Background: Many trials investigated the beneficial effect of physical activity (PA) on physical function (PF) in people with osteoarthritis (OA), but factors involved in this relationship are poorly understood. Considering the link between OA and obesity and obesity-related disorders, body composition (BC) could be one of these factors.

Objectives: To examine the relationships between baseline components of PA and 5-year PF scores, considering BC variables measured at 5 years as potential mediators in theses associations (Figure).

Methods: We used data from the KHQALA cohort, a French population-based multicenter cohort of 878 patients with symptomatic knee and/or hip OA, aged between 40 and 75 years old. Baseline PA intensity (Metabolic Equivalents of Task, MET), frequency (times/week), duration (hours/week) and type (weight-bearing or not) were assessed by the Modifiable Activity Questionnaire. PF was measured with the WOMAC questionnaire at 5 years (higher scores = greater functional limitations).

Skeletal muscle mass (grams) and fat mass (grams) were measured by dual X-ray absorptiometry (DXA) in 383 patients at 5 years. Fat mass index (kg/m²), appendicular mass fat (kg), % of fat mass, lean mass index (kg/m²), appendicular muscle mass (kg), skeletal muscle mass index (kg/m² or %) were calculated based on DXA data. Sarcopenia was defined according to the FNIH Sarcopenia Project recommendations. A causal mediation analysis was used to highlight the mediating role of BC variables. Bivariate analyses (multiple linear and logistic regressions) were performed to select the variables of interest. Separate generalized linear models were used to describe the relationships between PA components, PF and selected BC variables. Unadjusted and adjusted for baseline confounders (age, gender, number of comorbidities, disease duration, mental health and vitality scores) models were ran.

Results: A 1-MET increase in baseline PA intensity was significantly associated with an improvement in PF at 5 years (-3 points). Weight-bearing PA was also significantly associated with better PF scores (-5 points).

A 1-MET increase in PA intensity at baseline was associated with a subsequent decrease at 3 years in fat mass index (-0.86 kg/m²), an increase in skeletal muscle mass index (≥5%), and a decrease in % of fat mass (-2%). Non-weight-bearing PA was significantly associated with a decrease in fat mass index (-2.5 kg/m²).

A 1-point increase in PF score was associated with a reduction in skeletal muscle mass index (calculated from body mass index, -0.3%) and an increase in skeletal muscle mass index (calculated from height, +3.6 kg/m²). The presence of sarcopenia was significantly associated with a degradation of PF (+7 points).

Crude analyses indicated that 20.4% of the effect of baseline PA intensity on PF scores at 5 years was mediated by skeletal muscle mass index (calculated from height), 23.2% by fat mass index and 26.6% % of fat mass. Similarly, 19.3% of the effect of baseline PA type on PF scores at 5 years was mediated by fat mass index and 15.1% % of fat mass. After adjustment, we found no longer evidence of a mediating role of BC variables in these associations.

Conclusion: We found significant associations between a 1-MET increase in PA intensity, weight-bearing PA at baseline and improvement in PF at 5 years, without any mediating role of BC variables. Further studies are needed to better understand the factors involved in these associations, especially psychosocial variables.

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Background: Evidence suggests that periarticular muscles have a role in the pathogenesis of pain, but results have not been consistent. We recently reported that pain population is heterogeneous and consists of different subgroups of which the causes and mechanisms differ.

Objectives: To examine the association of muscle mass, leg strength, knee extensor strength, low-limb muscle quality with knee pain trajectories.

Methods: Data on 975 participants from a population-based older adult cohort study were utilised. Dual-energy X-ray absorptiometry was used to assess muscle/fat mass. Leg strength in both legs and dominant knee extensor strength were measured. Low-limb muscle quality was calculated (ie. leg strength divided by lower-limb muscle mass). The Western Ontario and McMaster Universities Osteoarthritis Index pain questionnaire was used to measure knee pain at each time-point. Radiographic knee osteoarthritis (ROA) was assessed by X-ray. Group-based trajectory modelling was applied to identify pain trajectories. Multinomial logistic regression was used for the analyses.

Results: A total of 975 participants [Mean±SD: age 62.2±7.4 years, body mass index (BMI) 27.8±4.6kg/m² and 51% of females] were included in the analysis. Three distinct pain trajectories were identified: ‘Minimal pain’ (53%), ‘Mild pain’ (34%) and ‘Moderate pain’ (13%). In multivariable analysis, both greater total and low-limb muscle mass were associated with an increased risk of ‘Mild pain’ [total muscle mass: relative risk (RR): 1.51 per SD increase, 95%CI: 1.14–1.98; low-limb muscle mass RR: 1.33 per SD increase, 95%CI: 1.07–1.66] and ‘Moderate pain’ [total muscle mass: RR: 2.57 per SD increase, 95%CI: 1.70–3.89; low-limb muscle mass RR: 2.03 per SD increase, 95%CI: 1.47–3.80] compared to the ‘Minimal pain’ trajectory group. After further adjustment for fat mass, these associations disappeared. Total muscle mass percentage was associated with a reduced risk of being worse pain trajectories. In relation to the ‘Minimal pain’ trajectory group, leg strength, knee extensor strength and quality were associated with a reduced risk of being in more severe pain trajectories after adjustment for covariates (RR=0.56 to 0.71 per SD increase, all P<0.05). Similar results were observed in those with ROA.

Conclusion: Muscle percentage, strength and quality, but not muscle mass itself are associated with a reduced risk of being more severe pain trajectories, suggesting that improving muscle composition, muscle function and power are of more clinically relevance to preventing the development and maintenance of worse pain trajectories.

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Besides, we calculated mean points in each parameter in the scale and compared it with the maximum score in this parameter (in percent). Table 2 shows the results. The most severe change was bone attrition.

### Table 2. Mean points of different WORMS parameters in examined knee joints.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>MFTJ (percent, total)</th>
<th>LFTJ (percent, total)</th>
<th>PFI (percent, total)</th>
<th>S-region (percent, total)</th>
<th>Total (percent, total)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cartilage</td>
<td>13,0% (n=24)</td>
<td>1,02% (n=1)</td>
<td>7,64%</td>
<td></td>
<td>2,91%</td>
</tr>
<tr>
<td>Marrow</td>
<td>0,00%</td>
<td>0,37%</td>
<td>0,00%</td>
<td></td>
<td>0,00%</td>
</tr>
<tr>
<td>Bone cysts</td>
<td>0,00%</td>
<td>0,00%</td>
<td>0,00%</td>
<td></td>
<td>0,00%</td>
</tr>
<tr>
<td>Bone atrophy</td>
<td>15,37% (n=24)</td>
<td>0,00%</td>
<td>-</td>
<td></td>
<td>1,75%</td>
</tr>
<tr>
<td>Osteophytes</td>
<td>6,59%</td>
<td>5,48%</td>
<td>8,33%</td>
<td></td>
<td>10,00%</td>
</tr>
<tr>
<td>Menisci</td>
<td>0,00%</td>
<td>0,93%</td>
<td>-</td>
<td></td>
<td>0,00%</td>
</tr>
<tr>
<td>Ligaments</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>0,00%</td>
</tr>
<tr>
<td>Synovitis</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>0,00%</td>
</tr>
<tr>
<td>Total</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>0,00%</td>
</tr>
</tbody>
</table>


We didn’t observe asymptomatic lesions in the medial meniscus, narrow abnormality in MFTJ and LFTJ, subchondral cysts in any location, ligament lesions. Despite minimal osteophytes almost in all individuals, they didn’t have any clinical features of knee OA.

**Conclusion:** MRI of the knee joints in the cohort of young relatively healthy individuals without clinical features of OA revealed irreversible structural changes characteristic of symptomatic OA. There is no association between symptoms and structural damage. Based on these, we can make an assumption about asymptomatic stage of OA. In order to distinguish between definitions of early asymptomatic OA as a disease onset and asymptomatic structural changes as reflection of metabolic disorders it is necessary to follow up and to perform an in-depth examination of these individuals.

**References:**


**Disclosure of Interests:** Natalia Martusevich Shareholder of: k, Svetlana Duben: None declared, Tatsiana Bondar: None declared, Katsiarina Gudkevich: None declared, Natalia Martusevich Shareholder of: k, Svetlana Duben: None declared, Tatsiana Bondar: None declared, Katsiarina Gudkevich: None declared.

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### A PROGNOSTIC MODEL OF PRE-RADIOGRAPHIC KNEE OSTEOARTHRITIS: DATA FROM THE OSTEOARTHRITIS INITIATIVE


**Background:** The improvement of the existing diagnostic methods to detect pre-radiographic knee OA (KOA) may facilitate the development of preventive strategies. It has been postulated that combining biochemical with clinical markers, may increase the prognostic power to detect who is at high risk for developing KOA.

**Objectives:** To validate and quality the ability of 6 proteins with biomarker potential to generate a prognostic model of knee OA prediction through the combination of validated OA biomarkers and clinical markers.

**Methods:** In the validation phase (Figure 1), 749 sera at the baseline visit belonging to participants from the Osteoarthritis Initiative (OAI) Cohort were randomly selected to blindly quantify 6 biomarkers using in-house custom sandwich microarrays built using the xMAP technology. Among these, only 540 participants have a Kellgren and Lawrence (KL) grade = 0-1 at the beginning of the OAI study in at least one knee. After a follow-up period of 98 months, 209 participants developed KOA in at least one knee (KL ≥ 2) and were classified as incident group, whereas 331 did not develop the disease (KL = 0-1) and were classified as not-incident group. Statistical differences between the outcome groups were assessed by non-parametric Mann-Whitney U tests. In the qualification phase (n=540), univariate regression analyses were carried out to investigate whether the individual biomarkers were associated with the risk of KOA development. A clinical prognostic model was defined by stepwise regression analysis using clinical non-radiographic variables significantly associated with the OA incidence. The utility of the potential biomarkers, alone or in combination, was evaluated by comparing the Area Under the Curve (AUC) of the clinical prognostic model with the biomarkers plus clinical prognostic models. In addition, sen

**Results:** The incident group showed significant higher serum concentrations at the baseline visit (p < 0.05) for all the potential biomarkers analyzed in this study. Moreover, 5 of them were also significantly associated with the future appearance of radiographic KOA, yielding Odds Ratios (OR) ≥ 10 per 10 µg/ml increase. Among all the possible combinations, the inclusion of 2 biomarkers to the clinical prognostic model showed a significant improvement of the predictive capacity (AUCs = 0.78 vs 0.82, p = 0.044) with 65% (95% Confidence Interval (95%CI): 60-70%) specificity and 88% (95%CI: 81-91%) sensitivity. Variables included in the regression model and all metrics comparing the biomarkers plus clinical prognostic model with the clinical prognostic model are shown in Figure 2A. The ROC curves of the biomarkers-only model, clinical prognostic model and biomarkers plus clinical prognostic model are represented in Figure 2B.

**Conclusion:** We have generated a prognostic model for the prediction of KOA by combining biomarkers and clinical variables, which showed a putative utility in the clinical setting by improving the predictive capacity of a clinical prognostic model to identify patients at a higher risk to develop radiographic KOA.

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**ARE WE OVERLOOKING OSTEOARTHRITIS? – A COMPARATIVE STUDY OF PAIN, FUNCTION AND QUALIFY OF LIFE IN PATIENTS WITH HAND OSTEOARTHRITIS AND RHEUMATOID ARTHRITIS**