

The Consequences of Youth Sport-Related Knee Injury Implications for Secondary Prevention of Osteoarthritis

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The Growing Burden of Osteoarthritis

Significance

OA is expected to become the 4th leading cause of disability worldwide by 2020



Increase in prevalence from 12.5% (2010) to 25% (2040)



Increase in prevalence from 26.6% (2012) to 29.5% (2032)

Increase in prevalence from 12% (2008) to 25 (2030)



Woolf et al 2003, Lawrence et al 2008, Vos et al 2012, Sharif et al 2016

Unsustainable Trends

Significance

OA is the fastest growing disease globally based on Years Lived with Disability



Increase in direct costs from \$2.9 billion (2010) to \$5.8 billion (2040)





54% increase in direct medical costs between 1993 and 2002

37% and 48% increase in hip and knee replacements between 2015 and 2020



Levy et al 1993, Woolf et al 2003, Pen et al 2005, Vos et al 2012, Sharif et al 2016, Hunter & Bowden 2017

Prevention of Osteoarthritis

Epidemiological Model

UPSTREAM SHIFT IN OUR APPROACH TO MANAGEMENT

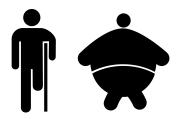
Primary Prevention

Secondary Prevention

Tertiary Prevention

SUSCEPTIBILE

Prevent or reduce risk factors in susceptible populations



PRE-SYMPTOMATIC

Identify and slow down the onset of OA in preclinical populations



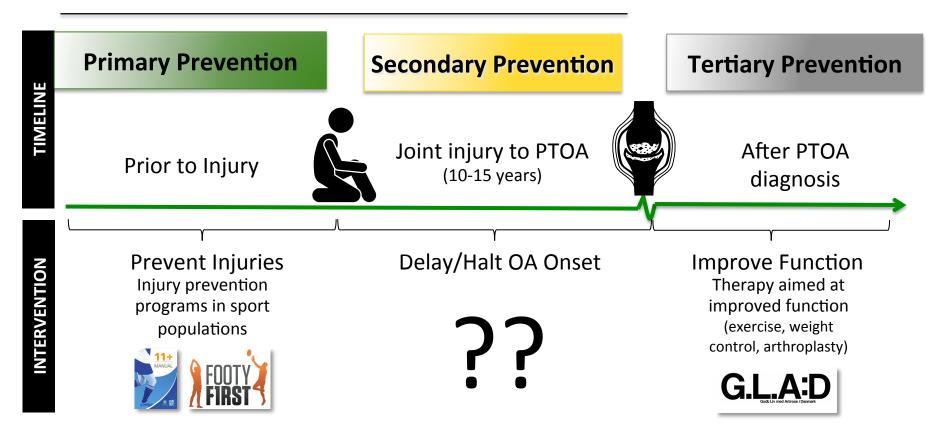
DISEASE

Slow progression in those with OA (improve function)



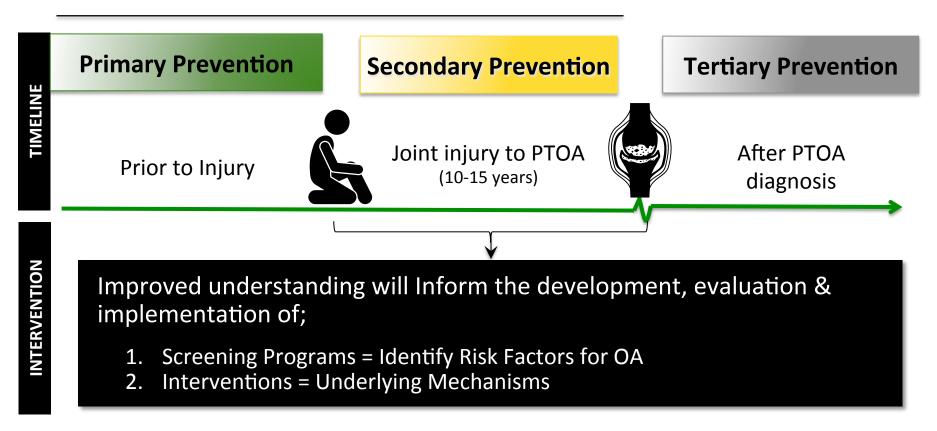
Prevention of Post-Traumatic Osteoarthritis

Timeline of Events & Interventions



Prevention of Post-Traumatic Osteoarthritis

Timeline of Events & Interventions



Alberta Youth PrE-OA Study

Ongoing Longitudinal Cohort Study



Secondary Prevention – Knee PTOA



0-3 yrs post-injury

3-10 yrs post-injury

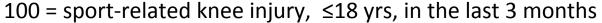




100 = sport-related knee injury, ≤18 yrs, 3-10 years earlier

100 = age, sex and sport matched controls





100 = age, sex, and sport matched controls



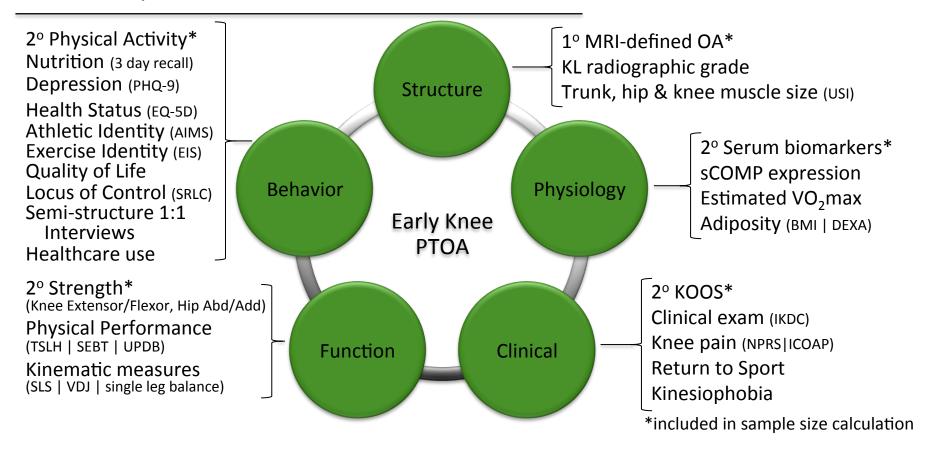
Followed annually on a diverse set of outcomes for 3 yrs.





Outcomes

PrE-OA Study



Participant Characteristics

Baseline Follow-up 1 3-10 years

PrE-OA Study

| Characteristics | Uninjured n=100 | Injured n=100 |
|--|--------------------|------------------|
| Sex (% female) | 55 | 55 |
| Age (yrs; median, range) | 22 (15-26) | 22 (16-26) |
| Age at Injury (yrs; median, range) | - | 16 (9-18) |
| Injury to Follow-up 1 (yrs; median, range) | - | 6.9 (3-10) |
| # Index Knee Surgeries | 0 | 63* |
| # Contralateral Knee Injuries | 0 | 23 [§] |
| # Contralateral Knee Surgeries | 0 | 15^{ϕ} |
| # Index Lower Limb Injuries | 17 | 13 |
| # Contralateral Lower Limb Injuries | 13 | 16 |

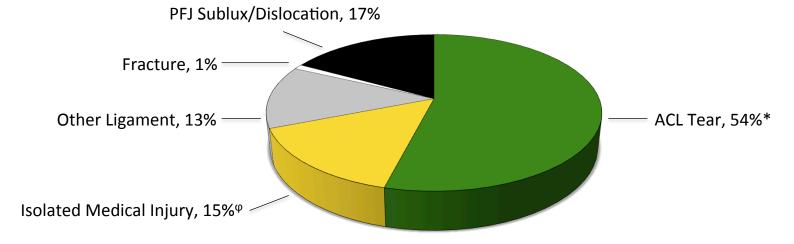
^{*54} ACL reconstructions, § φ11 of these were ACL reconstructions

Injury Definition

PrE-OA Study

Baseline Follow-up 1 3-10 years

KNEE INJURY Ligament, meniscal or other intra-articular tibio or patello-femoral injury requiring both MEDICAL CONSULTATION & DISRUPTED SPORT PARTICIPATION

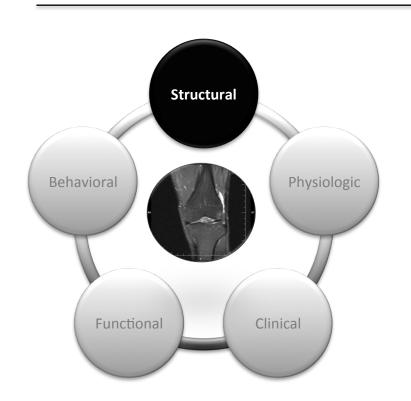


*all under went ACL reconstruction, 36 had concomitant meniscal injuries $^{\circ}7$ of these had arthroscopic surgery

Whittaker et al 2015, 2107

MRI-Defined OA

Surrogate Structural Outcome of Early PTOA



Hunter et al 2011

MRI Defined OA

Bilateral clinical series (1.5 Tesla): axial, coronal & sagittal proton density, proton density fat saturation

MOAKS rating by radiologist blinded to injury

MRI defined OA Criteria:

Osteophyte AND full-thickness cartilage loss OR

1 of the above plus 2 of the following; Sub-chondral bone marrow lesion Meniscal disruption Partial thickness cartilage loss

MRI-Defined OA

Surrogate Structural Outcome of Early PTOA



Structural MRI

Clinical

Functional

Physiological

Behavioural Beliefs

Whittaker et al 2017

Structural changes consistent with OA are not unique to ACL tears or damaged menisci

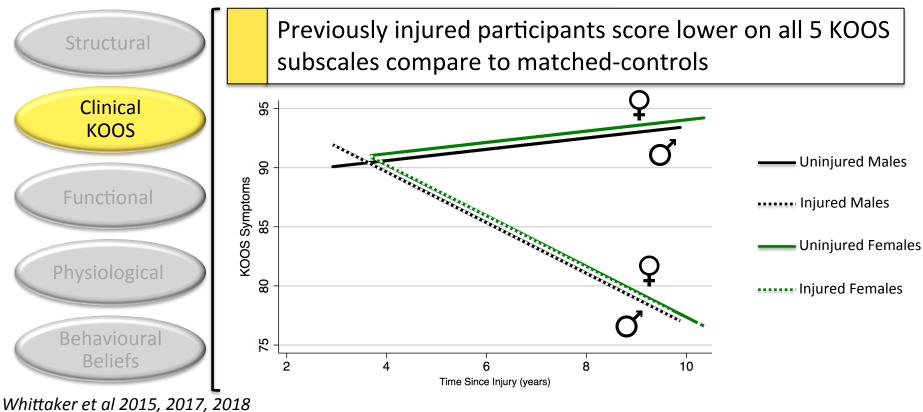
The odds of MRI defined OA 3-10 years after knee injury vary by injury history, injury type and surgery

| Surgery / Injury Type | Odds of MRI-defined OA (95%CI) | |
|-----------------------------|--------------------------------|--|
| Knee Injury | 10.0 (2.3,42.8)* | |
| Grade I-III MCL or LCL | 2.0 (0.18,22.1) | |
| 3° ACL ^φ | 11.5 (1.4,85.2)* | |
| Knee Surgery | 13.5 (1.7,99.4)* | |
| 3° ACL &/or meniscal injury | 14.5 (1.8,106.5)* | |

Clinical Symptomology (KOOS)

Knee Injury and OA Outcome Score – Modifiable Risk Factor

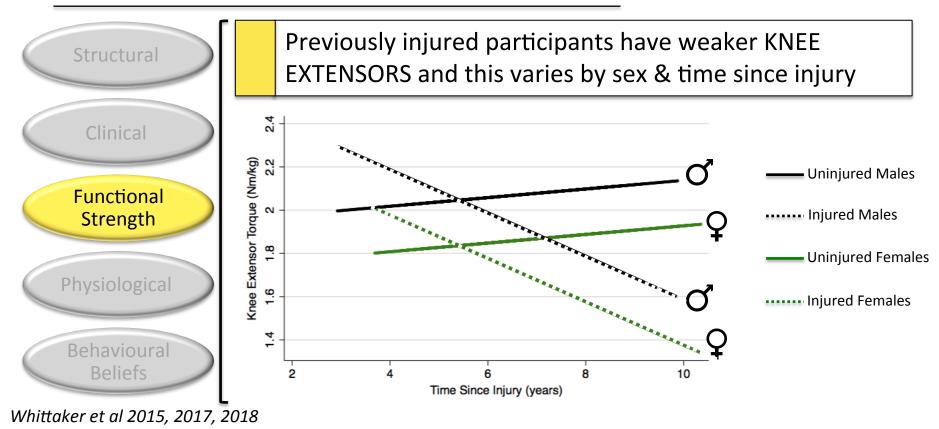




Knee Extensor Strength

Dynamometer – Modifiable Risk Factor

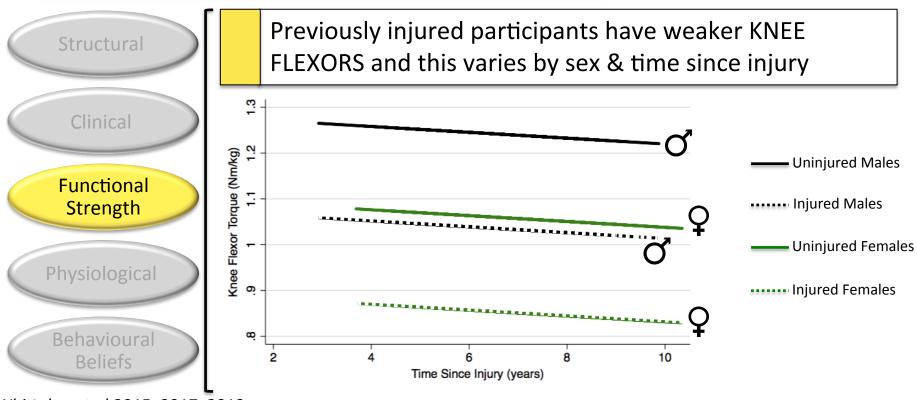




Knee Flexor Strength

Dynamometer – Modifiable Risk Factor



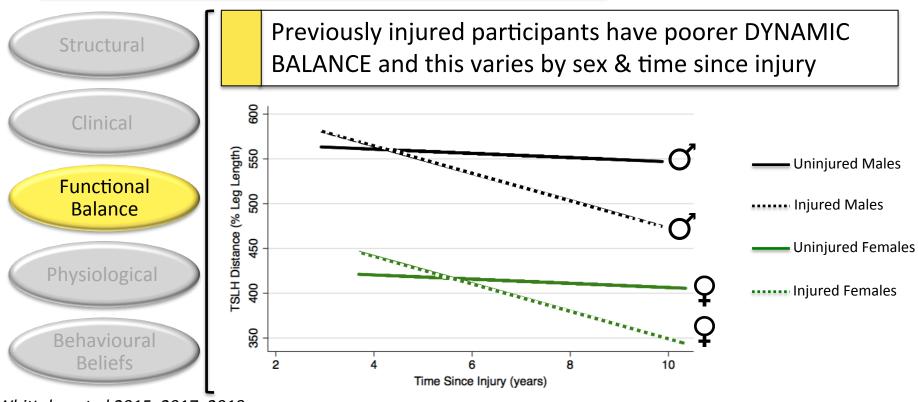


Whittaker et al 2015, 2017, 2018

Dynamic Balance

TSLH – Modifiable Risk Factor



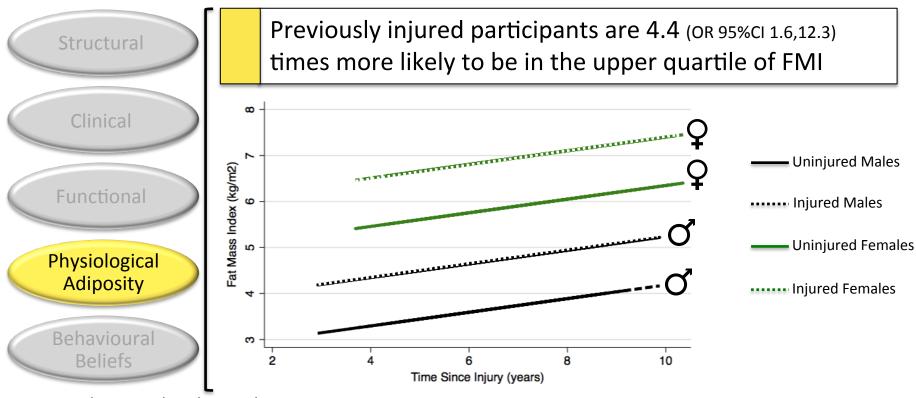


Whittaker et al 2015, 2017, 2018

Adiposity

Dual X-ray Absorptiometry- Modifiable Risk Factor



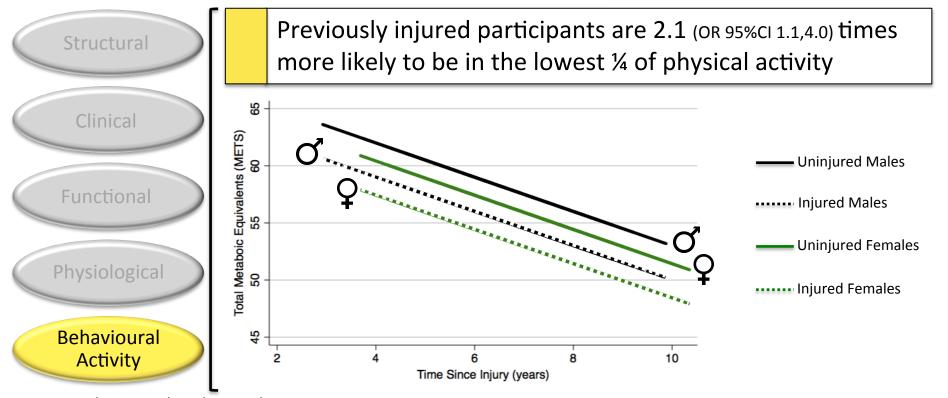


Toomey et al 2017, Whittaker et al 2015, 2017, 2018

Physical Activity (self-reported)

Godin Leisure Time Questionnaire - Modifiable Risk Factor



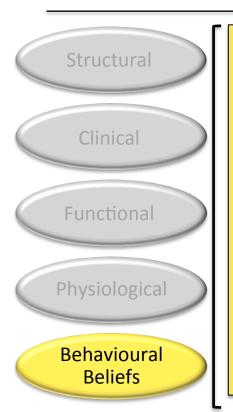


Toomey et al 2017, Whittaker et al 2015, 2017, 2018

Beliefs re: Sport, Injury and OA

Behavioral; Semi-structured Interviews (n=19, purposive sample)





Dominant themes:

Acceptance

Varying & often unrealistic degrees of acceptance about the impact of injury on sporting ability & future PTOA

Resiliency & Determination

Highly motivated to recover & meet injury with resiliency – do not pace well

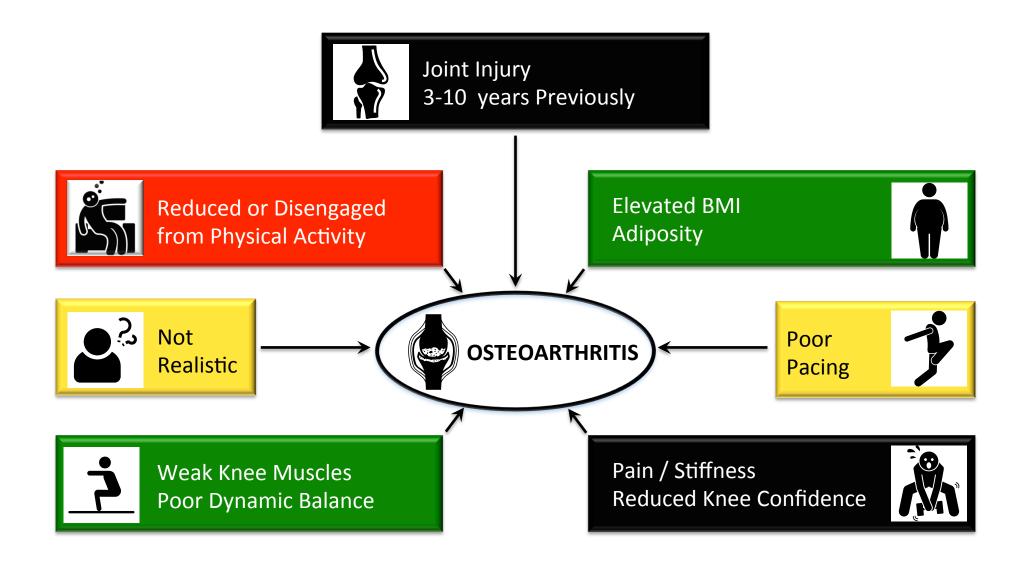
Knee Confidence

Knee requires ongoing protection

Evolving Athletic Identity

Influenced by injury & shifting life priorities

Ezzat et al 2018







ACL Tear ± Meniscus
Osteochondral Lesion

Re-injury No RTS Criteria





Lacking Info.

Elevated BMI, Adiposity





Reduced or Disengaged from Physical Activity

Poor Pacing





Frequent Flare-ups

Insufficient Rehabilitation
Pain / Stiffness
Reduce Knee Confidence





Weak Knee Muscles Poor Dynamic Balance Vitamin D, K, Deficient



Implications

Future Directions



Pre-existing Risk Factors

Do injured youth have ↑adiposity, ↓strength or participate in less physical activity at the time of injury

Mechanisms

What is contributing to changes in ↑adiposity and ↓strength? (Diet, activity, psychosocial factors, inaccurate beliefs, etc.)

Sub-groups

Are there different phenotypes / sub-groups of at risk individuals (i.e., OA phenotypes.)

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