Effect of Long-Term Vigorous Physical Activity on Healthy Adult Knee Cartilage

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ABSTRACT

TEICHTAHL, A. J., A. E. WLUKA, Y. WANG, A. FORBES, M. L. DAVIES-TUCK, D. R. ENGLISH, G. G. GILES, and F. M. CICUTTINI. Effect of Long-Term Vigorous Physical Activity on Healthy Adult Knee Cartilage. Med. Sci. Sports Exerc., Vol. 44, No. 6, pp. 985–992, 2012. Introduction: Whether participation in long-term vigorous physical activity affects knee cartilage is unclear and may depend on the state of knee health. We examined the association between vigorous physical activity during a decade and the subsequent changes in knee cartilage among healthy adults. We then examined whether this effect differed in those with and without bone marrow lesions (BMLs), as an indicator of preclinical joint damage. Methods: A total of 297 healthy adults age 50–79 yr were recruited. Physical activity was assessed via questionnaire at baseline (1990–1994) and at follow-up (2003–2004), and a score for persistence of vigorous physical activity score was determined. Each subject underwent knee magnetic resonance imaging in 2003–2004 and in 2006–2007. Cartilage volume, defects, and BMLs were measured using validated methods. Results: Persistent participation in vigorous physical activity was associated with worsening of medial knee cartilage defects (odds ratio (OR) = 1.5, 95% confidence interval (CI) = 1.0–2.3). In the subgroup with BMLs, but not in those without BML, persistent vigorous physical activity was associated with a significant worsening of medial knee cartilage defects (OR = 3.4, 95% CI = 1.0–16.5) and a trend toward an increased rate of loss of medial knee cartilage volume (21.6 mm$^3$ yr$^{-1}$, 95% CI = −0.4 to 43.6). Conclusions: In knees with BMLs, persistent participation in vigorous physical activity was associated with adverse cartilage changes in the medial compartment. This suggests that the long-term effects of vigorous physical activity may depend on the preexisting health of the joint. Key Words: PHYSICAL ACTIVITY, KNEE, OSTEOARTHRITIS, CARTILAGE, BONE MARROW LESIONS

Whether physical activity is beneficial or detrimental to the health of weight-bearing joints such as the knee is unclear. Although several studies have examined the relationship between physical activity and the incidence and progression of degenerative knee pathology, published data are conflicting (12,18,19,23,24,26). Whereas some studies have demonstrated the benefits of physical activity at the knee joint (12,19,23,24), others have found that physical activity may be to the detriment of the knee joint (18,20,26).

These conflicting data may have arisen for several reasons. Inherent individual factors such as age, obesity, gender, and a history of knee injury, as well as the variability in physical activity measures including the type, frequency, and duration of activity, may all have contributed to conflicting results. Although many of these variables, particularly physical activity measures, are difficult to standardize in population studies, efforts made to adjust for many of these confounders (e.g., history of knee injury) may yield more consistent results. Furthermore, perhaps the most significant contributor to inconsistent physical activity findings relates to the method for assessing knee structure. Radiography is a common method for assessing
the knee joint with measurement focusing on joint space narrowing, a surrogate measure of articular cartilage, and osteophytes. Radiological grading systems have been over-reliant on the presence of osteophytes for disease definition. This may have influenced the interpretation of previous results because studies have reported a higher prevalence of osteophytes in individuals exercising more vigorously (12,19,26), despite no changes occurring in the joint space, which is a surrogate measure of knee cartilage (19,26). Collectively, these radiographic studies may have overestimated the burden that physical activity imparts on the knee joint, based solely on one individual radiographic feature, that being the osteophyte.

Magnetic resonance imaging (MRI) provides a noninvasive and direct method for the visualization of all joint structures in vivo, including cartilage, and enables the examination of structural changes in populations before the onset of clinical osteoarthritis (OA), as defined by the American College of Rheumatology Clinical Criteria (1). MRI has therefore provided a sensitive means for examining the natural history of cartilage development across the lifespan and demonstrating how joint structures may respond to modifiable risk factors, such as physical activity. Physical activity has been shown to facilitate cartilage development in children (16), whereas forced immobility (e.g., due to spinal cord injury) results in rapid cartilage loss in adults (28,29). Cross-sectional MRI studies examining people without clinical knee disease have suggested that cartilage health may benefit from participation in a high-intensity exercise (11,22). This may be via mechanocellular transduction mechanisms on the chondrocyte. Although these findings support the importance of at least some form of physical activity for joint health, the long-term effect of physical activity on knee cartilage properties, such as cartilage volume, is unclear.

The effect of physical activity on the knee joint may also be affected by differences across individuals, such as the pre-existing health of the knee joint (27). Even before the onset of symptomatic knee OA, structural changes are present, including the presence of bone marrow lesions (BMLs) (3). BMLs are strongly linked to knee malalignment (14) and predict cartilage loss in both those with knee OA (8) and in asymptomatic cohorts (32,33). Histologically, BMLs represent areas of abnormal bone formation with excessive fibrosis and osteonecrosis (35). It has been proposed that the presence of a BML may result in a disturbance in the supply of nutrients and oxygen to the overlying cartilage plate, ultimately leading to aberrations in articular cartilage morphology (9,15,31). Because BMLs are also linked to malalignment (14), their presence may signify a joint that may adversely respond to further biomechanical stressors, such as those imparted by physical activity. Therefore, the factors contributing to the presence of BMLs, or the BML itself, may increase the susceptibility of a joint to further damage. This may be particularly evident in the context of physical activity, especially in the medial compartment where significant mechanical loads are focused during weight-bearing tasks (2).

The aims of this longitudinal MRI study were to test the hypotheses that 1) long-term participation in vigorous physical activity will benefit knee cartilage in an asymptomatic cohort of healthy adults; however, 2) there may be subgroups with asymptomatic preexisting structural knee changes (as measured by BMLs), which adversely predict a deleterious cartilage response to long-term physical activity.

**METHODS**

**Subjects.** Subjects were recruited from an existing cohort: the Melbourne Collaborative Cohort Study (MCCS), a prospective cohort study of 41,528 community-based individuals age 40–69 yr at MCCS inception (1990–1994) with the aim of examining the role of lifestyle and genetic factors in the risk of cancer and chronic diseases from middle age and beyond (10). Because our intent was to investigate subjects with no significant current or past knee disease, individuals were excluded if, at the current study’s inception at MRI baseline examination (2003–2004), they reported any previous knee injury requiring non–weight-bearing treatment for >24 h or surgery (including arthroscopy), any history of any arthritis diagnosed by a medical practitioner, or knee pain lasting >24 h in the previous 5 yr. A further exclusion criterion was a contraindication to MRI. We used quota sampling, whereby recruitment ceased when our target sample of ~300 subjects was achieved (n = 297) at baseline radiological examination in 2003–2004. The study was approved by The Cancer Council Victoria Human Research Ethics Committee and the Standing Committee on Ethics in Research Involving Human Subjects of Monash University. All participants gave written informed consent.

**Physical activity.** At MCCS recruitment between 1990 and 1994, subjects completed a questionnaire that collected demographic and physical activity information as previously described (22).

Questions relating to vigorous activity included: “On average, how many times a week do you exercise vigorously for a period of 20 min?” and “If you exercise vigorously three or more times a week, for how long have you been doing this level of activity?” “Vigorous” was defined by activity leading to sweating or shortness of breath, and examples such as swimming, tennis, netball, athletics, and running were listed. From these data, we extrapolated whether or not a subject had recently participated in vigorous physical activity for a period of at least 20 min at least once per week. At the time subjects were recruited into the present study (2003–2004) and at the time of the second MRI (2006–2007), vigorous physical activity was reassessed by asking, “Over the past 7 d, did you engage in strenuous sport and recreational activities such as jogging, swimming, cycling, singles tennis, aerobic dance, skiing, or other similar activities?”

Although the questions used were not identical, the examples given were identical or similar, thus capturing the same
forms of high intensity exercise, which, from here forward, will be classed as “vigorous.” From these data, we defined the persistence of vigorous physical activity grade, scored as follows: 0 = no participation in vigorous physical activity at both 1990–1994 and 2003–2004; 1 = participation in vigorous physical activity in either 1990–1994 or 2003–2004; or 2 = participation in vigorous physical activity in both 1990–1994 and 2003–2004. A timeline of data collection is presented in Figure 1.

**Anthropometric data.** Weight was measured, with bulky clothing removed, using electronic scales. Height was measured using a stadiometer, with shoes removed. Body mass index (BMI) was calculated as follows: weight (kg)/height (m)^2^. The 1990–1994 data were termed baseline.

**MRI and changes in knee joint structures.** Baseline and follow-up MRI scans of the dominant knee of each subject (defined as the lower limb from which the subject stepped off from when initiating gait) were obtained approximately 2 yr apart. A timeline of data collection is presented in Figure 1. Knees were imaged in the sagittal plane on a 1.5-T whole-body magnetic resonance unit (Philips) using a commercial transmit–receive extremity coil. Similar to a FLASH pulse sequence, the following parameters were used: a T1-weighted fat-suppressed three-dimensional gradient recall acquisition in the steady state, flip angle = 55°, repetition time = 58 ms, echo time = 12 ms, field of view = 16 cm, 60 partitions, matrix = 512 × 512, one acquisition time = 11 min 56 s. Sagittal images were obtained at a partition thickness of 1.5 mm and an in-plane resolution of 0.31 × 0.31 mm (512 × 512 pixels). In addition, a T2-weighted fat-saturated acquisition, repetition time = 3500–3800 ms, echo time = 50 ms, slice thickness = 3 mm, interslice gap = 1.0 mm, number of excitation = 1, field of view = 13 cm, and matrix = 256 × 192 pixels was also obtained in the coronal plane. A single trained observer (Y. W.) with more than 6 yr of experience in examining knee structural changes using MRI performed all the cartilage measures for baseline and follow-up scans, with quality checks performed by a second blinded investigator. All baseline and follow-up MRI examinations were analyzed around the same period, with the observer blinded to time and person.

Tibial cartilage volume was determined by image processing on an independent workstation using the Osiris software (University Hospital of Geneva, Geneva, Switzerland) as previously described (5,30). The volumes of the individual cartilage plates (medial and lateral tibial) were isolated from the total volume by manually drawing disarticulation contours around the cartilage boundaries on each section. The intraobserver coefficients of variation for the medial and lateral tibial cartilage volume measures were 2.1% and 2.2%, respectively (17). The change in cartilage volume was calculated by subtracting follow-up from baseline data. Annual change was then calculated by dividing this figure by the time between assessments.

Cartilage defects were graded on the MR images with a classification system that has been previously described for each compartment of the knee joint (7,14): grade 0 = normal cartilage, grade 1 = focal blistering and intracartilaginous low-signal intensity area with an intact surface and bottom, grade 2 = irregularities on the surface or bottom and loss of thickness <50%, grade 3 = deep ulceration with loss of thickness >50%, grade 4 = full-thickness cartilage wear with exposure of subchondral bone. Medial and lateral defects were graded for both the tibial and femoral compartments, and a tibiofemoral grade was calculated. Intraobserver reliability assessed in 50 MR images (expressed as intraclass correlation coefficient) was 0.90 for the medial and tibiofemoral compartment and 0.89 for the lateral tibiofemoral compartment, respectively (6). The change in defect grade was determined by subtracting the 2003–2004 defect grade from the 2006–2007 defect grade. A value ≥1 represented a worsening cartilage defect.

BMLs were defined as areas of increased signal intensity adjacent to subcortical bone present in either the medial or the lateral, distal femur or proximal tibia assessed from T2 fat-saturated images (3). Two trained observers (A. W. and D.-T. M.), who were blinded to patient characteristics, as well as sequence of images, together assessed the presence of lesions for each subject at the baseline MRI (3). The presence or absence of a BML was determined. A lesion was defined as present if it appeared on two or more adjacent slices and encompassed at least one quarter of the width of the medial and lateral compartment (3). The reproducibility for determination of the BML was assessed using 60 randomly selected knee MRIs (κ = 0.88, P < 0.001).

**Statistical analyses.** With 297 subjects, we had 80% power to show a correlation as low as 0.2 between physical activity measures and change in the various components of knee joint structure (α error = 0.05, 2-sided significance), thus explaining up to 4% of the variance of these measures. The change in tibial cartilage volume was initially assessed for normality before being regressed against the physical activity variables. Worsening tibiofemoral cartilage defects was categorized as a dichotomous outcome (yes/no); therefore, logistic regression was used. Known confounders were adjusted for in the linear and logistic regression models. These included baseline age, gender, and BMI. All analyses
grade 2 (participated in vigorous physical activity at both time points).

In subjects performing vigorous activity at one or both time points (persistence of vigorous physical activity score), cartilage defects were more likely to worsen in the medial tibiofemoral compartment (odds ratio [OR] = 1.5, 95% confidence interval [CI] = 1.0–2.3, P = 0.04) but not the lateral tibiofemoral compartment (Table 2). To more clearly examine the difference between people who had exercised persistently (i.e., exercised vigorously at both 1990–1994 and 2003–2004) and those who had not exercised vigorously at either time point, we excluded those subjects (n = 97, 36% of the cohort) who changed their participation in vigorous physical activity during the study period. We found that people who had exercised vigorously at both 1990–1994 and 2003–2004 were more likely to have an increased risk for worsening medial cartilage defects than those people who had never exercised at either time point (P = 0.04). There was no significant relationship between the persistence of vigorous physical activity and change in cartilage volume (Table 2).

The persistence of vigorous physical activity during the study period was not significantly associated with the prevalence of BMLs at the first MRI assessment in univariate analyses (OR = 1.3, 95% CI = 0.8–2.0, P = 0.32) or after adjustment for age, gender, and BMI (OR = 1.4, 95% CI = 0.8–2.2, P = 0.21). Within the whole population, 1%–2% of the variability of change in cartilage volume loss and the

were performed using the SPSS statistical package, standard version 15.0 (SPSS, Chicago, IL). \( P < 0.05 \) was considered statistically significant.

**RESULTS**

Of the original 297 subjects, 271 (91.2%) completed the longitudinal MRI component. Twenty-six subjects, 9 males and 17 females, were lost to follow-up because of death (n = 3), poor health (n = 4), withdrawal of consent (n = 10), development of a contraindication to MRI (pacemaker) (n = 4), and inability to make contact (n = 5). The subjects lost to follow-up tended to have a higher BMI (\( P = 0.06 \)) than those who completed the study but were not significantly different in other characteristics (data not shown). The subject characteristics for the longitudinal cohort, comparing those with and without knee BMLs in 2003–2004, are shown in Table 1. Besides the group with knee BMLs having a significantly greater proportion of people demonstrating lateral tibiofemoral cartilage defect worsening than the group without BMLs (\( P = 0.02 \)), no other significant differences existed between these two groups (Table 1). In terms of persistence of participation in physical activity, 128 subjects were graded 0 (never participated in vigorous physical activity), 97 graded 1 (participated in vigorous physical activity at one time point) and 46 subjects comprised

**TABLE 2. Relationship between the persistence of vigorous physical activity and change in knee cartilage morphology (n = 271).**

<table>
<thead>
<tr>
<th></th>
<th>Univariate Analyses</th>
<th>Multivariate Analyses†</th>
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<tbody>
<tr>
<td></td>
<td>95% CI</td>
<td>P</td>
</tr>
<tr>
<td>Cartilage volume§</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medial tibial</td>
<td>(-2.3 \ (-10.4 to 5.8))</td>
<td>0.57</td>
</tr>
<tr>
<td>Lateral tibial</td>
<td>(4.5 \ (-3.8 to 12.7))</td>
<td>0.29</td>
</tr>
<tr>
<td>Worsening tibiofemoral cartilage defects (yes/no)¨</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medial</td>
<td>(1.5 \ (1.0 to 2.1))</td>
<td>0.04</td>
</tr>
<tr>
<td>Lateral</td>
<td>(0.9 \ (0.7 to 1.3))</td>
<td>0.74</td>
</tr>
</tbody>
</table>

§ Annual change or risk in respective measure after adjustment for age, gender, and BMI.

¨ Results represented by regression coefficient for change in cartilage volume per increase in grade of persistent vigorous physical activity; where 0 = never exercised at a vigorous intensity, 1 = exercised at a vigorous intensity at one time point, 2 = exercised at a vigorous intensity at both time points.

" Results represented by OR for risk of worsening of cartilage defects per increase in grade of persistent vigorous physical activity; where 0 = never exercised at a vigorous intensity, 1 = exercised at a vigorous intensity at one time point, 2 = exercised at a vigorous intensity at both time points.

\( R^2 \) = coefficient of multiple determination: the proportion of variance in outcome explained by the terms in the multivariate regression model.
likelihood of defects showing progression were explained by these models (Table 2; $R^2$).

We also examined to see whether the effect of vigorous physical activity differed according to the presence of a BML at the first MRI. In subgroup analyses, among people with no BMLs in 2003–2004 ($n = 234$), the persistence in vigorous physical activity score was not significantly associated with change in knee cartilage volume or worsening knee cartilage defects (Table 3). Because the subgroups of people with baseline BMLs were small ($n = 37$), exact logistic regression was used to examine the relationship between the persistence of vigorous physical activity score and the change in cartilage morphology, with adjustment for age and BMI using categorization at their medians (57 and 25, respectively). Among those with BMLs in 2003–2004, the persistence of vigorous physical activity score was significantly associated with an increased risk for worsening medial knee cartilage defects before ($P = 0.02$) and after adjustment for potential confounders (OR = 3.4, 95% CI = 1.0–16.5, $P = 0.04$) (Table 3). This model explained 17% of the variability of change in the risk of worsening cartilage defects. After adjustment for confounders including age, gender, and BMI, the persistence of vigorous physical activity score tended to also be significantly associated with an increased rate of medial knee cartilage volume loss (21.6 mm$^3$·yr$^{-1}$, 95% CI = −0.4 to 43.6) in people with BMLs (Table 3). This model explained 14% of the variability in loss of medial cartilage volume.

**DISCUSSION**

In this longitudinal study of healthy older adults (mean age = 57.8 yr) with no history of knee injury or disease, persistent participation in vigorous physical activity was associated with an increased risk of worsening medial knee cartilage defects. In those with BMLs, but not in those without BML, there was worsening of cartilage defects and a trend toward increased loss of cartilage volume loss in the medial compartment: models relating physical activity to longitudinal change in knee structure in those with BML explained a considerably higher proportion of variation in structural change than in those without BMLs. These results suggest that in otherwise clinically healthy elderly knees, BMLs may identify those likely to have an adverse outcome from vigorous physical activity.

Previously, radiographic studies have found a history of high-intensity exercise to be associated with knee OA (12,19,26). However, examination of these studies found a common theme of a higher prevalence of osteophytes in individuals exercising more vigorously (12,19,26), despite no changes occurring in the joint space, which is a surrogate measure of knee cartilage thickness (19,26). A review of the literature confirmed this (27). These previous findings may therefore simplify bony adaptation to physical activity, rather than deleterious changes in other joint structures, such as cartilage. More recently, a delayed gadolinium-enhanced magnetic resonance imaging of cartilage (dGEMRIC) study demonstrated that adults at risk for knee OA were able to increase the glycosaminoglycan content in their knee cartilage with an exercise program that was performed three times a week for 4 months, which was deemed a beneficial response to exercise (24). In two independent cross-sectional MRI studies of subjects with no clinical knee disease, we showed that exercise at a sufficient level to cause one or a combination of tachypnea, sweating, and/or an increased pulse rate for at least 20 min was associated with increased tibial cartilage volume (11,22), suggesting that knee joint health may benefit from physical activity. This is further supported by the findings that physical activity benefits cartilage accrual in children (16), whereas forced immobilization (e.g., after spinal cord injury) in adults results in rapid cartilage loss (28,29). However, no study has

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**TABLE 3. Relationship between persistent vigorous physical activity and change in knee cartilage morphology between 2003–2004 and 2006–2007 according to subgroups based on the presence or absence of BML.**

<table>
<thead>
<tr>
<th>Cartilage volumea</th>
<th>95% CI</th>
<th>P</th>
</tr>
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<tbody>
<tr>
<td>Medial tibial</td>
<td>−4.4 (−13.3 to 4.5)</td>
<td>0.33</td>
</tr>
<tr>
<td>Lateral tibial</td>
<td>1.9 (−6.9 to 10.7)</td>
<td>0.68</td>
</tr>
<tr>
<td>Worsening cartilage defects (yes/no)b</td>
<td>1.3 (0.8 to 1.9)</td>
<td>0.25</td>
</tr>
<tr>
<td>Medial tibiofemoral</td>
<td>0.8 (0.5 to 1.2)</td>
<td>0.14</td>
</tr>
<tr>
<td>Lateral tibiofemoral</td>
<td>1.3 (0.8 to 2.0)</td>
<td>0.26</td>
</tr>
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</table>

| Prevalent baseline BMLs ($n = 37$) |
|------------------------------------|--------|---|
| Cartilage volumec | 10.4 (−9.5 to 30.4) | 0.30 |
| Medial tibial | 20.7 (−4.9 to 45.9) | 0.10 |
| Lateral tibial | 3.8 (1.1 to 16.9) | 0.02 |
| Worsening cartilage defects (yes/no)c | 1.9 (0.7 to 5.8) | 0.18 |

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* Annual change (linear regression analyses) or risk (worsening of cartilage defects) in the respective measure after adjustment for age, gender, and BMI.

* Results represented by regression coefficient for change in cartilage volume per increase in grade of persistent vigorous physical activity: where 0 = who never exercised at a vigorous intensity, 1 = exercised at a vigorous intensity at one time point, 2 = exercised at a vigorous intensity at both time points.

* Results represented by OR for risk of worsening of cartilage defects per increase in grade of persistent vigorous physical activity: where 0 = never exercised at a vigorous intensity, 1 = exercised at a vigorous intensity at one time point, 2 = exercised at a vigorous intensity at both time points. Exact logistic regression was used with adjustment for age and BMI using categorization at their medians (57 and 25, respectively).

$R^2$ = coefficient of multiple determination: the proportion of variance in outcome explained by the terms in the multivariate regression model.
examined whether long-term physical activity is beneficial or detrimental to knee cartilage. In this longitudinal study of people with no knee symptoms at baseline, we showed that persistent participation in vigorous physical activity is associated with an increased risk of worsening of medial knee cartilage defects but not change in cartilage volume. Cartilage defects are an early and sensitive measure of cartilage damage that predicts future cartilage loss and radiographic features of OA (6,20,21), and it may be that a longer length of time is required until cartilage volume loss occurs in the context of participation in long-term vigorous physical activity.

Although our results and previous radiographic studies (12,19,26) suggest that vigorous physical activity is to the detriment of the knee joint, this may be an oversimplification. Independent factors such as the preexisting state of health of the knee joint may be an important discriminator as to who responds adversely to vigorous physical activity. This concept has not been widely incorporated in previous studies. Histologically, BMLs demonstrate features consistent with ongoing bone trauma, including abnormal bone formation with excessive fibrosis, extensive bony remodeling with reversal lines and areas of osteonecrosis (35). Clinically, BMLs have been shown to be a potent risk factor for structural deterioration in knee OA (8) and are speculated to be linked to a mechanical origin (14), although this is not yet a consensus opinion. In the current study, we have used a BML subgroup to define early, preclinical knee joint disease and found that these individuals had an adverse cartilaginous response to long-term vigorous physical activity, as evidenced by progression of cartilage defects and a trend toward a loss of cartilage volume. This response was not evident in the subgroup with no BMLs. Such findings may be the consequence of loading imparted by physical activity on a joint that is mechanically abnormal, thus explaining the presence of BMLs. Therefore, the factors contributing to the presence of BMLs, or the BML itself, may increase the susceptibility of a joint to further damage. A likely biomechanical mechanism in this study is further supported by the cartilage changes in the medial but not the lateral tibiofemoral compartment. Knee joint loads are predominantly directed medially during weight-bearing tasks (2), and it may be that high-intensity exercise further increases medial compartment loads, predisposing aberrant mechanocellular transduction mechanisms to the chondrocyte. This may ultimately result in cartilage damage in the susceptible knee. Indeed, there is evidence that mechanoreceptors located in the articular cartilage may mediate aberrant chondroprotective responses in the osteoarthritic knee joint (25); in those prevalent although clinically asymptomatic BMLs, the same phenomenon may be occurring, indicating early joint damage with an impaired chondrocyte response.

An alternative explanation is that the presence of a BML is the consequence of a history of physical activity, and BMLs may therefore lie on the causal pathway between physical activity and knee cartilage changes. Subsequently, it is possible that our stratification of subgroups based on the presence of BMLs in 2003–2004 was a consequence of the preceding physical activity itself (13). However, we did not find that the persistence of vigorous physical activity score was associated with the presence of BMLs at baseline (data not shown). Therefore, it is more likely that the presence of a BML signified an abnormal knee joint with early degenerative change, whose cartilage was more susceptible to the effects of physical activity.

There are several potential limitations of this study. First, we were unable to differentiate between weight-bearing and non–weight-bearing physical activities, which may exert differential effects on knee cartilage change. This limitation reduced our ability to examine whether weight-bearing exercise in knees with BMLs are more prone to cartilage degeneration, given the likely mechanical origin of the BML (14). Nonetheless, if this were the case, inclusion of people performing vigorous non–weight-bearing activities would have led to differential misclassification, reducing our ability to demonstrate significant relationships. Furthermore, previous studies have found that questionnaire-derived information on vigorous physical activity correlates more closely with true physical activity levels and healthy outcomes (such as cardiovascular fitness) than less-vigorous activity data (4,34). Moreover, our classification of participation in vigorous physical activity was derived from data collected at two time points separated over a decade. We have assumed that those people who had participated in vigorous physical activity at baseline (1990–1994) and again at follow-up (2003–2004) had maintained similar exercise patterns in the intervening period. This is likely to be the case because we previously found an association between participation in vigorous physical activity between the two time points ($\chi^2 = 17.1, P < 0.001$) (12). Although not a focus of this study, physical activity in 2003–2004 was also associated with physical activity in 2006–2007 ($\chi^2 = 70.7, P < 0.001$). Because the aim of our study was to examine how participation of vigorous physical activity over one decade influenced subsequent knee joint changes, we did not present analyses related to the physical activity data from 2006 to 2007 because this assessment of physical activity may have been influenced by the structural changes at the knee. However, in this study, we grouped those people who had participated in vigorous physical activity at either 1990–1994 or 2003–2004 together (i.e., grade 1). This category was devised to identify people who had performed at least some physical activity, but whom were inconsistent in their exercise patterns. There were no significant differences in age, gender, BMI, or change in cartilage volume among those who either commenced or ceased participation in vigorous physical activity over the study period (data not shown). It is likely that our adopted approach of grouping these participants together may have resulted in nondifferential misclassification and thus reduced the likelihood of showing an effect. Although this is a conservative approach to the analysis, future studies will need to examine the effect of changes in exercise patterns
on articular structures. However, when we excluded those people who had changed their exercise patterns (i.e., grade 1), the magnitude, direction, and significance of the results were consistent with those reported (results not shown).

Furthermore, we have not adjusted for frontal plane knee varus–valgus alignment, which may act as a confounder to a change in cartilage properties. Moreover, it must be acknowledged that in post hoc analysis, there was only a trend for vigorous physical activity to be associated with medial knee cartilage loss in people with BMLs. Further studies are necessary to confirm these findings.

Finally, given the public health importance of physical activity, we are not proposing that individuals should reduce levels of physical activity because this may be detrimental to knee and overall health. Rather, our study suggests that further work is needed so that individuals can be stratified according to their risk for future deleterious joint changes in response to physical activity, with the potential for tailoring appropriate individualized exercise programs. For instance, it may be that obese individuals with varus-aligned knees and preexisting BMLs may benefit from non–weight-bearing exercises such as swimming, whereas those without these predisposing factors may safely exercise in a fully weight-bearing environment. Whether modification of other variables such as footwear and the surface on which exercise is performed warrants further investigation.

In this longitudinal study of community-based older adults with no history of knee injury or disease, persistent participation in vigorous physical activity was associated with deleterious medial knee cartilage changes in those with BMLs only. This suggests that the long-term effects of vigorous physical activity on cartilage may depend on the preexisting health of the knee joint. In clinically healthy older knees, BMLs identify those likely to have an adverse outcome from vigorous physical activity.

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The results of the present study do not constitute endorsement by the American College of Sports Medicine.

REFERENCES


