Tension Release Therapy: The Development of a New Integrated Behavioural Intervention for Knee Osteoarthritis

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Abstract

Background: Exercise-based approaches have been a cornerstone of physiotherapy management of knee osteoarthritis for many years. However, the magnitude of clinical effect is considered small to modest and the need for continued adherence identified as a barrier to clinical efficacy. While many exercise-based approaches incorporate an educational/coping component alongside muscle strengthening, there has been no previous attempt to integrate psychological techniques with muscle retraining. Building on these ideas, the aim of this study was to create a new integrated behavioural intervention for knee osteoarthritis, suitable for delivery by a physiotherapist.

Methods: Through literature review, we created a framework linking theory from pain science with emerging biomechanical concepts related to knee muscle co-contraction. Using recognised behaviour change theory, we then mapped a set of intervention components which were iteratively developed through ongoing testing and consultation with users.

Results: The underlying framework incorporated ideas related to central sensitisation to pain, maladaptive motor responses to pain and also focused on the idea that increased knee muscle co-contraction could result from changes in postural tone. Building on these ideas, we created an intervention with five components: making sense of pain, general relaxation, postural deconstruction, responding differently to pain and functional muscle retraining. The intervention incorporated a range of animated instructional videos to communicate concepts related to pain and biomechanical theory and also used EMG biofeedback to enable patients to visualise muscle patterns. User feedback was positive with patients describing the intervention as enabling them to “create a new normal” and to be “in control of their own treatment.” Furthermore, large reductions in pain were observed from 11 patients who received a full prototype version of the intervention.

Conclusion: We have created a new intervention for knee osteoarthritis, designed to empower individuals with capability and motivation to change muscle patterns and beliefs associated with pain. Preliminary feedback and clinical indications are positive, and this motivates future large-scale trials to understand potential efficacy. It is possible that this new approach could bring about improvements in the pain associated with knee osteoarthritis without the need for continued adherence to a muscle strengthening programme.

Trial Registration: ISRCTN51913166 (Registered 24-02-2020, Retrospectively registered)

Background

Knee osteoarthritis (OA) is a chronic long-term condition which results in pain, disability and reduced quality of life [1]. Estimates suggest that, in the UK, one in three people over 40 will develop knee pain within 12 years [2] and that 10% of the population over the age of 55 will be diagnosed with knee OA [3]. The prevalence of knee OA in the USA is over 4% [4] and this pattern is replicated across the world. Indeed, the global age-standardised prevalence for knee OA has been estimated to be 3.8% [4]. For many

individuals, symptomatic KOA will reduce their capacity to take physical exercise and lose weight, further exacerbating other comorbidities [5], such as cardiometabolic disorders. Furthermore, for many patients, conservative treatments do not provide sufficient long-term relief and they choose to undergo total knee replacement. However, as populations age and rates of obesity (a known risk factor [6]) rise, the increasing need for surgical management is putting healthcare systems under considerable strain. Given this huge societal cost, along with the individual suffering associated with the disease, there is an urgent need to explore new conservative methods to manage knee OA.

The universally recommended first line of clinical management for knee OA is a physiotherapist-delivered exercise programme. These programmes typically consist of muscle strengthening, advice to remain active [7] along with coping skills [8] and education about self-management. While this approach is supported by large-scales trials [9] and incorporated into national guidelines [10], the magnitude of clinical effect is considered moderate to small [11] and is known to diminish over time [12]. Exercise programmes which consist of two strengthening sessions per week [13], the minimum recommended by the ACSM [14], typically provide a 25-30% reduction in pain and/or function [9]. Furthermore, research has demonstrated that for approximately 40% of patients, exercise-based approaches do not provide any meaningful clinical [15] improvement in symptoms [9]. While adherence has been identified as an issue which may lower the true effectiveness of exercise-based approaches [16], it is unlikely to explain why, for a relatively large number of people, exercise provides no relief from knee OA pain.

The use of exercise-based approaches to manage knee OA has been a cornerstone of physiotherapy practice for over 30 years. During this period, there have been continual refinements in the frequency, duration and nature of the exercises [11] along with the addition of self-management components which aim to provide reassurance about the value of exercise in OA [17]. While there has been some focus on adjunctive therapies [18], such as cognitive-behavioural therapy [19, 20], there has been very little research investigating biopsychosocial interventions for knee OA [21], despite suggestions that this may improve clinical management [22, 23]. This is particularly concerning given the evidence of strong links between psychosocial factors and clinical pain/disability in this disease. For example, both catastrophising [24] and anxiety [25] have been associated with pain intensity, kinesiophobia linked to physical function [26] and emotional processing shown to mediate pain sensitisation [27] in people with knee OA.

While psychosocial factors can influence pain and function in knee OA, it is important to recognise the importance of mechanical factors. Over recent years, numerous experimental studies have demonstrated that patients with knee exhibit altered muscle coordination patterns, characterised by co-contraction of the hamstrings and quadriceps during daily tasks [28-30]. Whereas in a healthy person these muscles activate sequentially during walking, in individuals with knee OA these muscles contract simultaneously during the stance phase of gait. Recent studies have demonstrated the potentially damaging effect of these patterns, showing that increased co-contraction will increase the compressive loads on the joint surface [31], speed up the rate of cartilage loss [32] and increase the likelihood that patients will opt for a knee replacement at 5-year follow up [33]. Elevated co-contraction will increase stress on the articular

surface, on the bone, joint capsule and periarticular structures, and is likely to increased pain [34]. These findings indicate that co-contraction could be a potential intervention target [33].

In contrast to knee OA, the conservative management of low back pain (LBP), has evolved considerably over recent years. While core management originally consisted of muscle strengthening and exercise, a multidimensional approach addressing both biomedical and psychosocial factors is now widely used [35, 36]. Importantly, research has consistently shown that a large proportion of people with knee OA also suffer with LBP, with some estimates suggesting that this could as high as 57% [37]. While it is conceivable that knee pain leads to biomechanical compensation and altered spinal loading, it is also possible that there are common underlying psychosocial drivers which co-exist between these two conditions. If this is the case, then psychosocial approaches which have been applied successfully to people with LBP may prove effective for people with knee OA.

This paper describes the development of a new behavioural intervention for knee OA. This intervention integrates psychosocial concepts with emerging biomechanical theory relating to potential drivers of muscle co-contraction. The overall aim was to create an intervention which was which appropriate for facilitation by a suitably trained physiotherapist and was deliverable within UK NHS resources. In addition to describing the development process and final intervention, we also include some preliminary clinical findings.

Methods

The structure of the results section follows the guidelines for reporting intervention development studies set out by Duncan *et al.* [38]. Firstly, we report on the context, purpose, setting and target population (Section 1) after which we provide an overview of how published intervention development approaches contributed to our thinking (Section 2). In Section 3, we describe stakeholder contributions, and, in Section 4, we outline the theoretical ideas which underpin the new intervention. We then outline guiding principles which were prioritised during development (Section 5) and describe in detail the five components of the final intervention (Section 6). Section 7 provides insight into the evolution of the intervention after which we describe potential modifications for subgroups as well as uncertainties (Section 8). At the end of the results section, we present preliminary clinical findings (Section 9) and user perceptions (Section 10).

In order to develop our new intervention, we recruited 21 patients with knee OA, who received at least two face-to-face clinical sessions. Of these 21 patients, 11 received 5 or 6 sessions of a fully formed version of the intervention. Patients were included if they satisfied the ACR criteria [39] at the time of participation and had experienced knee OA pain for at least 6 months duration. In addition to the patients with knee OA, we recruited 45 health individuals in order to create a database of healthy EMG templates (see section 6). All participants provided informed consent to participate and ethical approval was obtained from a UK NHS research ethics committee (18/NW/0282). All procedures were performed in accordance with the Declaration of Helsinki.

Results

1 Context, purpose, setting and target population

Our aim was to develop a behaviour change intervention which specifically targeted both psychological and biomechanical factors related to pain in knee OA. The remit was to create an intervention which was suitable for delivery by an appropriately trained physiotherapist within a UK NHS outpatient clinic. As the UK NHS is a resource-limited healthcare setting, a total of six face-to-face clinical sessions was considered the maximum feasible duration. The aim was to create a physiotherapist-led intervention which would be appropriate for any level of knee OA severity, provided there was no significantly impairment in mobility, defined as an inability to walk at least 100 m unaided.

2 Overview of the intervention development process

We combined a range of different approaches in developing our new intervention [40]. Following the framework of intervention mapping [41] we performed a needs assessment in order to define specific changeable determinants of behaviour which had the potential to exacerbate pain in people with knee OA. Our needs assessment focused on published psychosocial research, emerging concepts on the drivers of muscle co-contraction and also included an exploration of patient's beliefs. The intervention was then iteratively developed in order to address each of the changeable determinants of behaviour using recognised behaviour change methods [42]. Our aim was to develop an intervention which was consistent with the COM-B (capability, opportunity and motivation) model as set out in the behaviour change wheel [43]. Throughout intervention development, we adopted a co-design/partnership approach to ensure that the views of users were fully represented.

Figure 1 illustrates the stages of intervention development. Following an in-depth literature review (Stage 1), we presented our findings to a group of four patients with knee OA and also a group of four physiotherapists. This consultation (Stage 2) allowed us to explore user perceptions of the theory and to understand beliefs and behaviours which were related to knee OA pain. An initial prototype of the intervention was then created (Stage 3). Between two and six sessions of this prototype intervention were delivered to 10 patients with knee OA by the lead physiotherapist (NB) (Stage 4). User feedback on this initial prototype (Stage 5) was obtained via three mechanisms: feedback directly to the physiotherapist after each session; interviewing of patients by a qualitative researcher; and through co-design workshops involving both physiotherapists and patients (see section below).

In order to respond to user feedback, the intervention was again refined/developed (Stage 6). This second iteration of the intervention was delivered to a further six patients (five-six sessions), again by the lead physiotherapist (Stage 4). Following this delivery, we used the same three mechanisms to obtain further user feedback (Stage 5), again refining the intervention as appropriate. At the end of this second iteration, the intervention was delivered to a further five patients (five-six sessions). During this final period of testing, only minor refinements were made in response to feedback made directly to the physiotherapist.

3 Stakeholders contribution to intervention development

Through our initial user consultation (Stage 2, Fig. 1), we explored patient's perceptions of their knee condition. This exploration was carried out following a presentation of the theory, allowing patients to contextualise their own experiences and to reflect on possible explanations for pain which were hitherto unknown to them. Discussion was then focused towards potential intervention components which patients would find engaging and which would fit with the COM-B model. Through consultation with physiotherapists, we were able to understand potential barriers and facilitators for delivery within the UK NHS. Discussions were analysed using a framework developed to understand the acceptability of healthcare interventions [44] and the findings used to specify changeable determinants of behaviour. The outputs from these discussions were also used to inform the guiding principles which were prioritised during intervention development. Through this process we created a specification for the initial intervention prototype.

In order to involve patients in the research process, we established a PPI (patient and public involvement) committee, which consisted of four individuals with a history (> five years) of knee OA. This committee met with the academic team every three-four months during the 18-month duration of the project and were consulted on various aspects of intervention development and research design, such as the format of the co-design workshops. Two members of the PPI group were included in the group of 10 participants who received the initial prototype of the intervention. However, no PPI members were included in the final 11 participants who received a fully formed version of the intervention and for whom we report clinical outcomes at the end of this section.

At the end of each physiotherapy session, patients were asked their opinion on the different aspects of the intervention and whether there has been any change in their pain-related beliefs. In addition, a subset of five patients were interviewed by an independent qualitative researcher to gain further insight into user perspectives and potential health benefits. With both these approaches, thematic analysis [45] was used to specify how the intervention could be improved. Three co-design workshops were held during intervention development (Stage 5, Fig. 1) involving at least four physiotherapists and at least four patients within each. At each workshop, the lead investigator presented the key principles of the intervention, after which the lead physiotherapist demonstrated the intervention on two patients. During the second part of the workshop, separate and combined, focus groups were held with patients and physiotherapists to understand user perspectives. Again, thematic analysis was used to develop specifications for subsequent iterations of the intervention.

4 Theoretical components

Our aim was to develop a behavioural intervention which integrated concepts from the fields of biomechanics, pain science and health psychology. One of the fundamental aims was to develop a behaviour change approach which would enable individuals to reduce co-contraction of the knee

muscles, as this is known to increase joint loading [31]. Two potential biomechanical mechanisms for co-contraction were identified from the literature. Firstly, that higher muscle activity on the lateral side of the knee could unload the medial compartment [31], which is most commonly affected by OA. However, modelling studies have disputed this theory [31]. The second mechanism explained co-contraction as a mechanism to increase knee stability in order to compensate for ligament laxity [46]. However, while this may be an effective strategy in response to a sudden perturbation [47], it is unclear whether such elevated muscle activation is required during everyday functional tasks. Instead, it is possible that more subtle changes in muscle timing and magnitude could be sufficient to stabilise the joint. Unfortunately, review of the literature related to these two mechanisms did not provide any indication of how to develop behaviour change approaches for decreasing knee muscle activation.

In parallel with our intervention development, research in our lab has sought to investigate the link between postural alignment and activation of the knee muscles. This work is based around a framework in which increased knee muscle activation is viewed as a direct consequence of subtle changes in postural alignment. In line with this idea, we have observed differences in knee muscle activation between groups of healthy people who habitually walk with small differences in trunk flexion [48]. We have also observed that people with knee OA walk with an increased flexion (forward lean) of the trunk [49]. Furthermore, we have shown that, when we instruct healthy people to increase their trunk flexion by a similar amount, knee flexor muscle patterns become similar to those associated with knee OA [50, 51] (Fig. 2). In this scenario, activity of the hamstring muscles is increasing in order to compensate for an anterior shift in the centre of mass. This idea of increased muscle activation as “biomechanical compensation” was subsequently adopted as a fundamental concept of the intervention.

Given the potential links between postural alignment and knee muscle patterns [48, 50], we reviewed the finding of studies investigating postural alignment in knee OA. Unsurprisingly, numerous studies showed altered postural alignment, characterised by a flexed posture [52], poor lumbo-pelvic alignment [53] and increase in forward spinal inclination [54, 55]. Concepts of muscle imbalance are well established within the field of physiotherapy [56, 57]. With this paradigm, altered postural alignment is believed to be the result of an alteration in inter-segmental muscle tension or muscle length [58]. For example, a shortened (or passively stiff) hip flexor muscle will limit posterior pelvic rotation (pelvic tilt) [59], preventing the pelvis from returning to a neutral position in upright standing (Fig. 3).

Without any biomechanical compensation, a short hip flexor will create an increase in forward spinal inclination. However, it is unlikely that an extremely flexed position would be adopted. Instead, it is possible an individual with a short hip flexor will “biomechanically compensate”, by flexing the hip, knee and ankle and by increasing lumbar lordosis (Fig. 3). This idea is consistent with the observation of a more flexed posture at all joint levels in people with knee OA [52]. It is important to note that the widely accepted concept of muscle imbalance is based on the idea of an imbalance between the strength and flexibility of the agonist or antagonist muscle acting over a joint [58]. However, our framework was based around the idea of postural abnormality being the result of chronic shortening of muscles, such as the hip

flexors, which attach directly onto the pelvis, and the associated biomechanical compensations required to stand upright and establish normal gaze alignment.

One mechanism which could explain why hip flexor muscles could become shortened/stiff is chronic understretch [60, 61], which may result from sedentary behaviour. Our research supports this idea, showing limited passive hip extension in healthy people who sit for prolonged periods and who are physical inactive [62]. We have also observed lower passive hip extension in people with knee OA, when compared to healthy controls [63], demonstrating that people with this disease have stiffer hip flexor muscles. Building on the ideas outlined above, our research has also shown a relatively strong link between trunk flexion angle in walking and passive hip extension [63]. This demonstrates that, in both healthy people and those with knee OA, increased passive stiffness of hip flexor muscles is associated with an increased forward inclination of the spine in walking. Given this observation, and the ideas presented above, we adopted the framework shown in Fig. 4, in which increased knee muscle activation is explained as a biomechanical compensation for an increase in passive stiffness of hip flexor muscles, which results from sedentary behaviours.

Other research focused on sedentary behaviour has demonstrated an association between increased sitting/physical inactivity and a reduction in the active range of transverse motion of the thoracic spine. [64]. While it is conceivable that physical inactivity may impact on ligamentous/osseous structures, we suggest that these finding indicate that prolonged sitting/physical inactivity results in increased stiffness of abdominal muscle structures which must lengthen to facilitate spinal rotation. Importantly, increased stiffness (or shortening) of the abdominal muscles will reduce the capacity for the rib cage to move superiorly relative to the pelvis [65] and could therefore affect postural alignment in standing. Similar to the ideas discussed above, such changes could trigger compensatory muscle activity to stand upright and maintain gaze alignment. This idea is depicted in Fig. 5.

There is now growing support for the concept that acute motor responses to pain, although protective in the short term, can lead to long-term adaptations which can perpetuate the pain experience [66]. In line with this idea, it is possible that muscle co-contraction may function to improve knee stability following acute injury [47]. However, increases in knee muscle co-contraction have been associated with increased contact force [67], accelerated loss of articular cartilage [32] and poor long-term clinical outcomes [33]. Therefore, it would appear to be a maladaptive motor response if maintained in the long term. While we have presented postural mechanisms to explain increased co-contraction, there is likely to be a degree of pain-related muscle contraction. Such elevated knee muscle activation is likely to increase nociceptor input, further exacerbating pain. Research supports the idea that such motor adaptations to pain are mediated through supraspinal mechanisms [68]. If this is the case, then they should be modifiable through effective behavioural interventions. Therefore, we adopted the framework shown in in Fig. 5, in which knee pain triggers increased knee muscle activation which in turn increases nociceptor input, triggering yet more pain. This idea is consistent with the idea of guarding behaviours in low back pain [69] and the pain-spasm-pain model which has also been studied in relation to low back pain [70].

There is now strong evidence to support the idea of central sensitisation to pain in people with knee OA [71, 72]. This can occur through a range of mechanisms, such as amplification of afferent impulses coming from the peripheral receptors, alteration of sensory processing in the brain and loss of descending antinociceptive mechanisms [73]. While pathophysiological mechanisms underlying central sensitisation are complex, the ultimate effect is amplification of neural signalling which results in generalised pain hypersensitivity (hyperalgesia) [73] and a decrease in pain threshold to normally non-painful stimuli (allodynia) [74]. While intense and continued nociceptive input is known to cause central sensitisation [75], it is also possible that emotional responses to pain [74] or pain expectations can influence sensitisation. This idea is consistent with research which has shown that knee OA pain activates areas of the brain which are involved with the processing of fear and emotions [76] and also with research which has demonstrated that psychosocial factors can mediate the association between hyperalgesia and knee pain [27].

Cross-sectional studies have shown links between a range of psychosocial factors and knee OA related pain. Characteristics such as pain catastrophising [77], kinesiophobia (fear of movement) [24], helplessness [78], self-efficacy [79], anxiety [25] and depression [25] have all been shown to be associated with clinical pain. It is possible that these factors may lead to avoidance of activity [80, 81] with associated physical deconditioning and this may explain, to some degree, the observation that psychosocial factors are also associated with impairments in physical function [26]. Given the well-established links between psychosocial factors and pain and the finding that psychosocial factors may influence central sensitisation [27], we postulated the existence of a three-way relationship between pain, emotional responses and central modulation of the pain experience (Fig. 6). With this idea, pain shapes beliefs and emotional responses, which in turn affects pain sensitisation, and the pain experience. This concept is consistent with research demonstrating that teaching skills to reduce negative pain-related thoughts and emotions can improve knee OA pain [82].

Figure 8 shows the fully integrated behavioural framework, obtained by combining the different mechanisms described above (Figs. 4–7). This framework shows the interrelationship between the postural mechanisms (Figs. 4 & 5), the maladaptive motor responses (Fig. 6) and the effects of central sensitisation on the pain experience (Fig. 7). It is important to stress that our proposed framework is not a comprehensive model to explain the onset of knee OA. Clearly, OA pain may have many origins, such as ligament rupture or other traumatic injury [83, 84]. Instead, we have attempted to include factors which could exacerbate knee pain, may relate to patient's beliefs, and which could be targeted through an effective behaviour change intervention.

Following the development of the theoretical framework (Fig. 8), we consulted with patients (Stage 2, Fig. 1) to identify beliefs which were related to their knee pain. Core concepts were presented from the framework and patients encouraged to contextualise their own pain experiences. A set of five beliefs (Table 1) were identified which were subsequently confirmed through further consultation, carried out during intervention testing (Stage 5, Fig. 1). While there was general consensus on the first three beliefs (Table 1), some patients did acknowledge the value of exercise and did not believe that exercise would

accelerate the disease process. Importantly, there were clear inter-individual differences in the degree to which patients were fearful or anxious about their pain, highlighting the need for an intervention which was tailored to individual beliefs.

Table 1
Patient's beliefs about their knee OA pain

	Pain-related belief
1	My knee OA pain is the inevitable result of wear and tear on the joint and is part of getting old
2	Certain activities (e.g. going downstairs) cause high levels of pain and so I need to avoid them wherever possible
3	Knee OA is a chronic condition and so the pain is something I am always going to have and may get worse in the future
4	I am fearful of my pain and is something which I cannot control
5	Too much exercise will increase the amount of wear on the joint and may mean that I need knee replacement sooner

The idea that muscular co-contraction could increase the stress on the knee joint resonated with most patients. Furthermore, patients were encouraged by the idea they could lean to move in a way which reduced pressure on their knee. When asked to reflect, patients felt that they were likely to tense their muscles in response to pain but that it was not something they had consciously thought about previously. The beliefs identified through our user needs assessment (Table 1) fit with the fear-avoidance model of musculoskeletal pain [85]. Specifically, we identified that pain was viewed as a wear and tear processes, that patients avoided certain activities and that escape/avoidance behaviours were apparent. By combining our understanding of patient beliefs (Table 1) with the theoretical framework (Fig. 8), we developed set of changeable determinants of behaviour. Before we describe this development, we highlight guiding principles for intervention development which were established as part of our consultation.

5 Guiding principles prioritised during intervention development

In order to facilitate patient learning, and increase capabilities and motivation, we attempted to draw on digital technologies wherever possible. In response to user suggestions, we worked with a local animation studio to create a range of instructional videos which were used to convey concepts integral to the intervention, such as those related to muscle function, postural control, pain science and pain-related beliefs. Each participant was provided with a subset of videos which, tailored (by the physiotherapist) to their individual needs. The format of each clip was the same, being approximately 1 minute in length and finishing with a question which reflected the learning outcome, e.g. "Do you understand that increased knee muscle tension could make your knee pain worse?" Participants watched the videos on a tablet

computer and had the option of repeating each clip if they did not fully grasp the learning outcome. To facilitate motor relearning, we used EMG biofeedback [86] from the knee muscles. To optimise usability, we created our own software which could be used to visualise simple on-off activity or, alternatively, used to contrast an individual's muscle pattern with a healthy average EMG profile for a given functional task.

One of our principle aims was to create a behaviour change intervention which would not require longer-term adherence to a prescribed programme of exercise, and which was consistent with the COM-B model. Rather than conditioning the knee muscles, our intervention was designed to change beliefs about pain and to change postural and functional muscle patterns, providing patients with capability, opportunity and motivation to self-management their condition. In this context, the clinician's role was of that of an educator, rather than a therapist. By developing an empathic relationship with the patient, the aim was to guide patients through a tailored, incremental learning process. While patients were required to practice certain procedures in the short-term to facilitate this learning, the ultimate aim was for patients to be able to integrate this learning into daily activity without the need to set aside specific time each day to practice. A relatively rapid transition from self-directed practice into daily activity was felt to be critical given that exercise adherence has been identified as a major barrier in the physiotherapy management of knee OA [16, 17].

6 Intervention components

In response to the integrated theoretical framework (Fig. 8) and the set of pain-related beliefs (Table 1), we mapped a set of five changeable determinants of behaviour (Table 2). Using the taxonomy of behaviour change methods [42], we then identified behaviour change techniques which were appropriate for each determinant (Table 3) and which were incorporated into our five intervention components. These components were: making sense of pain, general relaxation, postural deconstruction, responding differently to pain and functional muscle retraining. Each intervention component was associated with several determinants of behaviour change, corresponding techniques (Table 2) and was specifically tailored to the individual patient. The following text details each separate intervention component, explaining how the different behaviour change techniques (Table 3) were applied and how the component was individually tailored.

Table 2
Changeable determinants of behaviour, behaviour change methods and corresponding intervention components. Each determinant has been mapped back to the COM-B model of behaviour change. (COM-B refers to capability, opportunity and motivation)

Changeable determinant of behaviour	Behaviour change technique	Intervention component	COM-B
Recognise that increased knee muscle activation will increase load on the joint, potentially exacerbating pain.	Persuasive communication Using imagery	Making sense of pain	Motivation to engage in re-learning of muscle patterns
Recognise that emotional factors can impact on central sensitisation and affect the pain experience.	Persuasive communication Using imagery Consciousness raising	Making sense of pain	Motivation and opportunity to challenge pain-related beliefs
Develop awareness of acute muscular response to pain (e.g. knee bracing) and be able to consciously influence these patterns.	Consciousness raising Counterconditioning Bio(Feedback)	Making sense of pain General relaxation Responding differently to pain	Capability and opportunity to change muscular responses to pain
Understand the concept of biomechanical compensation and be able to reorganise postural muscle activity in order to prevent knee muscle activation in standing.	Using imagery Consciousness raising (Bio)Feedback	General relaxation Postural deconstruction	Capability and opportunity to change muscular control of posture in standing
Develop the ability to reduce muscular overactivity during functional tasks, such as walking.	Using imagery Consciousness raising Counterconditioning Bio(Feedback)	Responding differently to pain Functional muscle retraining	Capability and opportunity to change muscular coordination in everyday tasks

Component 1: Making sense of pain

We used persuasive communication and imagery (through animated videos) to challenge the belief that knee OA pain is the inevitable result of “wear and tear” and to convey the idea that increased muscle activation will increase knee loads, potentially exacerbating pain. See http://hub.salford.ac.uk/tension-release-therapy/making-sense-of-pain-video/ for an example. We also created animated videos to explain that “tensing muscles” in response to pain could further exacerbate pain. Drawing on the proven efficacy

of neuroscience education [87], we created educational materials [88] to explain the idea that brain processing will modulate the pain experience; that pain is not always a true representation of the status of tissues; and that psychosocial factors can modulate the pain experience [87]. We emphasised the idea of “escaping” from vicious cycles, both in terms of maladaptive muscular responses to pain (Fig. 6) and central sensitisation (Fig. 7), and the need to raise consciousness of habitual responses to pain. This component was tailored by increasing/decreasing the focus on central sensitisation which was determined by applying established principles for the recognition of central sensitisation in musculoskeletal pain [89].

Table 3
The five primary behaviour change methods

Behaviour change method	Definition
Persuasive communication	Guiding individuals toward the adoption of an idea, attitude, or action by using arguments or other means.
Using imagery	Presenting information in a pictorial format will aid the communication of conceptual ideas and facilitate the learning of new motor patterns
Consciousness raising	Providing information and feedback about the causes, consequences, and alternatives for a problem behaviour.
Counter conditioning	Encouraging the learning of healthier behaviours that can substitute for problem behaviours.
Feedback	Giving information to individuals regarding the extent to which they are accomplishing learning or performance.

Component 2: General relaxation

There were two primary aims to teaching general relaxation, firstly to initiate the process of postural deconstruction (see below) and secondly to raise consciousness of both emotional and muscular responses to pain. This focus is consistent with previous research supporting the use of relaxation interventions for arthritis pain [90] and the use of cognitive behavioural therapy interventions which incorporate relaxation for knee OA [20]. We specifically targeted three easy-to-observe characteristics of relaxation. The first was active contraction of the quadriceps muscles which was monitored using either EMG biofeedback or a patella glide test. Secondly, we monitored the degree of resistance to passive limb movement when the patient was lying supine. Finally, we monitored low level contraction of the abdominal muscles (focusing primarily on rectus abdominus) using manual palpation, along with a modified version of the Hi-Lo breathing assessment [91]. For the breathing assessment, reduced movement of the abdomen was taken as secondary confirmation of increased abdominal muscle tone, which has been associated with reduced abdominal volume during respiration [92].

The abdominal assessment was carried out in supine lying and in standing. Interestingly, all 21 participants demonstrated elevated abdominal tone in standing along with a tendency for scapula elevation when instructed to take a deep breath. To communicate the idea that increased abdominal tone

would alter breathing patterns, we created an animated video which can be accessed at http://hub.salford.ac.uk/tension-release-therapy/example-breathing-video/. To train relaxation of the abdominal muscles, patients were provided with a set of instructional videos which contrasted the altered breathing pattern with the target pattern (full diaphragmatic movement with minimal shoulder motion). These videos explained the mechanics of breathing and guided the development of a diaphragmatic breathing pattern, first in lying and then in standing. To reduce resistance to passive limb movement, the physiotherapist worked with the patient in a supine position to facilitate awareness of muscular holding. Using EMG biofeedback (or patella glide), patients were taught to develop a sense of relaxing-contracting the quadriceps, first in supine/sitting and then in a standing position. This intervention component was tailored by using customed breathing retraining and by the degree of focus on the teaching of release of resistance to passive limb movements.

Component 3: Postural deconstruction

Given the potential link between compensatory postural muscle activity and knee pain (Sect. 4), the primary focus of this component was on postural tone. Whereas postural alignment describes the relative orientation of body segments, postural tone can be thought of as “tonic (sustained) activation of muscles in order to provide specific postural attitude and generate force against the ground to keep the limbs extended” [93]. Based on the framework in Fig. 8, we suggest that passive stiffness of the hip flexor muscles and/or abdominals will drive a re-organisation of postural tone, through a set of compensatory increases in tonic muscle activity required to maintain upright standing and establish normal gaze direction. In line with this idea, the task for the clinician is to unpick (deconstruct) patterns of postural tone, facilitate the resetting of proprioceptive relationships between body segments and to re-establish a more optimal balance of postural muscle activity. While there are some parallels between this approach and the use of muscle imbalance physiotherapy to improve postural alignment [58], we stress that our approach has no focus on muscle strengthening. Instead, it teaches patients to make subtle changes to postural muscle activity through body-awareness training.

We devised a clinical protocol incorporating manual muscle testing, palpation and visual assessment along with EMG assessment of knee muscle activity. This enabled the clinician to identify hip flexor/abdominal stiffness and interconnected patterns of compensatory postural tone. Following this assessment, animated instructional videos were used to provide the patient with a conceptual understanding of their individual muscle/postural patterns. Patients were then guided through a set of procedures, the first of which required them to move from a flexed to an upright position. Performed slowly, this allowed the physiotherapist to identify the “tension point” at which there was a marked increase in tonic muscle activity in order to overcome hip flexor or abdominal stiffness. At this point, there was often an observable increase in quadriceps EMG which was visualised using the biofeedback software. Patients were then guided through a range of procedures, using abdominal breathing, segmental dissociation, positional awareness and gentle stretching to both reduce hip flexor and/or abdominal stiffness and raise awareness of compensatory postural tone. Following these procedures, the aim was to achieve an upright standing position without triggering compensatory tone.

This intervention component was highly tailored and based on an individualised assessment, the use of individually selected videos and an individually developed retraining plan. To illustrate the idea of postural deconstruction, Fig. 3 shows how compensatory tone can be triggered in the knee extensors, secondary to passive stiffness of the hip flexors. In Fig. 3a, a flexed position is adopted, and the hip flexor is in a slack position. Figure 3b shows the tension point at which there is no slack in the hip flexor, but an upright position has not been achieved. Figure 3c show compensatory knee flexion required to facilitate posterior pelvic rotation and upright standing. This knee flexion will trigger activation of the knee extensor muscles (compensatory tone). In this scenario, retraining would involve a focus on positional awareness of the knee, hip flexor lengthening/relaxation and a focus on moving past the tension point without triggering compensatory knee muscle contraction (visualised using EMG biofeedback). In addition, the clinician would focus on compensatory tone in the spinal erector muscles, which may be triggered to increase lumbar extension in order to achieve an upright position. EMG activity in the hamstring muscles would also be monitored as this may increase to balance any anterior shift in the centre of mass.

In our original framework (Fig. 8), increased passive stiffness of the hip flexor or abdominal muscles was associated with physical inactivity and prolonged sitting [62, 64]. This concept was explained to patients and they were encouraged to take regular walking exercise and break up periods of prolonged sitting by walking or standing for a few minutes. As part of this component, the physiotherapist challenged any beliefs relating to exercise avoidance. Patients were also encouraged to observe how postural tone in sitting, such as low-level hip flexor activity [94], might be carried through into standing, especially after sitting for prolonged periods. In addition, patients were encouraged to use the enhanced body awareness they had developed during the process of postural deconstruction to “make sense of stiffness.” Specifically, patients learned to recognise the difference between intrinsic joint stiffness (resulting from limitations in the joint and capsular structures) and muscle stiffness, which may result from prolonged periods of inappropriate patterns of compensatory postural tone.

Component 4: Responding differently to pain

Building on component 1 (making sense of pain), this component made use of EMG biofeedback to identify the presence of knee muscle contraction which was related to pain, pain expectations or pain-related beliefs. Using animated videos, we reiterated the idea that inappropriate muscle contraction could perpetuate the pain experience. EMG biofeedback was then used to raise consciousness of knee muscle activity in standing and to explore how expectations from pain provoking activities could trigger excessive muscular contraction. For example, many patients found stepping down painful and would over activate their knee muscles in anticipation of pain from this movement. Using EMG biofeedback, patients could observe this behaviour and were taught to ‘downregulate’ muscle activity before such tasks. In addition, we recognised that localised changes in knee muscle activity, related to previous pain experience, may trigger compensatory postural tone. Therefore, learning from this intervention component was integrated into the postural deconstruction procedures, describe above.

We suggest that this approach, of teaching patients to observe and consciously influence their muscular responses to anticipated pain, has parallels with cognitive restructuring techniques which have been used

previously in CBT programmes [95] for knee OA [19]. Specifically, the increased muscle activation is seen as a maladaptive pattern (problem behaviour), which can be brought into conscious awareness so that it can be substituted for a healthy behaviour (more relaxed knee muscle) through counter conditioning. Such muscle patterns are likely to be connected to past experience and beliefs about pain. Therefore, the clinician used this opportunity to continue to challenge patient's beliefs around the chronic nature of knee OA pain and also challenge the idea that certain movements should be avoided. Through this process patients were continually encouraged to reflect on their fear/anxiety about pain and to develop a sense of how such responses could be linked to maladaptive muscular behaviour. This component of the intervention was tailored by allowing the patient to choose the pain provoking movements and the degree to which the emotional beliefs underlying these patterns were explored during clinical sessions.

Component 5: Functional muscle retraining

This final component of functional muscle retraining was carried out once patients had developed competence in postural deconstruction and were able to minimise maladaptive muscular responses to pain. The first stage of this training involved the use of EMG biofeedback to enable patients to develop the ability to momentarily balance on one leg with minimal activation of knee muscles of the stance limb, as if about to initiate a step forwards/up/down. Once this had been mastered, the focus was on a set of patient-selected functional tasks (walking, stepping down, stepping up, sit-to-stand or stand-to-sit). For this second stage, the patient's EMG muscle profile was initially visualised against a healthy template, created from an EMG database of signals collected from a cohort of 45 healthy volunteers (aged 18–83). This visualisation provided the patient with a conceptual understanding of how their muscle patterns differed from those of a person without knee pain.

With a conceptual understanding of their knee muscle pattern, patients were instructed to repeat the selected task and to mentally experiment with a specific motor command. The precise choice of motor command was designed to normalise the muscle pattern and therefore selected based on the timing/magnitude of the patient's EMG signal. In some cases, animated videos were used to help convey the desired motor commands, using motor imagery [96]. For example, many participants exhibited prolonged quadriceps activity into midstance of walking (see Sect. 9). By using an instruction such as "imagine a rope pulling the leg forwards as you walk", the patient learned to associate the specific motor command with the desired motor behaviour, i.e. match their muscle pattern with the healthy template in the biofeedback software. In addition to using motor commands aimed at encouraging smooth movement, kinematic instruction was also employed where appropriate. For example, patients were discouraged from positioning their feet too anteriorly when performing a sit-to-stand movement as this requires greater acceleration of the centre of mass on movement initiation [97]. The functional muscle retraining was highly tailored as it used knowledge of patient's muscle activation (EMG signals) to create an individual training programme.

Intervention schedule

The final intervention was delivered as a course of six one-to-one clinical sessions (one every two weeks), each lasting 45–60 minutes and which was augmented with specific tasks that patients completed outside of contact sessions. The first clinical session typically covered making sense of pain (component 1) and relaxation (component 2). In sessions 2–4, this material was revised, and the patient taught postural deconstruction (component 3) and responding differently to pain (component 4). In the final two sessions, there was more focus on functional muscle retraining (component 5), however, this was determined on individual needs. Outside clinical sessions, patients practiced relaxation, postural deconstruction and the use of specific motor commands to influence muscle patterns. They were also encouraged to take regular exercise and to notice their emotional and muscular responses to pain. The ultimate aim of the intervention was to create capability, opportunity and motivation to change behaviour. In line with this philosophy, patients were instructed to gradually integrate as many of the ideas and practices into their daily life, removing the need to dedicate specific time each day to practice.

7 The evolution of the intervention

One of the principle changes that occurred during the development process was the way in which the intervention was delineated into different components. In the initial prototype, we defined intervention components which aligned with specific aspects of the development work, such as explanatory videos, instructional animations and biofeedback software. However, as the intervention developed, the components were redefined in order to align with learning objectives, such as making sense of pain, general relaxation and functional muscle retraining. In line with this idea, we moved from the original concept of an introductory video for patients, to explain psychosocial and biomechanical concepts, towards the practice of producing several short clips (< 1 minute) followed by a learning outcome. As the intervention progressed, the clinician was able to add these clips to a playlist on tablet computer (provided to the patient), gradually increasing the information that patients were required to digest after each session.

A major part of the development work was focused on the clinical procedures which formed the basis of the postural deconstruction component of the intervention. After experimenting with numerous strategies, we found that the idea of a “tension point” to be the most effective way to link our conceptual framework with patient’s kinaesthetic understanding. In line with the focus on postural tone, this idea shifted the objective from that of achieving a distinct postural alignment to a focus on the muscles used to stand erect. Another part of the developmental work focused on the functional muscle retraining. While our original idea had been to guide patients through a set of incremental activities for each task, it proved difficult to break down complex functional movements, such as walking. Therefore, following the preliminary balance training, the use of guided imagery was found to be the most appropriate method for changing motor patterns.

8 Intervention modification for subgroups and potential uncertainties

Musculoskeletal comorbidities are common in people who present with knee OA [37]. As our intervention addresses a range of psychosocial factors and whole-body patterns of postural tone, it would be appropriate for subgroups who have co-presenting musculoskeletal pain. Nevertheless, many people affected by knee OA experience a level of disability so great that it requires them to use a walking aid. While not within the remit of our original target population, further development of the intervention would be required in order to create suitable modifications for people with knee OA who have severe disability.

Through a rigorous intervention development process, we defined a set of five intervention components. However, the intervention was designed specifically for a UK NHS setting and therefore we did not investigate the potential of adapting the number of intervention sessions on an individual basis, which may lead to more optimal clinical outcomes. If extended delivery is feasible in other healthcare settings, we would suggest the use of the same five intervention components with increased repetition and relatively straightforward expansion of the learning activities. However, further development work would be required for this. Another potential uncertainty is the training a physiotherapist would require in order to develop a level of proficiency sufficient to deliver the intervention. However, wherever possible, we created supplementary material to facilitate patient learning, used established psychosocial techniques for the management of chronic musculoskeletal pain and used existing clinical physiotherapy assessment methods. Therefore, we suggest that a relatively short training course should prove sufficient and we are currently exploring how to design such a course.

9 Preliminary clinical results

The final 11 participants received five or six sessions of the intervention which, although not finalised, was considered sufficiently formed for clinical delivery. For this group (six male), the mean (SD) age was 60 (9) years, weight 83.7 (18.2) kg and height 1.72 (0.08) m. All satisfied ACR criteria all had a previously confirmed radiographically diagnosis of knee OA (KL grade unavailable). KOOS data were collected from each participant at baseline, 12 week follow up (immediately after the final intervention session) and at a long-term follow up (between 9–15 months from baseline). In addition to KOOS pain, we calculated the corresponding WOMAC pain from the final 5 items of the KOOS pain scale in order to facilitate comparison with other studies.

Table 4
Mean (SD) change in pain and function across 11 patients

		12 week follow up			9–15 month follow up		
	Baseline measure	Follow up	Change from baseline	% change	Follow up	Change from baseline	% change
KOOS pain	16.8 (5.1)	7.7 (4.0)	9.0 (3.3)	55 (17)	9.1 (7.4)	7.7 (6.4)	46 (41)
KOOS function	24.7 (13.6)	7.0 (4.5)	17.7 (12.2)	68 (23)	14.3 (12.7)	10.5 (15.3)	35 (50)
WOMAC pain	8.2 (2.4)	2.6 (2.1)	5.6 (2.2)	69 (20)	4.0 (4.1)	4.2 (3.7)	53 (41)

There were large changes in both pain and function immediately following the intervention (Table 4), with a reduction of 55% in KOOS pain, 68% in KOOS function and 69% in WOMAC pain. All participants reported an improvement in WOMAC/KOOS pain above the minimal threshold of 15% [15], with individual improvements in KOOS pain ranging from 33–88%. While reductions in average pain appeared to be maintained at long-term follow up, the improvement in KOOS function declined. However, these follow-up data were collected after a two-month nationwide UK lockdown (due to Covid-19) which is likely to have had an adverse effect on individuals' ability to self-manage their condition. Furthermore, it is important to stress that, while the 11 participants received five-six intervention sessions, the intervention had not been finalised and it is therefore these data may provide an indication of the minimum effect.

10 Patient perceptions of the intervention

Feedback from patients, captured during the final co-design workshop, was universally positive. Patients reported that the intervention allowed them to understand and challenge the way they move and react to pain. They also described the process as allowing them to "create a new normal", to be "in control of their own treatment" and to feel like they were addressing the "cause not the symptoms." Patients also commented positively on the "holistic approach" and explained that this put the "patient at the centre rather than the health professional or the treatment". One participant described the intervention as "genuinely life changing" as it had had both a psychological and physical impact and had resulted in her feeling "more energised."

Following interviews with three participants, who were within the final 11 patients receiving the fully formed intervention, a number of themes emerged. Firstly, that the intervention had "changed mind and body", giving them a new level of conscious awareness of their body movements. Secondly that "understanding is the key" and that the use of animated videos and EMG biofeedback was invaluable. When combined with individual discussion, this enabled patients to "reset expectations" about their knee pain and give them a new feeling of "responsibility" for their condition. A third theme related to the need

to “keep going with the new me”, recognising the importance of continued awareness in daily life. Finally, the importance of an empathic and positive attitude of the therapist was recognised as being crucial in changing patient’s beliefs about pain and guiding them through the learning process.

Discussion

To our knowledge, there have been no previous attempts to combine biomechanical and psychosocial treatment principles for knee OA. Our intervention is therefore highly novel, given the intervention of psychological pain management techniques with muscle biofeedback training. However, we acknowledge that some of the biomechanical underpinnings for the intervention are based on emerging concepts rather than on unequivocal evidence. Nevertheless, our preliminary clinical findings and positive user feedback motivate further research to investigate the proposed biomechanical framework and better understand the links between pain processing and muscle patterns. Interestingly, a recent study demonstrated a link between central sensitisation to pain and muscle co-contraction [98] in people with knee OA, supporting the idea of shared central mechanisms. This finding is encouraging and is consistent with the underlying framework for our new intervention.

A recent review concluded that individual’s beliefs about chronic knee pain will shape their attitudes and behaviours about how they manage their pain [17]. In line with this idea, our intervention was specifically designed to challenge patient’s beliefs and to provide a framework to explain the exacerbation of pain through mechanisms related to muscle overactivation and central pain processing. This approach contrasts with exercise-based approaches [11], which are likely to bring about clinical benefits through other mechanisms, such as those related to strength-related increases in functional capacity and strength-related confidence to perform activities of daily living [99]. Other emerging treatment paradigms for knee OA focus on gait retraining through the use of simple instruction to change foot progression angle, alter step width or medialise the knee position [100]. However, while these approaches have been shown to reduce the load on the medial compartment [100], they are associated with increased muscle co-contraction [101]. In contrast, our intervention was specifically designed to reduce elevated muscle activity. It is possible that this treatment target may lead to a reduction in focal bone loading, which has been linked to pain in knee OA [102]. However, further research is required to explore this idea.

We acknowledge that, while our proposed framework incorporated physical exercise, it did not specifically address other social and lifestyle factors. For example, factors such as obesity [103], sleep disturbance [104] and possible stressful life situations [105] which have been linked to chronic pain. Delineation of these boundaries was deemed appropriate in order to create an intervention which could be delivered within six face-to-face sessions. Nevertheless, we are confident that it would be straightforward to augment our intervention with other approaches for addressing general health, social and lifestyle factors, such as weight management, cognitive behavioural therapy and more extensive social support. However, we would emphasise that our intervention is designed to teach patients how to manage and react to pain differently and should therefore facilitate improved self-management across a range of social contexts.

In their recent consultation with intervention developers, Turner et al. [106]. identified a range of factors considered to indicate successful intervention development. Consistent with the factors they identified, we are confident that our intervention has been created using appropriate research evidence, incorporates stakeholder's views and is scientifically robust. We are also confident that it is appropriate for the UK healthcare system as it has been designed for delivery by a physiotherapist using relatively inexpensive equipment (EMG system, laptop and tablet computer). Following extensive user consultation, we are confident the intervention is acceptable to both patients and practitioners, meeting needs of the end user. Further testing is now required to understand potential effectiveness across different healthcare settings.

Conclusion

We have created a completely new behavioural intervention for knee OA which integrates ideas from pain science, biomechanics and health psychology, and which can be delivered by a physiotherapist. The intervention contains a focus on changing functional muscle patterns and teaching patients about how their beliefs and behaviours can shape the pain experience. The intervention is consistent with the COM-B model of behaviour change. User feedback was incredibly positive. However, while encouraging, our preliminary clinical data does not constitute proof of effectiveness. Therefore, larger trials are now required to understand whether this intervention could bring about long-term improvements in the pain associated with knee OA when delivered either within the UK NHS or other healthcare settings.

Declarations

Ethics approval and consent to participate

The following statement was provided on ethical approval in the methods section "…ethical approval was obtained from a UK NHS research ethics committee (18/NW/0282)." A statement was also provided in the methods section on consent to participate, "All participants provided informed consent to participate."

Consent for publication

Not applicable

Availability of data and materials

Full KOOS data collected from the final 11 participants can be downloaded at the following link: https://drive.google.com/file/d/1XYkj6PvWZ9sZHaFVdfduImdj5D4LvxoN/view?usp=sharing. Note that we will move this file to Figshare data repository should be manuscript be accepted for publication.

Competing interests

The authors declare that they have no competing interests.

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Authors contributions

SJP conceived the original study idea, performed the literature review, synthesised the underlying concepts, consulted with users to develop the final intervention and drafted the final manuscript.

NB synthesised the underlying concepts, collected laboratory data, delivered successive iterations of the intervention to the participants, consulted with users and commented on the final manuscript.

AEW consulted with users, interpreted qualitative data, refined the intervention and contributed to the drafting of the final manuscript.

RKJ synthesised the underlying concepts, assisted with data interpretation and made substantial revisions to the final manuscript.

CS created biofeedback software, collected laboratory data and contributed to data interpretation.

AJ synthesised the underlying concepts, assisted with data interpretation and contributed towards drafting of the final manuscript.

NEW consulted with users, contributed to the synthesis of underlying concepts, interpreted qualitative data and contributed to the drafting of the final manuscript.

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Figures

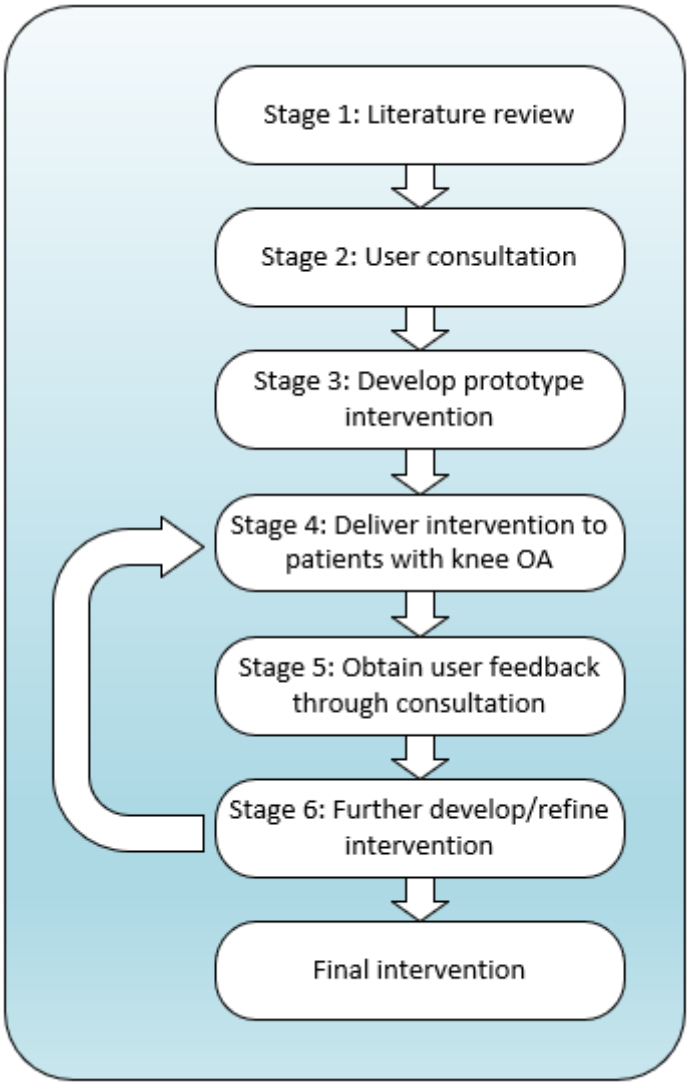

Figure 1

Schematic diagram to show the stages of intervention development

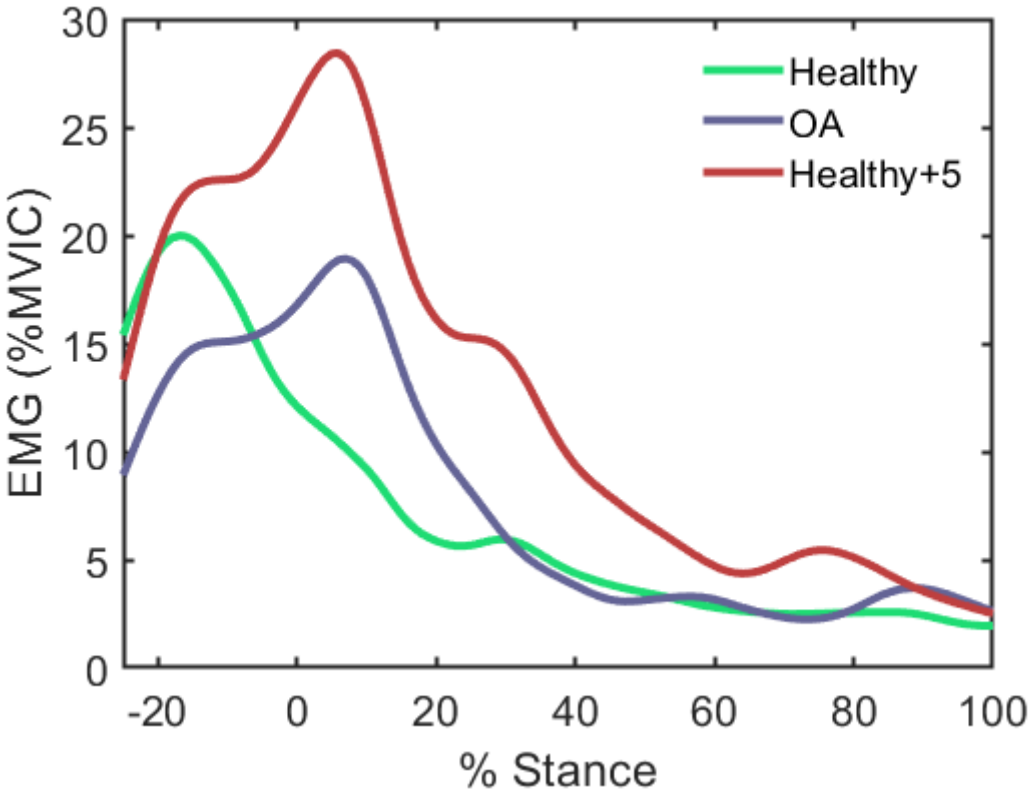

Figure 2

Medial hamstring EMG during walking in people with knee OA (blue), in healthy people (green) and in healthy people after instruction to increase trunk flexion by 5° (red). Note how the muscle pattern in the healthy people changes dramatically, becoming similar to the OA pattern, with increased trunk flexion. MVIC refers to maximal voluntary isometric contraction

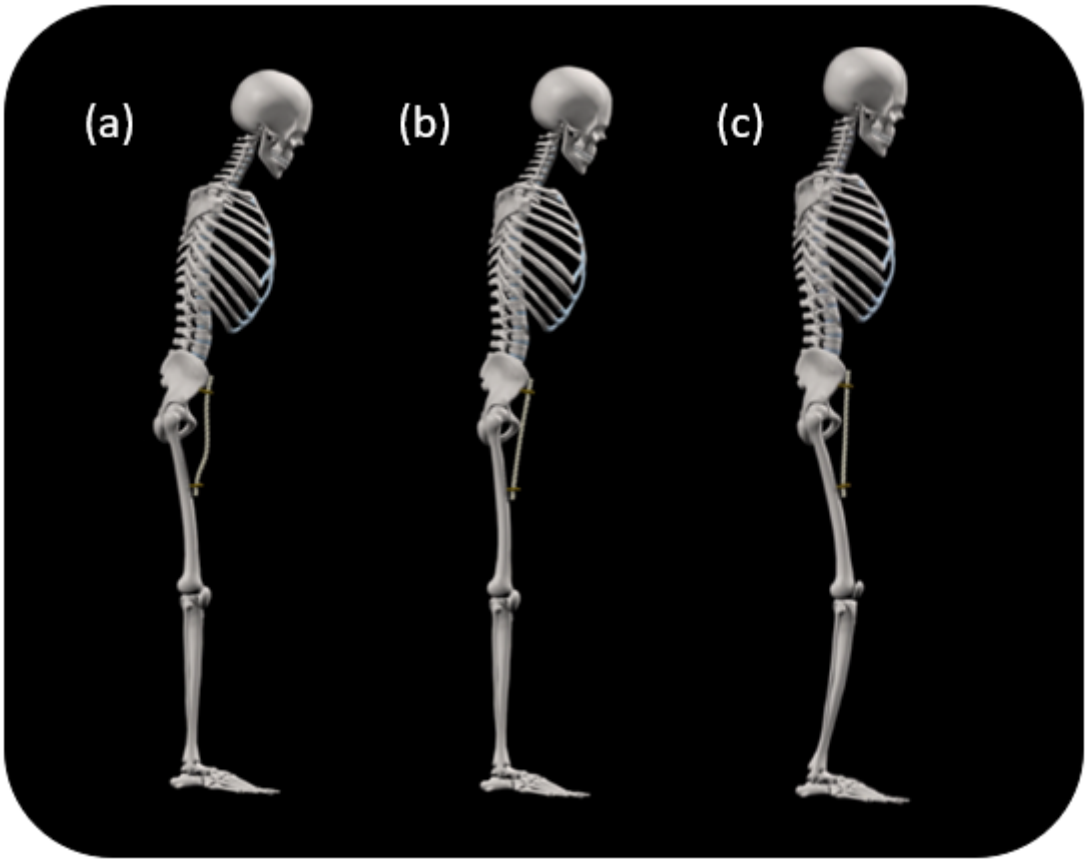

Figure 3

(a, b) A short hip flexor (illustrated as a rope) prevents the pelvis returning to a neutral position in standing. (c) Biomechanical compensation for a short hip flexor, consisting of a flexed hip, knee and ankle and an increased lumbar lordosis. Note there is still a slight flexion of the trunk. A full animation of this pattern can be viewed at: http://hub.salford.ac.uk/tension-release-therapy/biomechanical-compensation-video/

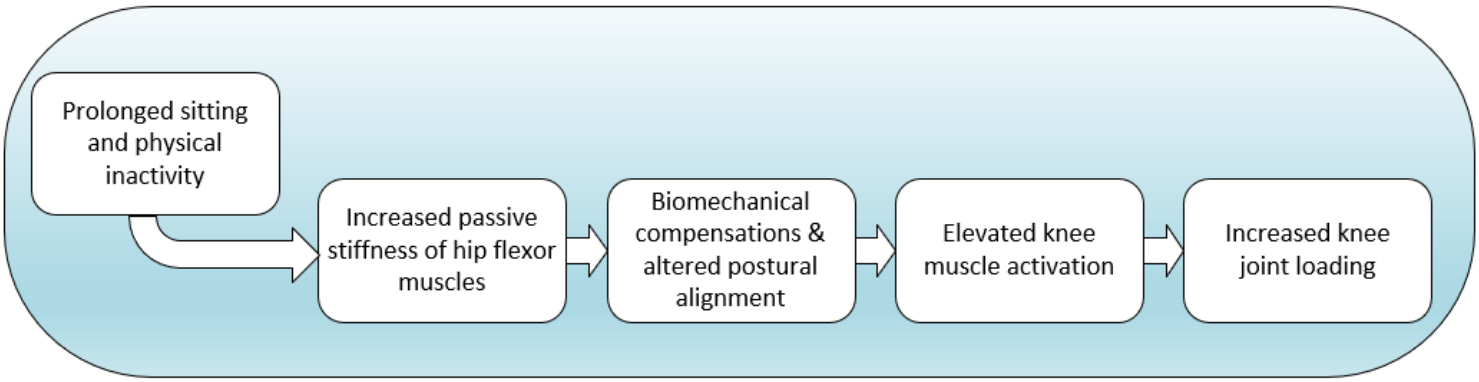

Figure 4

Postural framework to explain elevated knee muscle activation from increased passive stiffness of hip flexor muscles

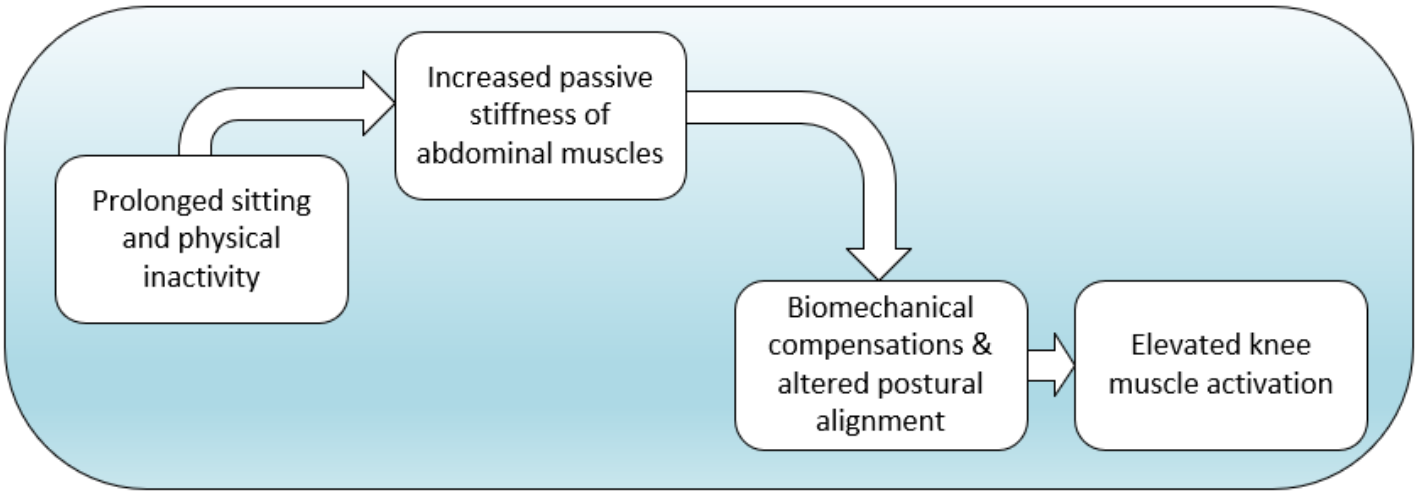

Figure 5

Postural framework to explain elevated knee muscle activation from increased stiffness of abdominal muscles

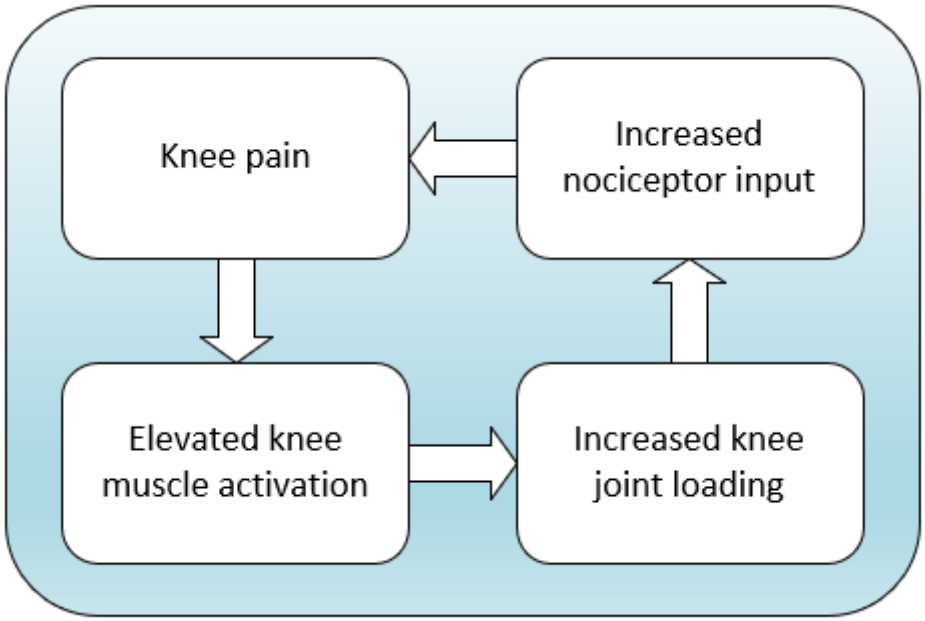

Figure 6

Maladaptive motor response to knee pain in which pain triggers increased knee muscle activity

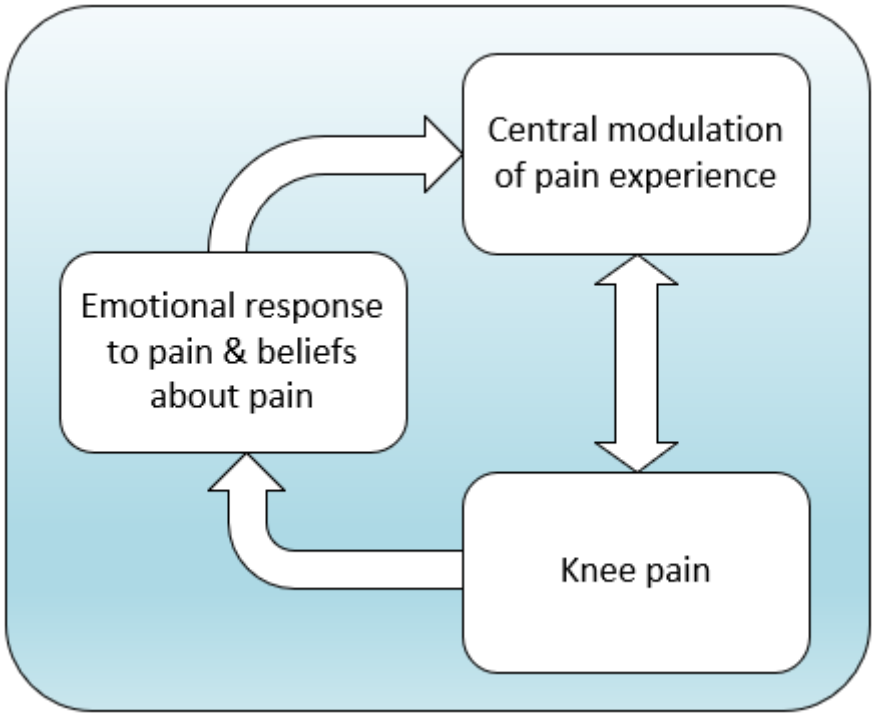

Figure 7

The relationship between knee pain, central sensitisation and the emotional response to pain

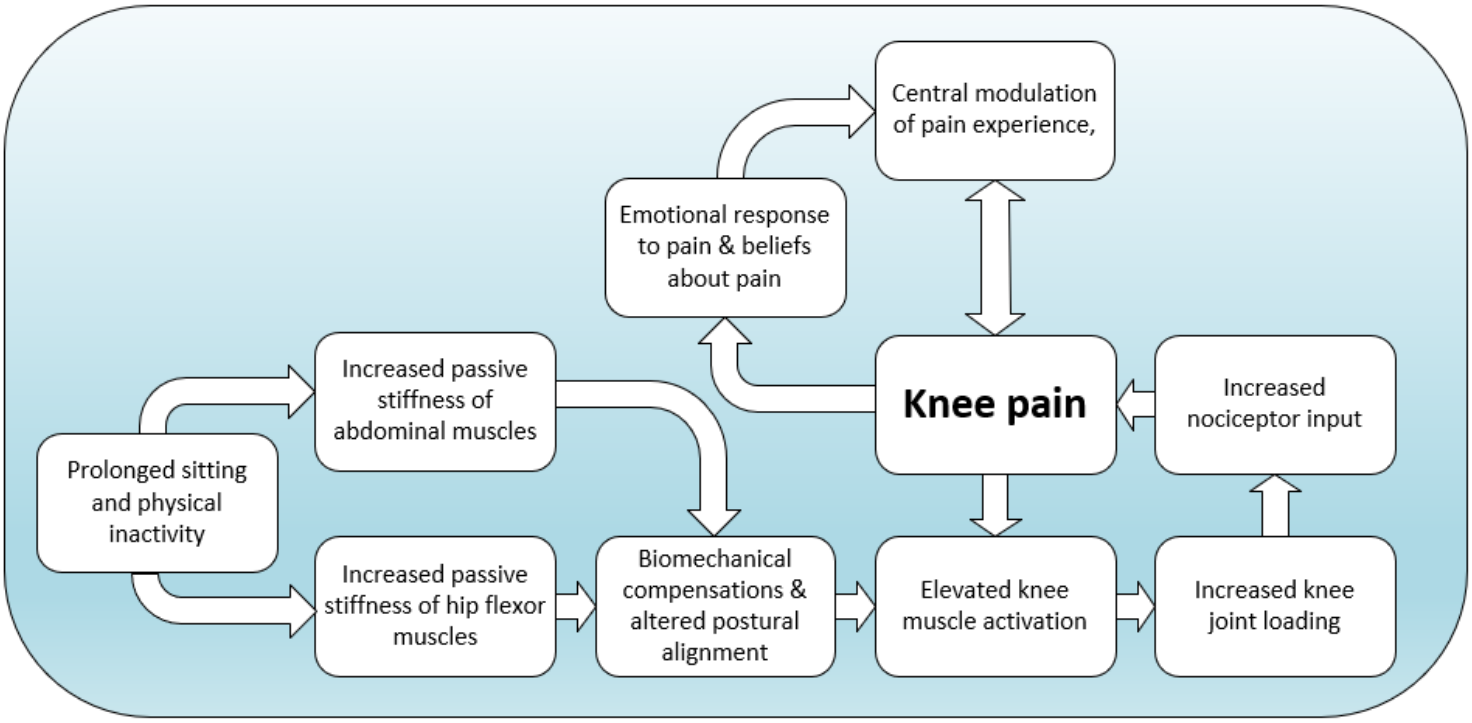

Figure 8

Integrated behavioural framework