	Gain frameMean (SD)(n=75)	Loss frameMean (SD)(n=79)
Physical activity		
Beliefs	6.4 (1.3)	6.6 (0.8)
Intentions	6.6 (0.9)	6.7 (0.9)
Pain medication		
Beliefs	**4.9 (1.9)[1]**	**5.5 (1.6)[1]**
Intentions	4.2 (2.4)	4.4 (2.6)

Note: Mean (SD) beliefs and intentions on a 7-point Likert scale; [1]p<.05.

Table 2 Osteoarthritis and Cartilage

Main effects of framing on beliefs about and intentions to adhere to OA health behaviors

	Exemplification x Gain frameMean (SD)(n=39)	Exemplification x Loss frameMean (SD)(n=37)	Informational x Gain frameMean (SD)(n=39)	Informational x Loss frameMean (SD)(n=37)
Physical activity				
Beliefs	6.4 (1.0)	6.8 (0.5)	6.3 (1.5)	6.5 (1.0)
Intentions	6.5 (1.1)	6.6 (1.0)	6.8 (0.4)	6.7 (0.7)
Pain medication				
Beliefs	**4.5 (1.9)[A]**	**6.0 (1.1)[B]**	**5.3 (1.9)[AB]**	**5.0 (1.7)[A]**
Intentions	4.0 (2.4)	4.9 (2.4)	4.4 (2.5)	3.9 (2.6)

Note: Mean (SD) beliefs and intentions on a 7-point Likert scale; Margins sharing a letter in the group label are not significantly different at the 5% level.

Table 3 Osteoarthritis and Cartilage

Interaction effects of framing and exemplification on beliefs about and intentions to adhere to OA health behaviors

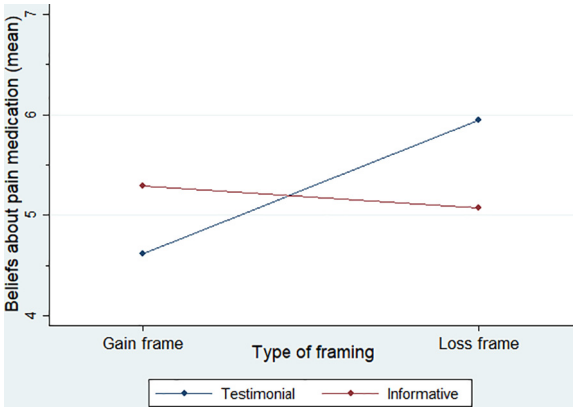

Figure 1 Osteoarthritis and Cartilage

Interaction plot of framing and exemplification on beliefs about pain medication.
Note: Mean of pain medication beliefs on a 7-point Likert scale "If I use pain medication when pain prevents me from being physically active, that is… good/pleasant/wise" (1=completely disagree, 7=completely agree).

9
CAN CONTINUOUS KNEE PAIN OUTCOME MEASURES BE HARMONISED?

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Purpose: Several numerical patient-reported outcome measures (PROMs) have been used for measuring knee pain. Each aims to measure the participant's experience of pain, but may address different pain characteristics. There is increasing need for pooling pain data from studies using different PROMs and implementing such techniques as Individual Patient Data (IPD) analyses. Harmonised data should give the same numerical value for pain severity, irrespective of the host PROM. Our recent meta-analysis of data taken from different patient groups (Georgopoulos et al., OARSI 2021, DOI: https://doi.org/10.1016/j.joca.2021.02.076) indicated that if knee pain PROMs are harmonised by linear transformation to a 0 to 100 scale (100 = worst pain), a score of 30/100 may correspond to the Patient Acceptable Symptom State (PASS). In the current study, we aimed to further explore the validity of a harmonised continuous pain PROM at the individual patient level. **Methods:** Using data from 4 studies of knee OA and knee injury, we conducted an IPD analysis of 3 commonly used knee pain PROMs

(Western Ontario and McMaster Universities Osteoarthritis Index-WOMAC Pain Subscale, Knee injury and Osteoarthritis Outcome Score-KOOS Pain Subscale, Numerical Rating Scales-NRS) in which each participant completed at least 2 of the 3 PROMs permitting paired analysis. Data were from WebEx: a randomised trial exploring the effectiveness of Internet-Based Exercises in individuals with knee OA, KICK and MenTOR: longitudinal studies exploring associations of biomarkers with clinical outcomes in individuals with an acute knee injury and in individuals with a symptomatic degenerative meniscal tear respectively, and NEKO: a cross-sectional study exploring neuromuscular control in individuals with knee OA. PROMs data were standardised to a 0 to 100 scale before analysis (100 = worst pain). WOMAC Pain subscale scores, if not otherwise available, were derived from corresponding items within KOOS. Strength of association (Spearman's rank-order correlation) and agreement between paired PROMs (Concordance correlation co-efficient) were determined. Variance estimates were calculated by linear regression using the whole study population. Bland-Altman plots were developed to visually assess whether there was evidence of heteroscedasticity and establish the limits of agreement (LoA) between pairs of standardised PROM scores.

Results: The 4 cohorts comprised diverse participant populations (Table 1). PROMs were strongly and significantly correlated with each other within each cohort (Table 2), and across the whole study population; NRS and KOOS; n=325 β=0.76 (95%CI: 0.65 to 0.88), SE=0.06, p<0.0001, R^2=0.35 (<0.0001), Intercept: 12.10 (95%CI: 7.01 to 17.19); NRS and WOMAC; n=430, β=0.65 (95%CI: 0.56 to 0.74), SE=0.05, p<0.0001, R^2=0.31 (<0.0001), Intercept: 21.77 (95%CI: 18.20 to 25.33); KOOS and WOMAC; n=325, β=0.86 (95%CI: 0.82 to 0.90), SE=0.02, p<0.0001, R^2=0.85 (<0.0001), Intercept: 13.44 (95%CI: 11.98 to 14.90). Mean differences between standardised PROMs were low, but limits of agreement were wide; NRS:KOOS: Mean difference: 2.49 (95%CI: 0.22 to 4.76), +LoA: 43.26, -LoA: -38.29; NRS:WOMAC: Mean difference: 10.25 (95%CI: 8.24 to 12.26), +LoA: 51.81, -LoA: -31.32 KOOS:WOMAC: Mean difference: 9.03 (95%CI: 8.15 to 9.90), +LoA: 24.78 -LoA: -6.73.

Conclusions: Different PROMs provide related data on knee pain. Although standardised scores from different PROMs give similar mean data (i.e. mean differences that may not be clinically important), we identified substantial heterogeneity within individuals' data. PROMs should ideally be selected according to clinical population and research question being studied, and interpreted carefully with respect to what aspects of their knee pain are important to the patient. Our results

Variables (Value, Range, %)	Descriptives			
	WebEx	KICK	MenTOR	NEKO
	Median (IQ Range)	Median (IQ Range)	Median (IQ Range)	Median (IQ Range)
No. participants	105	139	113	73
Female (n (%))	71 (68%)	25 (18%)	38 (34%)	44 (60%)
Age (y)	68 (60 to 73)	25 (21 to 33)	48 (40 to 53)	63 (55 to 69)
BMI (kg/m^2)	31 (28 to 34)	25 (23 to 28)	29 (23 to 35)	39 (26 to 34)
NRS (0-100)	50 (30 to 60)	30 (20 to 50)	50 (30 to 70)	46 (25 to 69)
KOOS-Pain Subscale (0-100)	-	36 (25 to 47)	42 (25 to 56)	42 (31 to 56)
WOMAC-Pain Subscale (0-100)	40 (25 to 50)	20 (10 to 35)[$]	35 (20 to 50)[$]	35 (25 to 50)[$]

BMI: Body Mass Index, **NRS:** Numerical Rating Scale, **KOOS:** Knee Injury and Osteoarthritis Score (pain subscale), **WOMAC:** Western Ontario and McMaster Universities Arthritis Index (pain subscale).
Data are n (%) or median (interquartile range)
[₸] KOOS was lineary transformed to a 0 to 100 scale where 100 represents worst pain.
[$] WOMAC: Scores here were a pseudoWOMAC pain scale, claculated, when otherwise available, from the Questions 5 to 9 in the KOOS pain subscale.

Table 1

Osteoarthritis and Cartilage

Participant demographics, descriptives, differences between PROMs pairs within all cohorts

Variables (PROMs combinations and test types)	Associations			
	WebEx	KICK	MenTOR	NEKO
NRS ~ KOOS (Pain subscale) association				
Spearman's coeffcicient (p)	-	0.54 (<0.0001)[†]	0.65 (<0.0001)[†]	0.64 (<0.001)[†]
CCC (p)	-	0.80 (<0.001)[†]	0.81 (<0.0001)[†]	0.80 (<0.001)[†]
NRS ~ WOMAC (Pain subscale) association				
Spearman's coeffcicient (p)	0.41 (<0.0001)	0.55 (<0.0001)[$]	0.60 (<0.0001)[$]	0.65 (<0.001)[$]
CCC (p)	0.74 (<0.001)	0.76 (<0.001)[$]	0.77 (<0.0001)[$]	0.79 (<0.001)[$]
KOOS (Pain subscale) ~ WOMAC (Pain subscale) association				
Spearman's coeffcicient (p)	-	0.89 (<0.0001)[†,$]	0.95 (<0.0001)[†,$]	0.92 (<0.001)[†,$]
CCC (p)	-	0.80 (<0.001)[†,$]	0.90 (<0.0001)[†,$]	0.88 (<0.001)[†,$]

CCC: Concordance Correlation Coefficient, **NRS:** Numerical Rating Scale, **KOOS:** Knee Injury and Osteoarthritis Score (pain subscale), **PROMs:** Patient Reported Outcome Measures, **WOMAC:** Western Ontario and McMaster Universities Arthritis Index (pain subscale).
[†] KOOS was lineary transformed to a 0 to 100 scale where 100 represents worst pain.
[$] WOMAC: Scores here were a pseudoWOMAC pain scale, claculated, when otherwise available, from the Questions 5 to 9 in the KOOS pain subscale.

Table 2

Osteoarthritis and Cartilage

Associations between PROMs within all cohorts

further suggest exercising caution in attempting to harmonise differing longitudinal pain PROM data within or between participants and studies. It may be that a dichotomisation at harmonised PASS threshold on a PROM, as proposed in our systematic literature review, may have greater tolerance to allow comparisons between different pain PROMs datasets, which will be tested in corresponding research.

10
DEVELOPMENT OF AN INTERVENTION TO IMPROVE IDENTIFICATION AND MANAGEMENT OF CONCOMITANT KNEE OSTEOARTHRITIS IN PERSONS WITH TYPE 2 DIABETES AND PHYSICAL INACTIVITY

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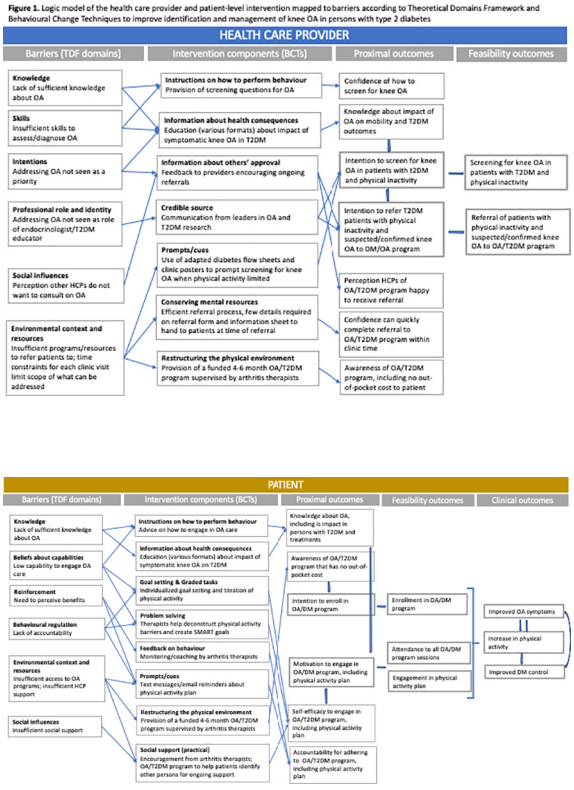
Purpose: Guideline recommendations exist for the management of knee osteoarthritis (OA) yet uptake of evidence-based OA care is suboptimal. In persons with type 2 diabetes (T2DM), the presence of concomitant symptomatic knee OA leads to functional impairments and physical inactivity which increases the risk for diabetes complications and cardiovascular events. Yet, these individuals are less likely to receive OA care due to the competing clinical demands of their T2DM. Improving uptake of knee OA care in persons with T2DM represents an opportunity to not only improve OA outcomes for under-treated individuals but also improve T2DM outcomes for a population at high risk of cardiometabolic sequelae. Implementation interventions seek to put evidence-based care into practice. The use of theory in developing complex interventions focuses on identifying determinants of behaviour and linking them to mechanisms of change to maximize intervention effectiveness. Involving stakeholders in a user-centered co-creation process can increase impact and sustainability. Our aim was to use a systematic process combining theory, stakeholder involvement and existing evidence for knee OA care to develop a multifaceted implementation intervention targeting both health care providers (HCPs) who treat T2DM and patients with T2DM and knee OA, to improve the uptake of evidence-based OA care in persons with T2DM and knee OA who are physically inactive. A broader aim was to outline this process of systematically developing a complex intervention that seeks to change behaviours of HCPs and patients to provide template for researchers tackling other problems.
Methods: The TOP-DM (Treating Osteoarthritis in Persons with Diabetes Mellitus) intervention development followed a systematic stepped process informed by the UK Medical Research Council (MRC) guidance for developing complex interventions informed by theory. First, we identified target behaviours through consultation of a User Advisory Panel (UAP) composed of stakeholders from family medicine, endocrinology, patients, physical therapy, rheumatology, and Arthritis Society, a Canadian non-profit organization offering an existing arthritis rehabilitation program. Second, we interviewed HCPs who treat T2DM (family physicians, endocrinologists, diabetes educators; n=18) and patients with T2DM and knee OA (n=18) to identify determinants of: 1) Assessing and treating knee OA (HCPs); and 2) Seeing and engaging in OA care (patients) using the Theoretical Domains Framework (TDF). We mapped identified TDF domains to operationalizable literature-supported behavioural change techniques (BCTs) to determine potential intervention components. Third, we conducted semi-structured stakeholder meetings with members of our UAP, individually and in groups, to ascertain acceptability and feasibility of identified BCTs, including content and modes of delivery. Fourth, the research team selected final intervention components informed by the prior steps, and constructed a logic model.
Results: Together with our UAP, we identified modifiable target behaviours at the HCP and patient level. At the HCP level, barriers to assessing and treating knee OA were: lack of sufficient knowledge and skills to assess OA, no intention to address OA, OA care not seen within the professional role and identity for some providers (endocrinologist/diabetes educator), perceiving that other HCP may not want to treat OA, and paucity of government-funded OA resources and programs to refer patients to for treatment. At the patient level, barriers to seeking and engaging in OA care were: insufficient knowledge and belief in personal capability to engage in OA care, need for reinforcement, social support and behavioral regulation, including accountability, insufficient support from HCPs, and paucity of accessible programs and resources for OA care. These barriers were mapped to BCTs and found to be acceptable by interviewed stakeholders; we modified content/delivery in response to stakeholder feedback. The final intervention components incorporated a range of BCTs at the HCP and patient level. These include, at the HCP level: provision of screening questions to help in the assessment of knee OA, education about the impact on symptomatic OA in persons with T2DM, cues in the work environment to prompt OA screening in persons with physical inactivity, development of an OA/T2DM program for persons with T2DM in conjunction with Arthritis Society, and an efficient referral process to the OA/T2DM program; and at the patient level: education about OA (multiple modes of delivery), the Arthritis Society-delivered OA/T2DM program that comprises goal setting, problem solving, feedback on behaviour, and support to enable engagement in the program including individualized physical activity plan. We present a logic model of the proposed intervention in Figure 1.
Conclusions: We integrated theory and stakeholder involvement to develop a multifaceted intervention to increase identification of knee OA in persons with T2DM and physical inactivity within diabetes care and improve uptake and engagement in evidence-based OA management. The final intervention leverages the existing government-funded Arthritis Society arthritis rehabilitation program infrastructure that supports potential spread, scalability and sustainability. The feasibility of the intervention components will be assessed and refined using a rapid-cycle improvement strategy before further evaluation in a cluster randomized pilot trial.



11
IDENTIFICATION AND FUNCTIONAL CHARACTERIZATION IMBALANCED OSTEOARTHRITIS ASSOCIATED FIBRONECTIN SPLICE VARIANTS

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Purpose: Transcriptome-wide analyses demonstrated *FN1* is the highest expressed gene in articular cartilage and is significantly upregulated with ongoing OA pathophysiology. *FN1* encodes fibronectin, a dimeric glycoprotein, particularly localized in the pericellular matrix directly surrounding the chondrocytes. Fibronectin mediates a wide variety of cellular interactions by binding to extra cellular matrix proteins and to chondrocytes via integrins. The main integrin-binding domain of fibronectin is the RGD motif, which binds multiple integrin heterodimers, including the classic fibronectin receptor integrin $\alpha5\beta1$. *FN1* can give rise to 27 different splice variants, which can occur at three major sites, called extra domain A (A), extra domain B (B), and variable region