Knee Osteoarthritis
By Flavia Yazigi, PhD

Although rheumatic diseases (RD) have low death rates, they are one of the primary causes of compromised quality of life and absenteeism from work, with attendant economic and social consequences\[^1, 2\]. In Portugal, RD are responsible for 40 to 60% of cases of prolonged physical incapacity in daily activities, 43% of cases of absenteeism from work, and 35 to 41% of early retirements due to illness\[^3\].

Osteoarthritis (OA) is the most prevalent rheumatic disease and represents a great risk to the quality of life of the individual due to its effect on lower extremity based activities (such as walking up and down stairs, climbing and squatting)\[^1, 4, 5\] and the consequent loss of autonomy. OA is considered a disease of the whole joint that may result from multiple pathophysiological mechanisms\[^6\] and involves joint degeneration, including degradation of articular cartilage and subchondral bone\[^2, 7, 8\], which can lead to bone sclerosis and formation of bone cysts and marginal osteophytes.

The causes of OA are not completely understood but it is thought to be a complex, adaptive response of the joints to biomechanical, genetic and environmental events \[^9\]. In OA, imbalances in subchondral bone turnover can lead to thickened, low-quality bone, which contributes to cartilage loss, joint space narrowing and thinning of the trabecular bone\[^10\].

OA signs include molecular, morphological, biochemical and biomechanical changes in both cells and the extracellular matrix, which lead to tissue softening, fibrillation, ulceration, loss of articular cartilage, sclerosis, and eburnation of subchondral bone, osteophytes and subchondral cysts. Recent studies have demonstrated that low-grade inflammation plays a pathophysiological role in OA. The severity and symptoms of OA, as well as its progression and the consequent impairment in physical fitness, may be mediated, in part, by the extent of chronic inflammation\[^11, 12\].

In general OA affects knees, hips, hands and spine joints; however, the knees are the most commonly affected weight bearing joint\[^13\]. Knee osteoarthritis (KOA) can compromise an individual's quality of life and exhaust considerable healthcare resources, making this rheumatic disease a stand out among the public health problems in many countries\[^14-16\]. In Portugal, the National Observatory for the Rheumatic Disease (ONDOR) reported a prevalence of KOA in the Portuguese population of 11.1% (IC95%: 9.4-13.1)\[^17\], and this value is expected to increase due to increases in obesity\[^18\] and an aging population\[^19\].

KOA can occur in both joints of the knee, patellofemoral and tibiofemoral joints, but it is more common in the medial tibiofemoral compartment, most likely because the medial compartment bears 60–80% of the compressive loads in the neutrally aligned knee during normal walking\[^20\] and because of the higher frequency of varus malalignment\[^21\].

Age, obesity, occupation, lower limb muscle weakness, previous knee injury and misalignment are the main KOA risk factors reported in the literature, however, only obesity, occupation and strength are considered modifiable factors\[^22-25\]. In the predictive model presented by Zhang and coworkers (2011)\[^26\], six risk factors were related to KOA incidence (age, sex, BMI, occupation, family history and knee injury) and four risk factors were related to KOA progression (age, sex, knee injury and sports). Inflammation and biomechanical factors, such as joint loading, are also recognized risk factors that can exacerbate the progression of KOA \[^27-29\].
Symptoms and Quality of Life

When clinically evident, KOA causes joint pain, tenderness, functional impairment, morning joint stiffness, joint position stiffness, crepitus, occasional effusion and variable degrees of inflammation without systemic effects\[^{30}\]. In general, these symptoms compromise both joint stability and the capacity to perform movements during daily activities.

Pain is the main symptom of KOA and can result from the wear of the subchondral bone, which is widely innervated\[^{31}\], or from local factors, such as synovial inflammation caused by by-products of cartilage degradation. The manifestation of this pain can have a mechanical or an inflammatory pattern\[^{32}\]. Mechanical pain is related to knee joint movements and weight-bearing activities, intensifying with increased knee joint strain and disappearing after short rest. Additionally, it can increase with prolonged periods of inactivity, and can disappear after some gentle movement of the joint.

The occurrence of inflammatory pain is less predictable, is often described as burning, may be accompanied by swelling and a sensation of warmth, and can be increased by different factors\[^{32}\]. Synovitis may activate sensory nerves, leading to pain symptoms and neurogenic inflammation\[^{6}\]. Pain can inhibit muscle activity and, therefore, contribute to the loss of strength, alterations in loading, and changes in gait velocity\[^{33, 34}\]. Although arthrogenic muscle inhibition (AMI) has been well documented after knee joint injury, pain and excessive intra-articular fluid, which is associated with the inhibition of alpha motor neurons due to abnormal afferent information from sensitized articular receptors, also compromise the ability to fully activate the quadriceps and hamstring muscles in KOA\[^{35, 36}\].

The World Health Organization's definition of Quality of Life (QOL) is a broad ranging concept that is affected in a complex way by the person's physical health, psychological state, level of independence, social relationships, personal beliefs and their relationship to salient features of their environment\[^{37}\].

The symptoms of KOA can deeply affect a patient's QOL; therefore, the implementation of interventions to control these symptoms and improve overall quality of life should be considered. In this dissertation, the dimensions of the patient's life that were considered with respect to their level of health and QOL included physical well-being, functional ability, emotional well-being, and social well-being\[^{38}\].

Screening/Diagnosis

The resources available for KOA diagnosis include clinical evaluations (specialist examination and questionnaires) and biochemical or imaging methods\[^{39-41}\]. The American College of Rheumatology (ACR) established three levels of diagnostic criteria for KOA: clinical only (92% sensitivity; 75% specificity), clinical and radiological (95% sensitivity; 69% specificity) or clinical and laboratorial (91% sensitivity; 86% specificity)\[^{39}\].

Considering financial constraints, the clinical diagnostic criteria are the most viable option for primary care. According to the clinical criteria, KOA diagnosis should be based on the presence of knee pain in combination with at least three of the following variables: age>50, short-lived morning stiffness (<30 min), crepitus, tenderness, bony enlargement and no palpable warmth\[^{39}\]. More recent recommendations from the European League Against Rheumatism (EULAR) for clinical KOA diagnosis are based on three symptoms (persistent knee pain, morning stiffness and functional impairment) and three clinical signs (crepitus, restricted movement and bony enlargement)\[^{42}\]. A recent study concluded that crepitus could be an important symptom for detection of KOA progression in the patellofemoral joint\[^{43}\].
Laboratory and imaging methods are costly and the connection between the results and KOA symptoms are not clear, particularly in the initial stages (pre-radiographic KOA, Kellgren-Lawrence radiographic grade 1)\(^44\). Although x-rays alone can provide a bone overview, the Kellgren-Lawrence (K-L) severity index is considered a useful method for KOA detection in epidemiological studies\(^45\). Magnetic resonance imaging (MRI), despite being a very expensive technique, is a sensitive tool that can identify joint components and some cartilage degeneration in the early stages\(^41, 46, 47\). According to Schipohof and coworkers\(^48\), the MRI definition for tibiofemoral osteoarthritis (definite osteophyte and full-thickness cartilage loss or a combination of these factors with other MRI OA features) is a more sensitive method for detecting structural KOA than the Kellgren-Lawrence method\(^48\). However, more studies are necessary to verify which MRI findings in early OA are clinically important\(^43, 49\).

For public health purposes, self-report questionnaires are still considered a valid and accessible method for KOA screening, but it is necessary to improve diagnostic instruments to ensure that interventions occur in the early stages.

The available KOA-related questionnaires can be organized into two groups: patient outcomes and screening instruments. The first group includes questionnaires related to patient outcomes (functionality, signs, symptoms and quality of life)\(^50-55\), while the Western Ontario and McMaster Universities Arthritis Index (WOMAC)\(^50\) and the Knee Injury and Osteoarthritis Outcome Score (KOOS)\(^52, 56\) are widely used screening instruments.

The WOMAC has been validated with three types of scales: visual analog\(^50\), Likert\(^50\) and a numerical rating scale (NRS)\(^57\). The NRS allows an immediate evaluation and can be used on the phone or with a computerized touch screen (pain: Intraclass Correlation Coefficient (ICC)=0.915, rho=0.88; stiffness: ICC=0.745, rho=0.77, function: ICC=0.940, rho= 0.87).

The KOOS is considered an extension of the WOMAC and is a specific instrument developed to assess patients' perceptions about their knees, functional status and knee-related quality of life. The KOOS was validated with a sample of 21 participants with Anterior Cruciate Ligament (ACL) injuries. Its test-retest reliability after a 9-day interval showed an ICC of 0.75 for the Daily Living subscale (ADL), 0.81 for the Sport and Recreation subscale (Sport/Rec), 0.86 for the knee-related Quality of Life subscale (QOL), 0.85 for the Pain subscale and 0.93 for the Other Symptoms subscale\(^52\) (O.Symptoms). The KOOS’s responsiveness over 6 months was verified by assessing the effect size (QOL=1.65; Pain=0.84; ADL=0.94; O.Symptoms=0.87 and Sport/Rec=1.16)\(^52\). The construct validity of the KOOS was assessed in comparison to the SF-36 questionnaire\(^56\).

**Obesity and Knee Osteoarthritis**

Knee Osteoarthritis (KOA) is highly prevalent in obese individuals\(^22\). The analyses published in 2010 by International Obesity Task Force (IOTF)\(^58\), a part of the International Association for the Study of Obesity (IASO), estimated that, worldwide, approximately 1.0 billion adults are currently overweight and a further 475 million are obese.

In Portugal, more than 50% of the population is overweight (25<BMI≤29.5 kg/m\(^2\)) or obese (BMI≥30 kg/m\(^2\)), with a prevalence in adults (18-64yrs) of 66.6% (males) and 57.9% (females) and a prevalence in older adults (≥65yrs) of 70.4% (males) and 74.7% (females)\(^59\). With increasing rates of obesity\(^18\) and the aging of the Portuguese population\(^19\), the expectations are that the prevalence of KOA will increase.
There is a bidirectional interaction between KOA and obesity (Fig. 1) where weight load exacerbates mechanical pain, a symptom that markedly affects the individual’s quality of life. Mechanical pain can cause irritability, sleeplessness, depression\(^\text{[60]}\), and physical and psychological changes that may aggravate the disease, providing a general loss of functionality and, thereafter, inactivity. The majority of patients with KOA do not achieve the recommended levels of physical activity\(^\text{[61, 62]}\), which can lead to weight gain and obesity\(^\text{[63]}\). The combination of obesity and KOA creates a vicious cycle of pain, physical activity reduction, loss of functionality, and disease progression leading to more physical activity avoidance\(^\text{[64]}\), weight gain and increased pain. These cycles can be worsened with depressive symptoms, which are associated with obesity and KOA symptoms\(^\text{[60, 65, 66]}\), where each can trigger and influence the other, further compromising the quality of life.

In addition to local effects due to increased joint loading, obesity has systemic metabolic effects in KOA\(^\text{[67]}\) caused by the higher concentrations of inflammatory markers (such as TNF-\(\alpha\) and leptin) that are predominantly secreted by adipose tissue in obese individuals. These, in turn, can induce the production of IL-6 and C-reactive protein (CRP)\(^\text{[68]}\). The pathogenesis of obesity is characterized by hypothalamic inflammation and a subsequent, central resistance to leptin, which compromises the normal role of high leptin concentration to reduce food intake and increase energy expenditure. In addition, leptin increases the synthesis of TNF-\(\beta\), a stimulator of osteophyte formation\(^\text{[69]}\). The resulting low-grade inflammation plays a pathophysiological role in OA because it can affect muscle function, lower the individual’s pain threshold, and affect chondrocyte homeostasis, leading to degenerative changes in cartilage\(^\text{[11, 28, 70]}\).
Weight loss in the prevention and treatment of KOA is gaining increasing importance because it provides a reduction in the load exerted on the knee during daily activities and can decrease the pro-inflammatory action of cytokines and adipokines, which are strongly activated by obesity. Furthermore, in addition to reductions in pain manifestation, obesity is a controllable risk factor and decreasing its occurrence should contribute to a reduction in KOA progression.

Walking is one of the most important functionalities of the human being and is essential for autonomy and an independent life. The aerobic capacity and walking ability in different populations have been evaluated by the Six Minutes Walking Test (6MWT), and reference values related to obesity and KOA have been published.

Walking, as well other weight bearing movements, is the most common exercise pattern recommended for obese individuals when initiating their weight loss exercise program, however, the existence of knee pain and other KOA symptoms could be a constraint for exercise motivation and adherence, especially if symptoms of depression are also present.

**Exercise on Knee Osteoarthritis**

The recommendations for KOA management include pharmacologic and non-pharmacologic approaches. In general, and according to ACR, pharmacologic modalities can include acetaminophen, oral and topical NSAIDs, tramadol, and intra-articular corticosteroid injections.

Exercise is an effective non-pharmacological treatment for the management of KOA and is recommended by the Osteoarthritis Research Society International (OARSI), by the American College of Rheumatology (ACR) and by the European League Against Rheumatism (EULAR). Other non-pharmacologic modalities include weight loss in overweight patients, medial wedge insoles for valgus KOA, subtalar strapped lateral insoles for varus KOA, medially directed patellar taping, manual therapy, walking aids, thermal agents, tai chi, self-management programs, and psychosocial interventions.

Aerobic, aquatic and resistance exercise have been shown to help interrupt the cycle of pain-physical activity reduction, control KOA symptoms, improve posture and physical fitness, and act to modify risk factors such as body mass index (BMI) and muscle weakness. However, the benefits of an exercise program depend on participant adherence, which, according Bennel (2011) is very difficult to achieve in individuals with KOA symptoms.

Several reports indicate that an exercise program for KOA should be a broad intervention that includes cardiorespiratory training, lower limb strengthening, flexibility, gait training, and balance and posture training. In addition, weight reduction (in cases of obesity), patient education and a psychological approach should be considered.

- **Cardiorespiratory training**: Aerobic exercise is one of the main physical fitness components and is correlated with an improvement in cardiovascular and respiratory function, a reduction in cardio-metabolic disease risk factors and a reduction in morbidity and mortality. In addition to providing individuals with KOA the ability to perform daily living activities that require sustained aerobic metabolism, cardiorespiratory training has been shown to effect pain control and reduce disability. The Six Minutes Walking test has been widely used for aerobic fitness assessment and reference values for obese adults, obese adults with KOA and healthy individuals are shown in table 1.
### Table 1: Reference values of 6MWT for KOA, obese and health subjects.

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Age (years)</th>
<th>Sample Characteristics</th>
<th>Mean (m)</th>
<th>SD (±)</th>
<th>Speed (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hulens et al. (2003)</td>
<td>82</td>
<td>18-65</td>
<td>BMI≤26 kg/ m²</td>
<td>722</td>
<td>64</td>
<td>2.0</td>
</tr>
<tr>
<td></td>
<td>85</td>
<td>18-65</td>
<td>BMI≥27 kg/ m²</td>
<td>591</td>
<td>54</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>133</td>
<td>18-65</td>
<td>BMI≥35 kg/ m²</td>
<td>539</td>
<td>68</td>
<td>1.5</td>
</tr>
<tr>
<td>Beriault et al. (2009)</td>
<td>21</td>
<td>40-65</td>
<td>BMI=37±10 kg/ m²</td>
<td>452</td>
<td>9</td>
<td>1.3</td>
</tr>
<tr>
<td>Capodaglio et al (2013)</td>
<td>227</td>
<td>20-60</td>
<td>BMI=43±5 kg/ m²</td>
<td>564</td>
<td>62</td>
<td>1.6</td>
</tr>
<tr>
<td>Miller et al (2006)</td>
<td>87</td>
<td>≥60</td>
<td>KOA BMI≥30 kg/ m²</td>
<td>442</td>
<td>8</td>
<td>1.2</td>
</tr>
<tr>
<td>Messier et al (2013)</td>
<td>150</td>
<td>≥55</td>
<td>KOA BMI=33±5kg/ m²</td>
<td>480</td>
<td>15</td>
<td>1.3</td>
</tr>
<tr>
<td>Wang et al (2011)</td>
<td>84</td>
<td>≥55</td>
<td>KOA</td>
<td>331</td>
<td>9</td>
<td>0.9</td>
</tr>
<tr>
<td>Kennedy et al. (2005)</td>
<td>150</td>
<td>50-73</td>
<td>KOA</td>
<td>412</td>
<td>123</td>
<td>1.1</td>
</tr>
<tr>
<td>Kervio et al. (2003)</td>
<td>12</td>
<td>60-70</td>
<td>Apparently healthy</td>
<td>545</td>
<td>21</td>
<td>1.5</td>
</tr>
<tr>
<td>Jones et al. (1998)</td>
<td>861</td>
<td>60-64</td>
<td>Apparently healthy</td>
<td>639</td>
<td>88</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>1566</td>
<td>65-69</td>
<td>Apparently healthy</td>
<td>600</td>
<td>102</td>
<td>1.7</td>
</tr>
<tr>
<td></td>
<td>1813</td>
<td>70-74</td>
<td>Apparently healthy</td>
<td>580</td>
<td>100</td>
<td>1.6</td>
</tr>
<tr>
<td>Troosters et al. (1999)</td>
<td>51</td>
<td>50-85</td>
<td>Apparently healthy</td>
<td>631</td>
<td>93</td>
<td>1.8</td>
</tr>
<tr>
<td>Camarri et al. (2005)</td>
<td>33</td>
<td>55-75</td>
<td>Apparently healthy</td>
<td>659</td>
<td>62</td>
<td>1.8</td>
</tr>
<tr>
<td>Carvalho et al. (2008)</td>
<td>32</td>
<td>65-71</td>
<td>Apparently healthy</td>
<td>515</td>
<td>28</td>
<td>1.4</td>
</tr>
</tbody>
</table>

- Lower limb strength training: Muscles constantly interact with the synovial joints, and this can influence the osteoarthritis process. The main goal of recommended strength training is to maintain basic muscle functions (movement, joint stability, the absorption of mechanical shock and proprioception, avoiding muscle weakness, improving body stability, and posture and body weight support). In addition to being considered essential for the performance of daily activities, strength training has a positive effect on the prevention of lower limb injury, one of the KOA risk factors. Weakness of the quadriceps is a frequent occurrence in KOA and previous clinical trials have shown that quadriceps strength training is effective for the improvement of pain and physical fitness.

Obesity combined with sarcopenia, termed sarcopenic obesity, is also closely associated with the prevalence of KOA. Intensive strength training can change thigh composition and has shown promise in treating the underlying biomechanical (knee-joint loading)
and inflammatory disease pathways\textsuperscript{[108]}. However, in cases of varus malalignment, its effects on pain and on the external Knee Adduction Moment (KAM) have been questionable\textsuperscript{[92]}. In response to this and based on biomechanical analyses, recent approaches suggest that the strengthening of hip abductor muscles can reduce medial knee loading\textsuperscript{[93]}.

Reference values for knee strength in adults with KOA are cited in table 2.

<table>
<thead>
<tr>
<th>Article Authors</th>
<th>Year</th>
<th>Strength Protocol</th>
<th>N Age (years±SD)</th>
<th>BMI±SD (Kg/m²)</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sanchez-Ramirez et al.\textsuperscript{[109]}</td>
<td>2013</td>
<td>Isokinetic extension (60º/s)</td>
<td>N=284 (62±8)</td>
<td>29±5</td>
<td>Ext: 0.8±0.4 Nm/kg</td>
</tr>
<tr>
<td>Bennell et al.\textsuperscript{[110]}</td>
<td>2013</td>
<td>Isometric extension</td>
<td>N=82 (62±7)</td>
<td>30±4</td>
<td>Ext: 1.44 ±0.4Nm/kg</td>
</tr>
<tr>
<td>Diracoglu et al.\textsuperscript{[111]}</td>
<td>2009</td>
<td>Isokinetic extension (60º/s)</td>
<td>N=51 (56±10)</td>
<td>24±5</td>
<td>Ext: 114±40 Nm</td>
</tr>
<tr>
<td>Malas et al.\textsuperscript{[112]}</td>
<td>2013</td>
<td>Isokinetic extension (60º/s)</td>
<td>N=61 (59±7)</td>
<td>-</td>
<td>Ext: 80±33 Nm</td>
</tr>
<tr>
<td>Glass \textsuperscript{[113]}</td>
<td>2013</td>
<td>Isokinetic extension (60º/s)</td>
<td>N=2404 F:(63±8) M:(62±8)</td>
<td>31±6</td>
<td>F: 67±25 Nm M: 122±52 Nm</td>
</tr>
<tr>
<td>Kean et al.\textsuperscript{[114]}</td>
<td>2010</td>
<td>Isokinetic extension (60º/s)</td>
<td>N=20 (54±9)</td>
<td>30±4</td>
<td>1.43±0.5Nm/Kg</td>
</tr>
<tr>
<td>Lim et al.\textsuperscript{[115]}</td>
<td>2009</td>
<td>Isometric extension</td>
<td>N=184 (65±8)</td>
<td>29±5</td>
<td>1.33±0.5 Nm/kg</td>
</tr>
<tr>
<td>White et al.\textsuperscript{[116]}</td>
<td>2012</td>
<td>Isokinetic extension (60º/s)</td>
<td>N=1788 (67±8)</td>
<td>&lt;25 25-&lt;30 30-&lt;35 ≥35</td>
<td>1.17±0.4 Nm/kg 1.1±0.4 Nm/kg 1.0±0.4 Nm/kg 0.82±0.3 Nm/kg</td>
</tr>
<tr>
<td>Lund et al.\textsuperscript{[117]}</td>
<td>2008</td>
<td>Isokinetic extension (60º/s)</td>
<td>N=79 (≥50)</td>
<td>Normal</td>
<td>Ext:71±26 Nm</td>
</tr>
<tr>
<td>Trans et al.\textsuperscript{[118]}</td>
<td>2009</td>
<td>Isokinetic extension (60º/s)</td>
<td>N=52 (60±10)</td>
<td>29±6</td>
<td>Ext: 58.5±21 Nm</td>
</tr>
</tbody>
</table>

Table 2: Reference of main published articles of knee strength and KOA.
Flexibility: Patients with symptomatic KOA have poorer flexibility in both the affected and unaffected leg\textsuperscript{76}, mainly in the hamstrings muscles\textsuperscript{119}. Inclusion of stretching exercises is recommended in every exercise training program for adults\textsuperscript{96} and can improve the range of motion and physical function in individuals with KOA\textsuperscript{86}.

Gait training: In addition to being considered an aerobic exercise, walking is one of the most important functionalities of the human being and is essential for autonomy and an independent life\textsuperscript{76}. Functional limitations imposed by KOA symptoms and by obesity cause obese individuals with KOA to employ different gait patterns to find better balance and avoid pain. Hulens and his team (2003)\textsuperscript{78} suggest that knee pain is an important predictor of the 6MWT in obese individuals, based on their observation that the distance walked by this group in the study was significantly inferior to lean individuals (table 1). A correct walking pattern could generate some difficulty and joint discomfort during practice; therefore, a multicomponent training has increasingly been suggested. In addition to strength, flexibility and aerobic components of training, gait training should be considered\textsuperscript{94}.

Stability and posture training: KOA includes symptoms of instability\textsuperscript{95}. Recent evidence has suggested that changes in lower limb biomechanical factors during weight-bearing activities may have substantial impact on the ability to maintain a neutral spine posture while moving the extremities in a manner that is independent of the trunk. Stability and posture training is therefore imperative for proper movement and function in all daily activities.

Weight reduction (in cases of obesity): The OARSI recommendations\textsuperscript{71} for weight loss in the treatment of KOA are gaining increasing importance\textsuperscript{72-75}. For a general weight loss program, the ACSM guidelines\textsuperscript{96} recommend a reduction of 5-10% of initial weight over 3-6 months by an intervention of moderate to intense aerobic exercise, resistance-training and behavior intervention. In cases of KOA, Messier and coworkers (2005)\textsuperscript{73} reported that a weight reduction of 1 kg was associated with a knee load reduction of 4 units per step; a clinically meaningful reduction when considered over the many steps performed each day.

Education: This intervention should include general information for a healthy lifestyle and specific information about KOA, its implications and alternatives for managing and living with it. Likewise, posture strategies and gentle movements can help in pain control. The routine of pain self-assessment is essential for understanding its response to exercise and for management of exercise intensity. The essence of an educational program should be to help patients learn to live with the disease while improving their quality of life and reaching a feeling of wellness\textsuperscript{83, 84, 120}.

Psychological approach: Factors such as fear, anxiety, and depression have adverse effects on the disability in people with KOA\textsuperscript{60, 65, 88} and like pain, depression is considered a major obstacle for KOA management\textsuperscript{60, 82}. An individual’s perception of pain severity can be influenced by centrally mediated factors and behavioral components\textsuperscript{121}. A structured exercise group class can be an effective way to increase motivation by providing a social support for exercise adherence and, thereby, promoting lifestyle changes\textsuperscript{96}. In addition, exercise and physical activity improve factors related to psychological distress and these changes could lead to improved pain and function in people with KOA.
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