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Lower limb clinical and radiographic osteoarthritis in former elite male athletes

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Abstract
Purpose To investigate the prevalence of lower extremities clinical and radiographic OA in former elite male athletes and referents from the general population and to examine its association with the participants' demographic characteristics.
Methods Two hundred and eighteen former elite male athletes (soccer, volleyball, martial arts, track and field and basketball players, and skiers) and 181 male controls that reported no systematic athletic activity were examined by means of questionnaire, clinical and radiographic evaluation. Exclusion criteria were age younger than 40 years and a positive history of lower extremity surgery, bone or soft tissue trauma and inflammatory arthropathy.
Results Overall, the prevalence of clinical OA between former elite athletes (15.6 %) and controls (14.4 %) was similar (n.s.). The prevalence of radiographic OA was significantly higher ($p = 0.03$) in former elite athletes (36.6 %) compared with controls (23.9 %). All the participants with clinical OA who underwent radiographic examination also had radiographic OA. The prevalence of clinical and radiographic OA was similar (n.s.) between former athletes of different sports. Age, body mass index (BMI) and occupation variably predicted the prevalence of hip, knee and ankle OA in both study groups.
Conclusions In the absence of major bone and soft tissue lower limb trauma during their athletic career, former elite athletes may not be at increased risk of developing clinical OA. Radiographic signs of OA present at a significantly higher incidence and possibly precede the clinical onset of OA. Age, BMI and occupation are identified as strong predictors of the development of OA in former elite athletes.
Level of evidence Case–control prognostic study, Level III.

Keywords Osteoarthritis · Former elite athletes · Hip · Knee · Ankle

Introduction

Athletic activity has become a significant component of healthy lifestyle nowadays [12]. As a consequence, the number of professional and recreational athletes in all kinds of sports is rising [7]. However, the association between sports and OA currently remains controversial. Participation in load-bearing and impact sports is considered to place athletes at risk of suffering injuries, commonly to the lower extremities [27]. Even without injury, repetitive joint loading is a possible cause of chondral lesions and subsequent OA [6, 16, 22, 40]. In contrast, the effect of moderate impact and torsional joint loading on the risk of OA in athletes with a normal musculoskeletal system remains unclear [36].

To date, experimental, epidemiological and clinical studies have addressed the hypothesis that mechanical joint loading during sport participation induces or accelerates

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cartilage degeneration in load-bearing joints and have produced overall contradictive results [30, 39]. In general, the individual predisposition for degenerative joint disease, differences in training models and variations in injury patterns make it difficult to establish a clear relationship between athletic activity and the occurrence of OA [6, 17, 28, 32, 34]. Although several studies have supported the association between joint injury and degeneration [5], the prevalence of osteoarthritis in the absence of significant joint trauma has scarcely been examined. In addition, the current evidence base presents significant variability regarding the nature of sport involved, level of subject participation (professional or amateur), occupation and study design. Clinical studies, in particular, have seldom performed a comprehensive assessment of sport-induced OA by combining medical records, clinical and radiographic examination. Among the athletic population, elite professional athletes are realistically subjected to the highest impact of repetitive lower limb joint loading. Therefore, comparing former elite athletes with referents of non-systematic athletic activity would be expected to better elicit differences, if present, in OA prevalence and consequently establish in full scale the effect of sports on lower limb OA.

Although clinical and radiographic signs of OA are actually part of a single biologic abnormality, in clinical practice, they often have a diverse occurrence in the course of OA disease. Therefore, the purpose of this study was to investigate the prevalence of lower extremities clinical and radiographic OA in a comparatively large number of former elite athletes and controls from the general population and examine its association with their demographic characteristics, in the absence of major bone or soft tissue joint injury. A questionnaire, clinical and radiographic evaluation were combined to provide a more valid assessment of the occurrence of lower limb clinical and radiographic OA. It was hypothesized that, in this setting, the prevalence of lower limb OA would be significantly higher in former elite athletes compared with controls.

Materials and methods

Former elite male athletes were recruited through athletic associations of soccer, skiing, volleyball, martial arts, basketball and track and field. Male athletes were identified who had participated in national and at least once in World or European championships or international competitions during their career and remained active at least until the age of 25. The control group consisted of male participants from the same geographical district who had been classified as completely healthy at the time of their compulsory military service (approximately at the age of 20 years) and reported no systematic athletic activity [13, 33]. Exclusion criteria for the two groups were age younger than 40 years and a positive history of lower extremities surgery, bone or soft tissue trauma (fracture, dislocation, meniscal, chondral or ligamentous injuries) or inflammatory arthropathy.

Study design

The study was performed using a questionnaire, clinical and radiographic examination. The questionnaire was intended to collect demographic and lifestyle data and rule out lower extremity soft tissue or bone trauma. It included items on physician-diagnosed hip, knee and ankle OA (yes/no). The onset of OA before and after the age of 45 years, the need for hospital admission, physician-diagnosed major musculoskeletal injury, current body mass index (BMI) and BMI at the age of 20 were recorded [8, 18]. Activity level at the time of the survey was recorded using the Tegner activity score. The current occupation was classified by the investigators into five grades: (1) mainly sitting; (2) mainly walking and standing; (3) mixed, including a variety of tasks with bending and twisting but seldom lifting heavier than 35 kg; (4) heavy, including a variety of tasks with bending, twisting and daily lifting of materials over 35 kg; and (5) continuing motion with frequent maximal lifts [18, 31].

Clinical examination was performed by the senior investigator (M.E.I.) and focused on joint range of motion, pain, crepitus and malalignment. All measurements were performed according to the AAOS guidelines [1, 23]. The participants were considered to have clinical osteoarthritis, when the questionnaire revealed considerable joint pain or disability during the preceding year, and at least one additional clinical sign of OA was positive [2, 23]. Lower extremities radiographs were obtained in the standing position and classified according to the Kellgren and Lawrence classification scale. An independent examiner, blinded to the participant's clinical status, determined the presence of joint space narrowing, osteophytes, subchondral sclerosis and cysts. Positive radiographic joint arthritis was defined as grade 2 or higher in the Kellgren and Lawrence classification scale [37]. All the participants signed an informed consent form approved by the Aristotle University of Thessaloniki Institutional Review Board (ID No. 353-22/6/2007) to participate in the study. The study was performed according to the STROBE guidelines for case–control studies.

Statistical analysis

All data analyses were performed using the Statistical Package for Social Sciences SPSS version 15 (IBM Corp., Armonk, NY). A priori power analysis was performed to

calculate sample size. A total sample of 300 was found to produce $a > 0.80$ for two independent proportions of 0.35 and 0.25, and $a > 0.60$ for proportions of 0.15 and 0.14. The independent-samples t test and Mann–Whitney test were used to examine differences in anthropometric and demographic characteristics of the study and control groups. To examine differences in OA prevalence, either clinical or radiographic, between the two groups, the z test was used. Binary logistic regression analysis was performed to examine the extent to which epidemiologic factors account for the prevalence of lower extremity clinical and radiographic OA. Three models were used in the primary analysis: adjusted for age; age and BMI; age, BMI and occupation accordingly. The significance level was set at $p < 0.05$.

Results

Figure 1 shows the disposition of the study participants. The questionnaire was distributed to 391 former elite athletes and 313 controls. The response rate was 84.4 and 86.6 %, respectively. After excluding those who did not meet the inclusion criteria, clinical examination was performed on 218 former athletes and 181 controls. Radiographs of the lower extremities were obtained from 172 and 163 participants, respectively.

Table 1 Baseline characteristics (mean ± SD) of the study and control groups

	Former athletes ($n = 218$)		Control group ($n = 181$)
Age (years)	50.1 ± 8.5	n.s.	50.7 ± 10
Height (cm)	179.6 ± 6.8	n.s.	178.5 ± 7.5
Weight (kg)	86.4 ± 10.6	n.s.	86.4 ± 6.5
Weight 20 years (kg)	72 ± 8*	0.01	68.8 ± 5.4*
BMI	26.7 ± 2.5	n.s.	27.1 ± 1.2
BMI at 20 years	22.3 ± 1.6*	0.01	21.5 ± 1.6*
Activity level (Tegner)[a]	8 (7–10)*	0.005	3 (1–5)*
Occupation (mean)	2.3	–	2.4
Category 1/2/3/4–5	27/108/63/20		48/46/57/30
Hours of weekly training	12.5 ± 4.8	–	–
Years of sports participation	15.3 ± 6.3	–	–

* Significant at the 0.01 level

[a] Median (range), Mann–Whitney test

Table 1 shows the baseline characteristics of the two groups. Age, BMI and occupational level between the two groups were similar (n.s.). As expected, former athletes had a significantly higher Tegner activity score compared with controls ($p = 0.001$). A significant difference ($p = 0.01$)

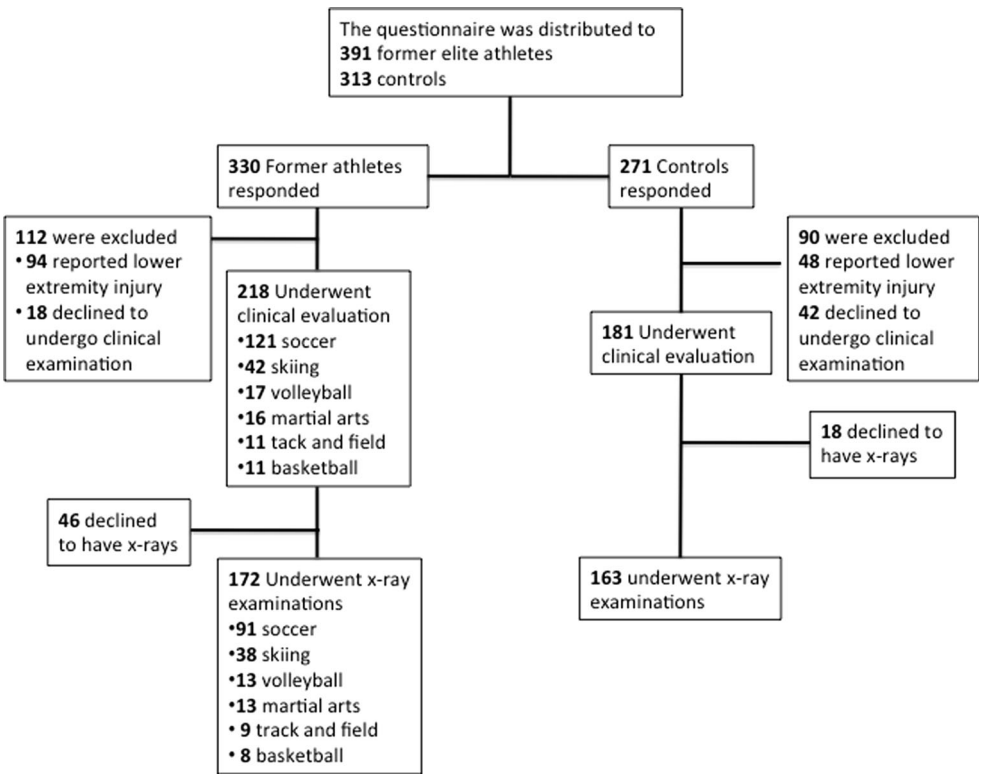

Fig. 1 Flowchart that shows the disposition of the study participants

was also observed in BMI at the age of 20, indicating a more rapid increase in body weight over the years for the control group.

Prevalence of clinical and radiographic OA

The prevalence of clinical and radiographic signs of OA in the two groups is depicted in Table 2. Clinical lower limb OA was evident in 34 former elite athletes (15.6 %). The hip, knee and ankle joints were affected in 3.2, 8.7 and 3.6 % of the cases, respectively. Crepitus was the commonest finding for the knee and ankle joints, while ROM deficit was commonest for the hip joint. Overall, the prevalence of clinical OA between former elite athletes and controls (14.4 %) was similar (n.s.).

The prevalence of radiographic OA was significantly higher in former elite athletes compared with controls (36.6 and 23.9 %, respectively, $p = 0.03$, Table 2). Radiographic OA was established in all the participants with clinical OA who underwent radiographic examination. Osteophyte formation was the commonest finding followed by subchondral sclerosis. Compared with controls, knee radiographic OA was significantly higher in former elite athletes ($p = 0.048$). In particular, hip and knee osteophytes and subchondral sclerosis as well as subchondral cyst formation were significantly more common in former athletes than controls ($p = 0.045$).

In respect of the type of athletic activity, the prevalence of either clinical or radiographic OA was similar (n.s.) between the six subgroups of former elite athletes (Table 3). Knee radiographic OA was significantly more common in soccer and basketball players compared with controls, whereas clinical hip OA showed a significantly higher prevalence in skiers ($p = 0.045$).

Correlation with demographic characteristics

The estimated odds ratios (ORs) and 95 % confidence intervals (CIs) for clinical and radiographic OA in respect of age, BMI and occupational level are depicted in Table 4. In the former elite athletes group, BMI significantly affected the prevalence of radiographic OA ($p = 0.01$). In addition, former elite athletes older than 45 years had significantly higher odds to develop radiographic OA ($p = 0.05$). Regarding controls, age and occupation independently predicted the occurrence of clinical and radiographic OA ($p = 0.02$).

Table 5 presents the ORs and 95 % CIs separately for hip, knee and ankle joint OA after adjusting for age, BMI and occupation. In the former elite athletes group, BMI and occupation significantly explained the prevalence of clinical OA of the hip joint ($p = 0.05$). In the control group, age significantly affected the prevalence of clinical hip and knee OA ($p = 0.02$ and $p = 0.01$, respectively) and radiographic hip OA ($p = 0.001$). Similarly, BMI significantly affected the prevalence of clinical hip OA ($p = 0.045$), whereas occupation significantly predicted clinical and radiographic hip and knee OA ($p = 0.05$) in controls.

Table 2 Prevalence of clinical and radiographic signs of osteoarthritis in the two study groups

	Former athletes ($n = 218$)				Control group ($n = 182$)			
	Hip	Knee	Ankle	Total	Hip	Knee	Ankle	Total
Clinical OA	7	19	8	34	6	15	5	26
ROM deficit	26	30	21		21	23	15	
Pain	21	17	12		16	12	9	
Crepitus	5	42	32		3	29	21	
Malalignment	2	32	1		1	14	0	
	Former athletes ($n = 172$)				Control group ($n = 163$)			
	Hip	Knee	Ankle	Total	Hip	Knee	Ankle	Total
Radiographic OA	16	35*	12	63*	12	21	6	39
Osteophytes	45*	110*	79		20	84	64	
Narrowing	21*	56*	18		13	43	11	
Sclerosis	41	77	64		21	58	61	
Subchondral cysts	8	9*	5		4	2	9	

OA osteoarthritis, *ROM* range of motion

* Significant at the 0.05 level compared with the control group

Table 3 Prevalence of clinical and radiographic OA in respect of the type of athletic activity

	Soccer ($n = 121$)	Skiing ($n = 42$)	Volleyball ($n = 17$)	Martial arts ($n = 16$)	Track and field ($n = 11$)	Basketball ($n = 11$)
Clinical OA (%)	19 (15.7)	8 (19)	2 (11.7)	2 (12.5)	2 (18.2)	1 (9.1)
Hip	3	4	0	0	0	0
Knee	11	4	1	1	1	1
Ankle	5	0	1	1	1	0
Radiographic OA (%)	37 (40.6)	13 (34.2)	4 (30.8)	3 (23.1)	2 (22.2)	3 (37.5)
Hip	9	6	0	0	0	0
Knee	20	7	2	1	1	3
Ankle	8	0	2	2	1	0

Table 4 Odds ratios and 95 % CIs for clinical and radiographic OA among former elite athletes and controls

	Former elite athletes group						Control group					
	Clinical OA			Radiographic OA			Clinical OA			Radiographic OA		
	OR	95 % CI	p value	OR	95 % CI	p value	OR	95 % CI	p value	OR	95 % CI	p value
Age			n.s.			n.s.			**0.02***			**0.01****
<45	1.00			1.00			1.00			1.00		
45–51	0.96	0.36–2.56	n.s.	0.44	0.19–1.00	**0.05***	0.11	0.03–0.41	**0.02***	0.16	0.05–0.51	**0.02***
>51	1.57	0.59–4.13	n.s.	0.66	0.29–1.54	n.s.	0.22	0.06–0.86	**0.03***	0.52	0.19–1.49	n.s.
BMI			n.s.			**0.01****			n.s.			n.s.
<25	1.00			1.00								
25–28	1.24	0.48–3.2	n.s.	1.71	0.70–4.15	n.s.	1.00			1.00		
>28	0.60	0.23–1.56	n.s.	0.34	0.15–0.76	**0.01****	0.78	0.27–2.29	n.s.	1.07	0.40–2.92	n.s.
Occupation			n.s.			n.s.			**<0.001****			**<0.001****
1	1.00			1.00			1.00			1.00		
2	0.39	0.06–2.47	n.s.	0.68	0.10–4.74	n.s.	0.03	0.03–0.25	**0.01****	0.04	0.01–0.19	**<0.001****
3	0.25	0.06–1.06	**0.06***	0.48	0.10–2.39	n.s.	0.10	0.03–0.41	**0.01****	0.07	0.02–0.24	**<0.001****
4–5	0.46	0.10–2.03	n.s.	0.35	0.07–1.78	n.s.	0.12	0.04–0.41	**0.01****	0.10	0.03–0.33	**<0.001****

Significant p values are highlighted in bold

* Significant at the 0.05 level

** Significant at the 0.01 level

Discussion

The most important finding of the present study was that, when participants with a known history of joint trauma or instability were excluded, clinical lower limb OA was similar between groups whereas radiographic OA was significantly more prevalent in former elite athletes. The prevalence of OA did not differ significantly between former elite athletes of different sports.

The current evidence base regarding the association of athletic activity and OA presents considerable discrepancies in methodology. Consequently, they may well have underestimated the true incidence of early osteoarthritic symptoms. Similarly, self-reported questionnaires, commonly distributed by mail, lack the benefit of objectively confirming and staging the disease. However, a larger number of participants can be included. To date, studies combining questionnaires, clinical and radiographic examination have scarcely been presented and were confined to a limited number of participants. Therefore, we believe that our methodology offered a considerable advantage by identifying different clinical and radiographic stages of the disease in a comparatively large number of former elite athletes, whereas cases of major bone or soft tissue lower limb trauma were excluded through preliminary screening via questionnaire. It should be noted that compared with plain radiography, magnetic resonance imaging (MRI) offers advanced imaging of the joints without exposure to radiation. However, because of the significantly higher cost of MRI and longer duration of the

Table 5 Odds ratios and 95 % CIs for clinical and radiographic OA of the hip, knee and ankle joints after adjusting for age, BMI and occupation

	Adjusted for age			Adjusted for age and BMI			Adjusted for age, BMI and occupation		
	OR	95 % CI	*p* value	OR	95 % CI	*p* value	OR	95 % CI	*p* value
Clinical									
Hip									
FEA	1.033	0.95–1.18	n.s.	1.31	0.99–1.73	**0.05***	2.26	0.98–5.28	**0.05***
C	1.17	1.06–1.29	**0.02***	2.55	1.02–6.37	**0.045***	3.28	1.03–10.45	**0.045***
Knee									
FEA	1.012	0.96–1.07	n.s.	0.95	0.78–1.15	n.s.	1.49	0.81–2.73	n.s.
C	1.06	1.01–1.12	**0.01****	1.11	0.69–1.79	n.s.	2.42	1.39–4.21	**0.002****
Ankle									
FEA	0.96	0.87–1.06	n.s.	1.02	0.77–1.35	n.s.	0.86	0.31–2.4	n.s.
C	0.99	0.91–1.09	n.s.	0.86	0.38–1.96	n.s.	1.4	0.64–3.05	n.s.
Radiographic									
Hip									
FEA	1.02	0.96–1.08	n.s.	1.16	0.95–1.41	n.s.	1.39	0.71–2.73	n.s.
C	1.1	1.04–1.16	**0.001****	1.34	0.78–2.3	n.s.	1.88	0.99–3.52	**0.05***
Knee									
FEA	1.03	0.99–1.08	n.s.	0.96	0.79–1.08	n.s.	1.14	0.67–1.92	n.s.
C	1.04	0.99–1.08	n.s.	1.05	0.70–1.57	n.s.	2.11	1.33–3.36	**0.002****
Ankle									
FEA	1.01	0.95–1.08	n.s.	1.01	0.79–1.27	n.s.	0.52	0.21–1.32	n.s.
C	0.98	0.90–1.07	n.s.	0.84	0.39–1.77	n.s.	1.63	0.79–3.36	n.s.

Significant *p* values are highlighted in bold

FEA former elite athletes, *C* controls

* Significant at the 0.05 level

** Significant at the 0.01 level

examination, a combination of clinical and radiographic examination of lower limb joints was selected.

The prevalence of clinical OA was found to be similar between former elite athletes and controls. There is still significant controversy regarding the association of high intensity athletic activity and osteoarthritis. Konradsen et al. [15] compared a group of 30 ex-competitive long distance runners with no history of severe limb injury with 27 matched controls through questionnaire, clinical and radiographic examination. Their results showed no significant difference in OA prevalence between groups. Interestingly, a lower rate of radiographic knee OA and joint complaints in the study group was reported by White et al. [41] when comparing female physical education teachers with a matched control group. Other studies, however, have advocated the impact of systematic sport activity on OA development [9]. Lindberg et al. [24] and Roos et al. [32] compared 286 former soccer players with a mean age of 55 years with an age-matched control group and found an increased prevalence of OA signs on hip and knee radiographs.

Findings of this study demonstrated a significantly higher prevalence of radiographic compared with clinical signs of OA in both groups. This finding is consistent with previous studies. Lane et al. [19–21] examined the progression of radiographic OA of the hips and knees in 28 runners and 27 non-runner controls at 2, 5 and 9-year follow-up. They found that both runners and controls showed a progressive increase in radiographic signs of OA. However, differences between the two groups were not significant. Osteophyte formation as part of the bone remodelling process is an early radiographic finding and may precede cartilage degeneration. In a study on active high-level teenage athletes, Auberge et al. [3] recorded obvious radiographic alterations including bone age delay, epiphyseal plate thinning or thickening, dystrophic metaphyseal cysts, subchondral epiphyseal osteocondensations and flattening of the wrist epiphysis. The prognostic value of these data is still unknown. Schmitt et al. [35] found only a slight decrease in hip function of former elite javelin throwers and high jumpers at an average 20 years after retirement despite considerable radiographic degenerative changes. It is unclear whether enhanced muscle training and proprioception allows athletes to continue nearly normal function of their joints despite progressive and radiographically apparent degeneration. Although there is

evidence that radiographic signs are an early indication of OA, most authors agree that they cannot solely determine joint OA.

The current evidence base has identified bone and ligamentous joint injury as one of the strongest predictors of the occurrence of OA in the athletic population [25, 38]. The risk of articular cartilage degeneration may be increased by joint instability resulting from ligament rupture, by altering the congruence of opposing articular surfaces and increasing the magnitude of impact loading, shear and compression forces in the joint. Apart from direct trauma and instability, joint degeneration during top-level athletic activity is considered to occur through repetitive or torsional loading. However, experimental studies have shown that the articular cartilage can withstand substantial impact loads without apparent damage. Buckwalter and Woo demonstrated in vitro an anabolic response by chondrocytes to moderate mechanical loading in specific rates, whereas cell degeneration was only induced when the loading rate and magnitude were significantly increased [4]. Our data indicated that when major joint injury and instability were excluded, former elite athletes were not at increased risk of developing clinical OA, which is consistent with the hypothesis of joint adaptation to repetitive loading and motion. However, diagnosing injuries of the articular cartilage or subchondral bone is challenging. Because articular cartilage lacks innervation and clinical as well as imaging examinations may be inconclusive, occult joint injury is often under-diagnosed and may deteriorate with unrestricted activity. Microtrauma or subclinical joint instability (for example partial ACL insufficiency) may trigger slight alterations of joint function that may hardly be perceived clinically but have substantial long-term effects. We believe that distinguishing between the consequences of impact loading and joint injuries during sport participation is essential for achieving a better understanding of the association between sports and OA.

The association between lower limb OA in the general population and demographic characteristics has been examined before. Large epidemiologic cohorts have documented strong associations of hip and knee OA with factors like age, BMI and occupation [10]. Increased BMI has been correlated with the occurrence of hip and knee OA both through hospital admission and joint replacement rates [11, 26]. Metabolic factors, in addition to the mechanical effect of obesity on the development of OA, have recently been investigated by studies focusing on metabolic active adipose tissue [29]. Studies on former elite athletes have also identified correlations between these factors and the occurrence of OA. Kettunen et al. [14] found decreased hip rotation values in former elite athletes with increased BMI. A higher incidence of hip and knee disability in former elite male athletes with increased BMI has also been established along with a significant increase in the odds of developing OA with increasing age [13]. Findings of this study also confirmed a variable impact of these factors on clinical and radiographic signs of hip and knee OA in former elite athletes. Interestingly, stronger associations were observed in controls. It is unclear, however, if this can be attributed to a protective effect of systematic athletic activity on joint degeneration, possibly due to enhanced joint proprioception, as suggested by previous studies that advocated the hypothesis of joint adaptation to repetitive loading.

There are certain limitations in our study. First, although the total number of former elite athletes included in the study was comparatively large, the number of participants in certain sports was not adequate to allow for an analysis of OA outcomes between individual subgroups. A second limitation is that the absence of significant lower limb joint trauma was determined solely by means of questionnaire and medical records. Although evidently a direct appraisal of joint injury through arthroscopic visualization or MRI would be more accurate, this would hardly be feasible in our cohort. Clearly, as in most studies of this nature, recall and response bias may have influenced the data collection process.

Conclusion

The primary hypothesis of the study was partially confirmed. Although clinical and radiographic signs of OA are actually part of a single biologic abnormality, in day-by-day clinical practice, they often have a diverse appearance in the course of OA disease and consequently pose diagnostic and prognostic dilemmas to the clinician. Viewed in this context, our findings suggest that in the absence of major bone and soft tissue lower limb trauma during their athletic career, former elite athletes may not be at increased risk of developing clinical signs of OA. Radiographic signs of OA present in former elite athletes at a higher incidence and possibly precede the clinical onset of OA. Age, BMI and occupation are identified as strong predictors of the development of OA in former elite athletes.

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