professional football players. Wien Klin Wochenschr 2010;122(2):40–3. https://doi.org/10.1007/s00508-010-1341-1
29. Kuijt MTK, Inklaar H, Gouttebarge V, Frings-Dresen MHW. Knee and ankle osteoarthritis in former elite soccer players: a systematic review of the recent literature. J Sci Med Sport 2012;15(6):480–7. https://doi.org/10.1016/j.jsams.2012.02.008
30. Lee HH, Chu CR. Clinical and Basic Science of Cartilage Injury and Arthritis in the Football (Soccer) Athlete. Cartilage 2012;3(1):63S–8S. https://doi.org/10.1177/1947603511426882
31. Snoeker B, Turkiewicz A, Magnusson K, Frobell R, Yu D, Peat G, *et al*. Risk of knee osteoarthritis after different types of knee injuries in young adults: a population-based cohort study. Br J Sports Med 2020;54(12):725–30. https://doi.org/10.1136/bjsports-2019-100959
32. Driban JB, Hootman JM, Sitler MR, Harris KP, Cattano NM. Is participation in certain sports associated with knee osteoarthritis? A systematic review. J Athl Train 2017;52(6):497–506. https://doi.org/10.4085/1062-6050-50.2.08
33. Alentorn-Geli E, Samuelsson K, Musahl V, Green CL, Bhandari M, Karlsson J. The association of recreational and competitive running with hip and knee osteoarthritis: A systematic review and meta-analysis. J Orthop Sports Phys Ther 2017;47(6):373–90. https://doi.org/10.2519/jospt.2017.7137
34. Schram B, Orr R, Pope R, Canetti E, Knapik J. Risk factors for development of lower limb osteoarthritis in physically demanding occupations: A narrative umbrella review. J Occup Health 2020;62(1), e12103. https://doi.org/10.1002/1348-9585.12103
35. Arøen A, Løken S, Heir S, Alvik E, Ekeland A, Granlund OG, *et al*. Articular cartilage lesions in 993 consecutive knee arthroscopies. Am J Sports Med 2004;32(1):211–5. https://doi.org/10.1177/0363546503259345
36. Campbell AB, Knopp MV, Kolovich GP, Wei W, Jia G, Siston RA, *et al*. Preoperative MRI underestimates articular cartilage defect size compared with findings at arthroscopic knee surgery. Am J Sports Med 2013;41(3):590–5. https://doi.org/10.1177/0363546512472044
37. Figueroa D, Calvo R, Vaisman A, Carrasco MA, Moraga C, Delgado I. Knee chondral lesions: incidence and correlation between arthroscopic and magnetic resonance findings. Arthroscopy 2007;23(3):312–5. https://doi.org/10.1016/j.arthro.2006.11.015
38. Lahm A, Spank H, Zilkens C, Kasch R. Comparison of different methods of semiquantitative assessment and subjective scores for retropatellar articular cartilage evaluation in advancing osteoarthritis. Ortop Traumatol Rehabil 2023;25(6):297–305. https://doi.org/10.5604/01.3001.0054.2881
39. van der Heijden RA, de Kanter JL, Bierma-Zeinstra SM, Verhaar JA, van Veldhoven PL, Krestin GP, *et al*. Structural abnormalities on magnetic resonance imaging in patients with patellofemoral pain: A cross-sectional case-control study. Am J Sports Med 2016;44(9):2339–46. https://doi.org/10.1177/0363546516646107
40. van Meer BL, Oei EH, Bierma-Zeinstra SM, van Arkel ER, Verhaar JA, Reijman M, *et al*. Are magnetic resonance imaging recovery and laxity improvement possible after anterior cruciate ligament rupture in nonoperative treatment? Arthroscopy 2014;30(9):1092–9. https://doi.org/10.1016/j.arthro.2014.04.098
41. De Kanter JLM, Oei EHG, Schiphof D, Van Meer BL, Van Middelkoop M, Reijman M, *et al*. Prevalence of small osteophytes on knee MRI in several large clinical and population-based studies of various age groups and OA risk factors. Osteoarthr Cartil Open 2021;3(3), 100187. https://doi.org/10.1016/j.ocarto.2021.100187
42. Hart HF, van Middelkoop M, Stefanik JJ, Crossley KM, Bierma-Zeinstra S. Obesity is related to incidence of patellofemoral osteoarthritis: the Cohort Hip and Cohort Knee (CHECK) study. Rheumatol Int 2020;40(2):227–32. https://doi.org/10.1007/s00296-019-04472-9
43. Laberge MA, Baum T, Virayavanich W, Nardo L, Nevitt MC, Lynch J, *et al*. Obesity increases the prevalence and severity of focal knee abnormalities diagnosed using 3T MRI in middle-aged subjects–data from the Osteoarthritis Initiative. Skeletal Radiol 2012;41(6):633–41. https://doi.org/10.1007/s00256-011-1259-3
44. Kumm J, Turkiewicz A, Zhang F, Englund M. Structural abnormalities detected by knee magnetic resonance imaging are common in middle-aged subjects with and without risk factors for osteoarthritis. Acta Orthop 2018;89(5):535–40. https://doi.org/10.1080/17453674.2018.1495164
45. Alberti KGMM, Zimmet P, Shaw J. IDF Epidemiology Task Force Consensus Group. The metabolic syndrome–a new worldwide definition. Lancet 2005;366(9491):1059–62. https://doi.org/10.1016/S0140-6736(05)67402-8
46. Kanbay M, Jensen T, Solak Y, Le M, Roncal-Jimenez C, Rivard C, *et al*. Uric acid in metabolic syndrome: From an innocent bystander to a central player. Eur J Intern Med 2016;29:3–8. https://doi.org/10.1016/j.ejim.2015.11.026
47. Boer CG, Hatzikotoulas K, Southam L, Stefánsdóttir L, Zhang Y, Coutinho de Almeida R, *et al*. Deciphering osteoarthritis genetics across 826,690 individuals from 9 populations. Cell 2021;184(18):4784–4818.e17. https://doi.org/10.1016/j.cell.2021.07.038
48. Srikanth VK, Fryer JL, Zhai G, Winzenberg TM, Hosmer D, Jones G. A meta-analysis of sex differences prevalence, incidence and severity of osteoarthritis. Osteoarthr Cartil 2005;13(9):769–81. https://doi.org/10.1016/j.joca.2005.04.014
49. van der Heijden RA, Oei EH, Bron EE, van Tiel J, van Veldhoven PL, Klein S, *et al*. No difference on quantitative magnetic resonance imaging in patellofemoral cartilage composition between patients with patellofemoral pain and healthy controls. Am J Sports Med 2016;44(5):1172–8. https://doi.org/10.1177/0363546516632507
50. Joseph GB, Hou SW, Nardo L, Heilmeier U, Nevitt MC, McCulloch CE, *et al*. MRI findings associated with development of incident knee pain over 48 months: data from the osteoarthritis initiative. Skeletal Radiol 2016;45(5):653–60. https://doi.org/10.1007/s00256-016-2343-5