Pain and joint dysfunction:
How are they related?

Paul W. Hodges DSc MedDr PhD BPhty(Hons) APAM(Hon) FACP FQA FAHMS FAA
NHMRC Leadership Fellow (Level 3) & Professorial Fellow
Director, NHMRC CCRE Spinal Pain, Injury & Health
The University of Queensland

ccre spine centre of clinical research excellence
Spinal Pain, Injury & Health NHMRC funded centre
MyBackPain.org.au
The University Of Queensland AUSTRALIA

1

How is pain related to joint dysfunction?

What is pain?

What mechanisms explain the relationship between pain, movement and joint dysfunction?

What are the clinical implications?

2

How is pain related to joint dysfunction?

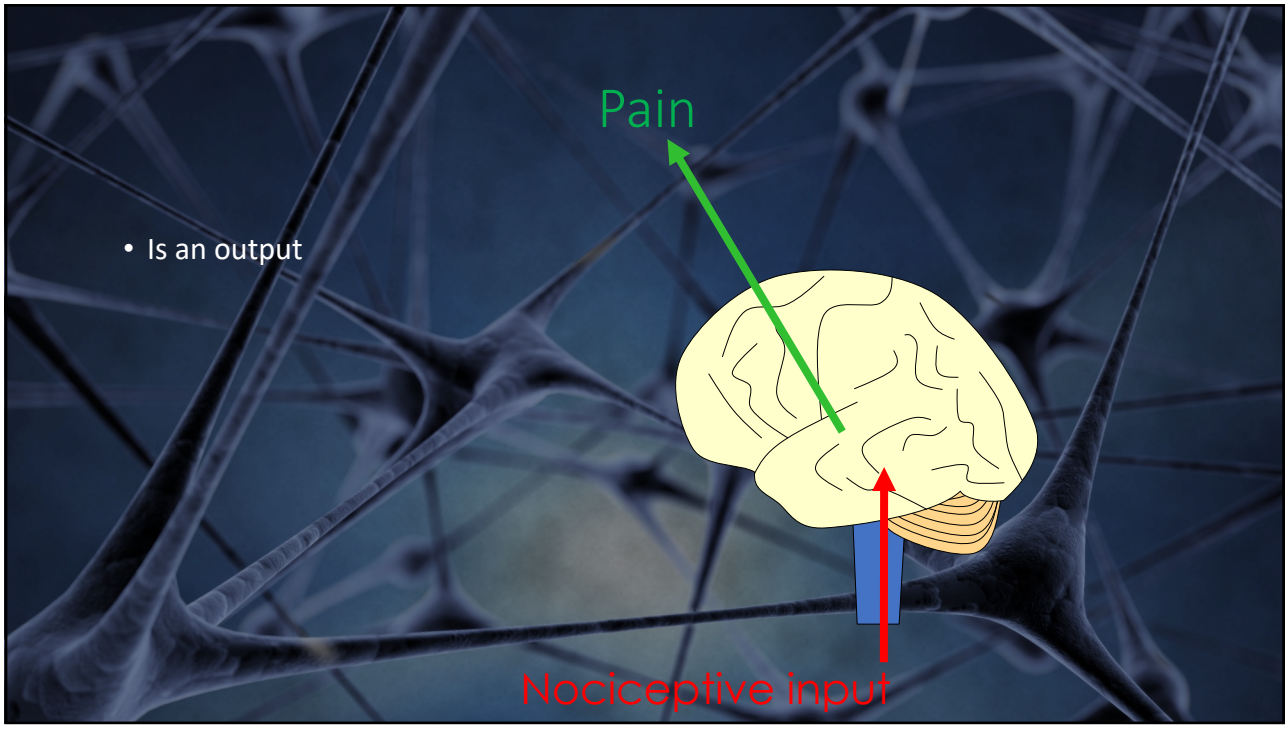
What is pain?

What mechanisms explain the relationship between pain, movement and joint dysfunction?

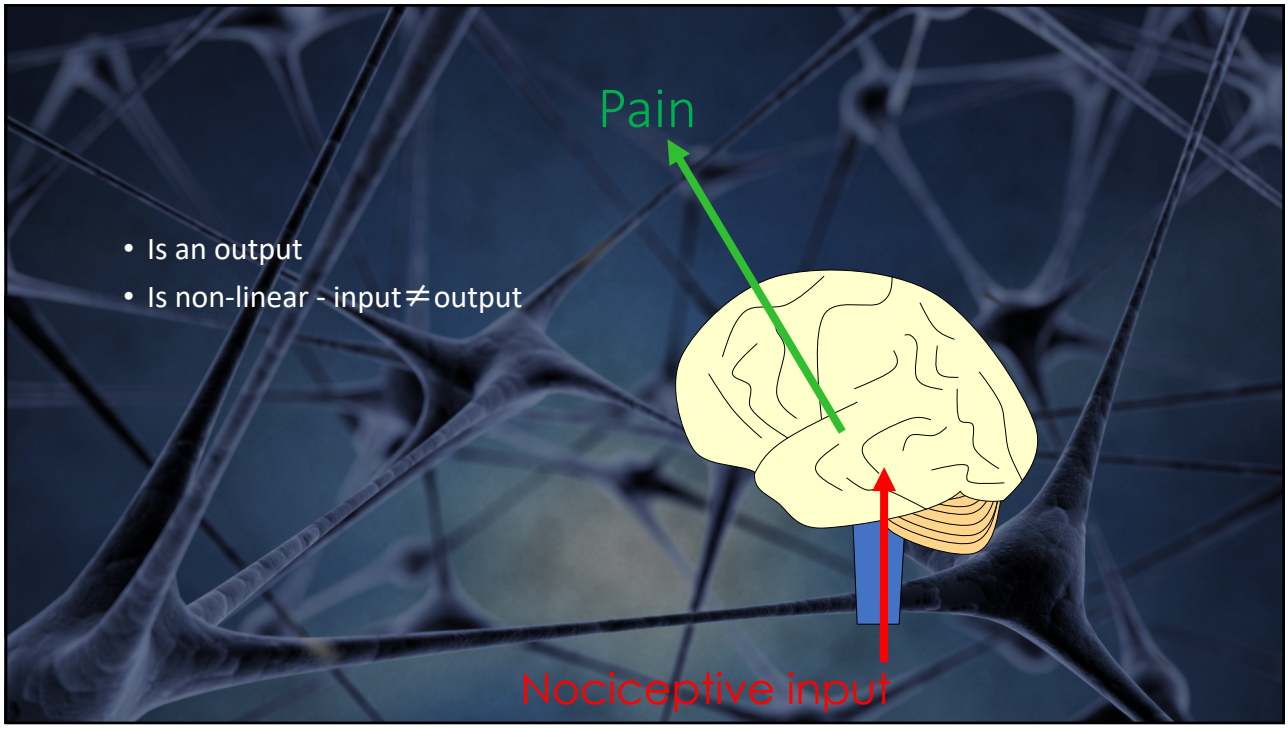
What are the clinical implications?

3

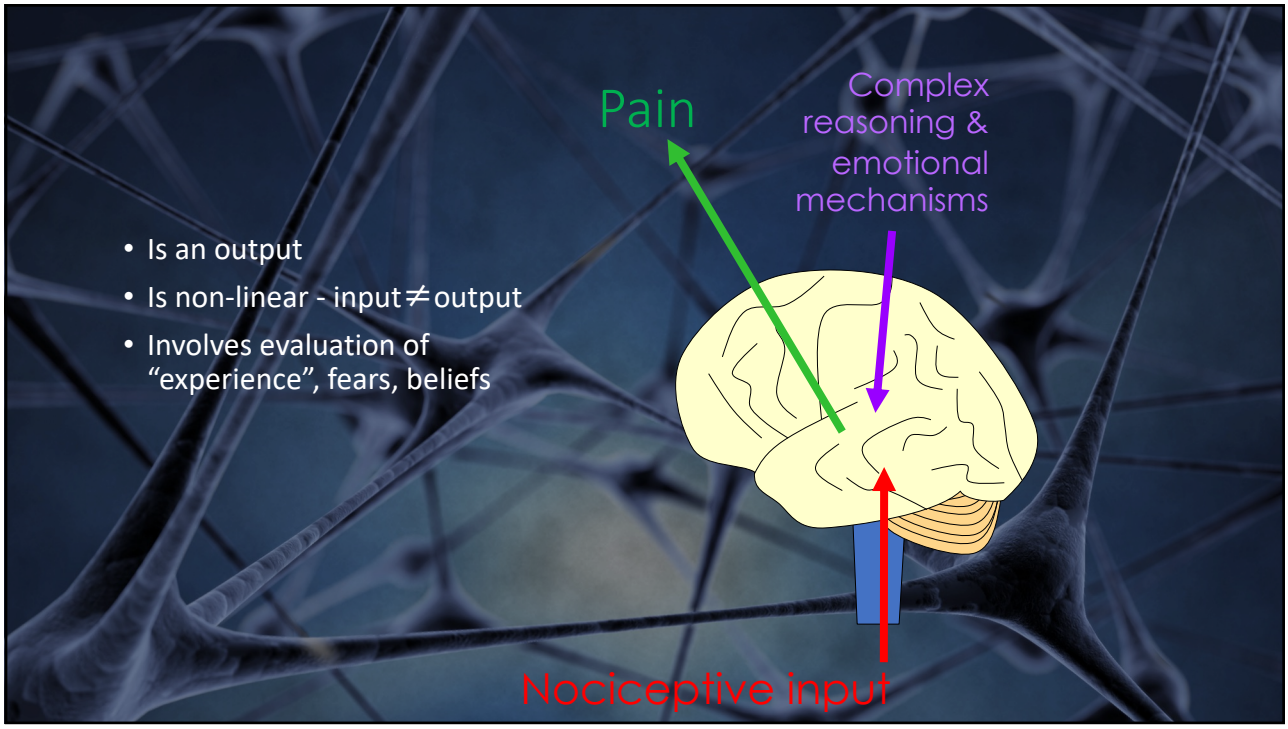
- Is an output



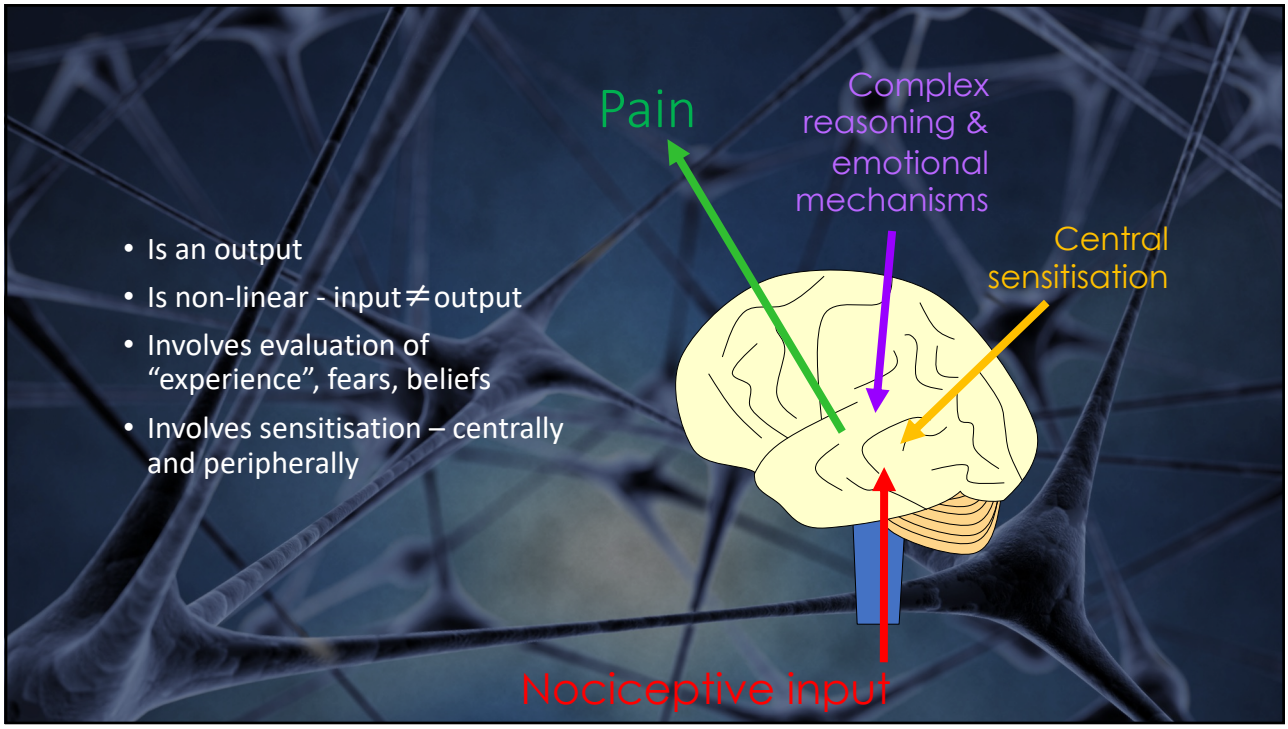
4


Pain
• Is an output
• Is non-linear - input ≠ output
Nociceptive input

5

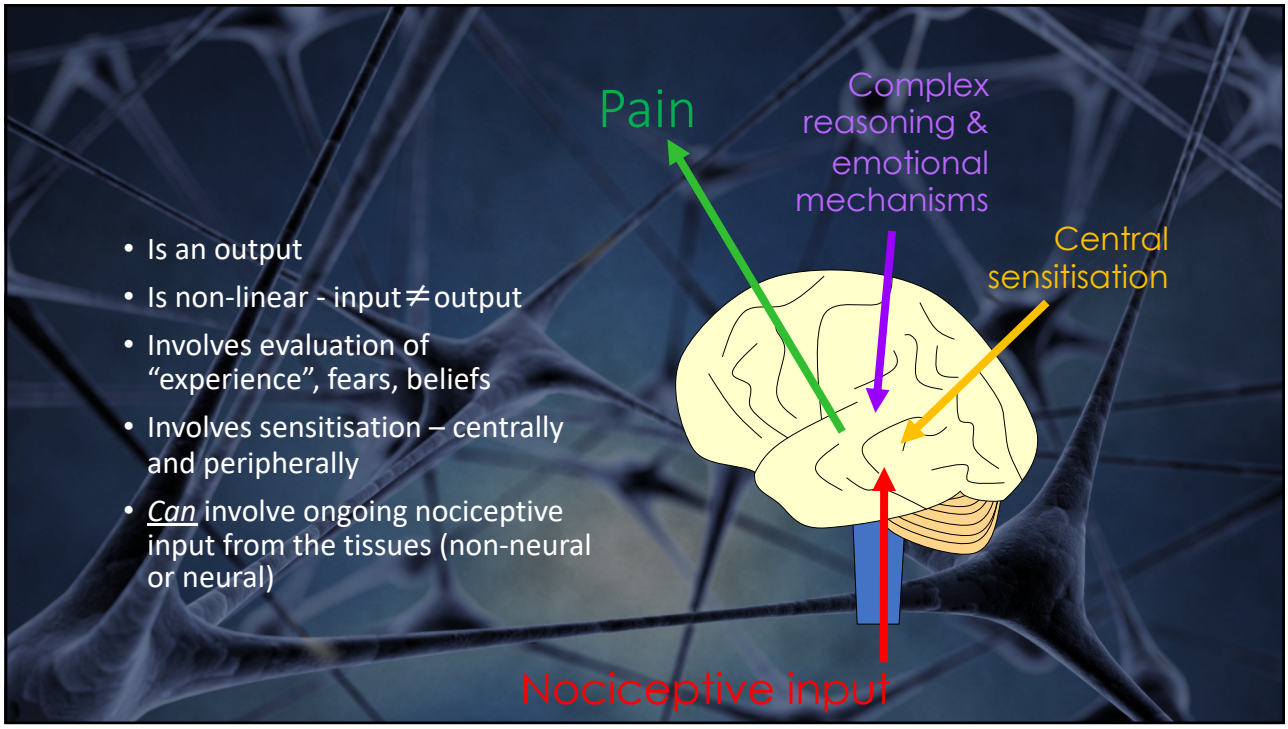

Pain
Complex reasoning & emotional mechanisms
• Is an output
• Is non-linear - input ≠ output
• Involves evaluation of "experience", fears, beliefs
Nociceptive input

6



- Is an output
- Is non-linear - input ≠ output
- Involves evaluation of "experience", fears, beliefs
- Involves sensitisation – centrally and peripherally

7



- Is an output
- Is non-linear - input ≠ output
- Involves evaluation of "experience", fears, beliefs
- Involves sensitisation – centrally and peripherally
- *Can* involve ongoing nociceptive input from the tissues (non-neural or neural)

8

Pain neurobiology: Not all pain is the same

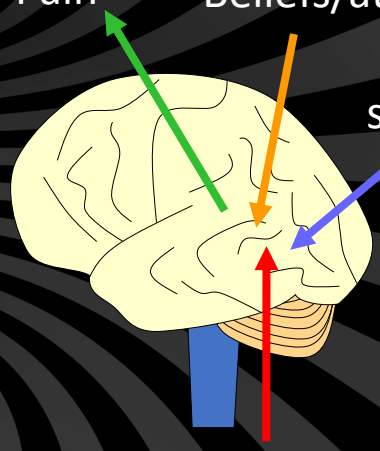
Pain maintained by;

Nociceptive mechanisms

Nociplastic mechanisms

Neuropathic mechanisms

IASP 2020



9

Nociceptive pain (IASP definition)

- Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors.

10

Neuropathic pain (IASP definition)

- Pain caused by a lesion or disease of the somatosensory nervous system.

11

Nociplastic pain (IASP definition)

- Pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain.

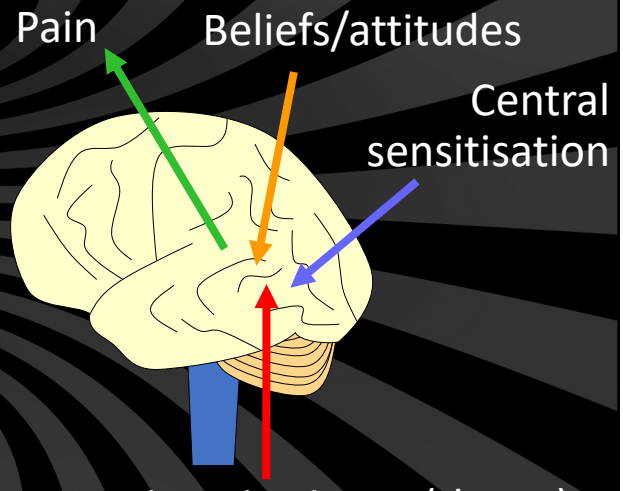
12

Pain neurobiology: Not all pain is the same

Pain maintained by

Pain will rarely present neatly in a single box – the aim is to consider the predominant mechanism to guide selection of treatment

IASP 2020



13

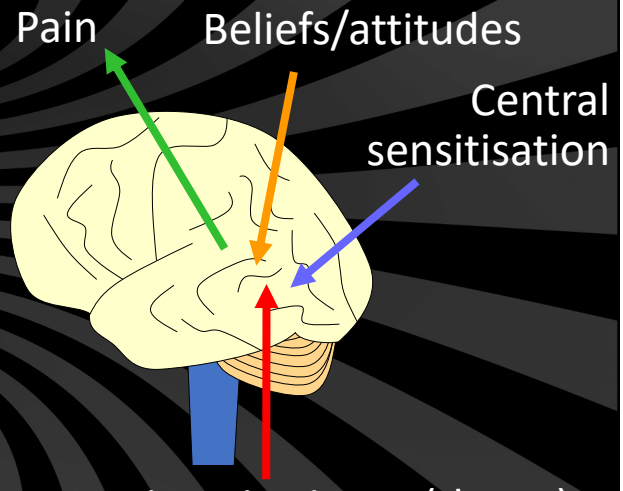
Pain neurobiology: Not all pain is the same

Nociceptive/movement-related
- Arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors.

Nociplastic/central/central sensitization
- Arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain

Neuropathic
- Pain caused by a lesion or disease of the somatosensory nervous system

IASP 2020



14

Pain neurobiology: Not all pain is the same

Nociceptive/movement-related

- Arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors

Modify tissue loading

Nociplastic/central/central sensitization

- Arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain

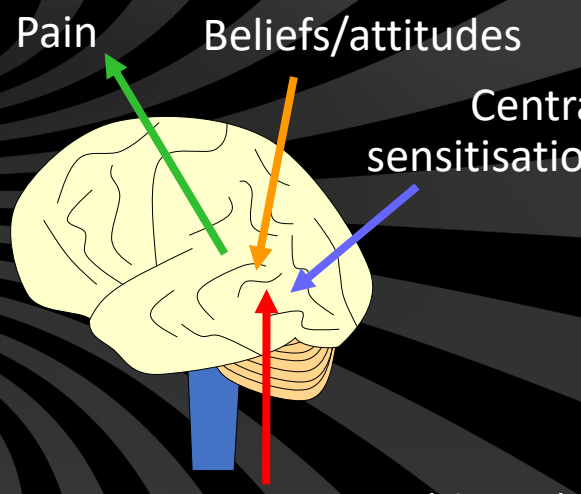
Reduce amplification - psychological, education, etc

Neuropathic

- Pain caused by a lesion or disease of the somatosensory nervous system

Modify nerve loading + reduce amplification

IASP 2020



15

Is joint loading relevant?

If load on tissues causes nociceptor discharge which contributes to the pain experience

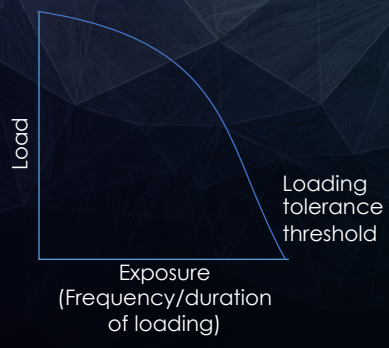
Then tissue loading might be relevant and it may be helpful to change it to reduce the contribution of nociceptive input to ongoing pain

16

When is joint loading a problem

17

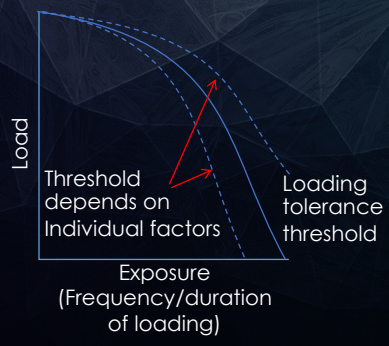
Relationship between joint loading and dysfunction



Dye (1996) *Clin Orthop Rel Res*

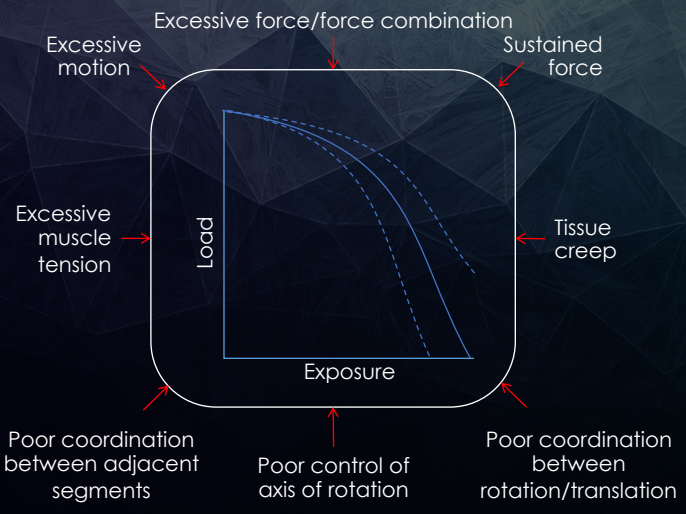
18

Relationship between joint loading and dysfunction

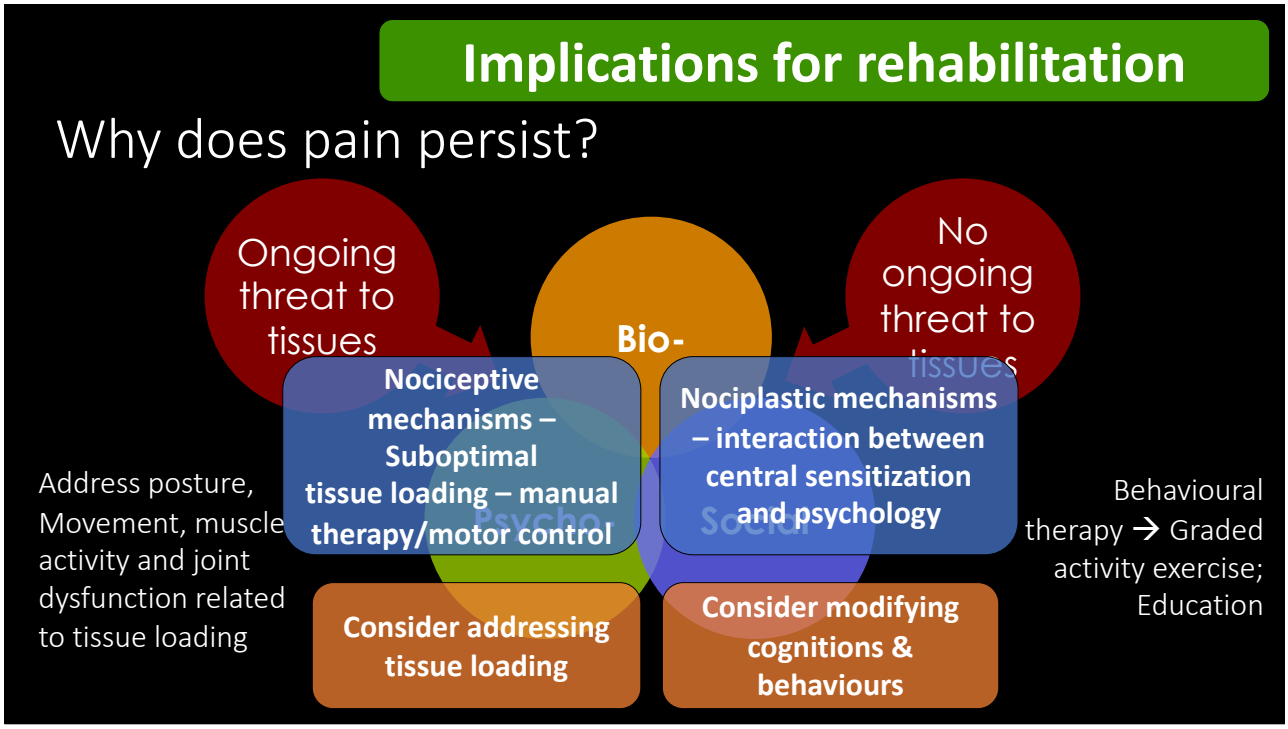


19

Causes of suboptimal tissue/joint loading



20



21

Research Paper

PAIN®

Features and methods to discriminate between mechanism-based categories of pain experienced in the musculoskeletal system: a Delphi expert consensus study

Muath A. Shraim[a], Kathleen A. Sluka[b], Michele Sterling[c], Lars Arendt-Nielsen[d], Charles Argoff[e], Karl S. Bagraith[f], Ralf Baron[g], Helena Brisby[h], Daniel B. Carr[i], Ruth L. Chimenti[j], Carol A. Courtney[k], Michele Curatolo[l], Beth D. Darnall[m], Jon J. Ford[n], Thomas Graven-Nielsen[o], Melissa C. Kolski[p], Eva Kosek[q,r], Richard E. Liebano[s], Shannon L. Merkle[t], Romy Parker[u], Felipe J. J. Reis[v,w], Keith Smart[x], Rob J. E. M. Smeets[y,z], Peter Svensson[aa], Bronwyn L. Thompson[ab], Rolf-Detlef Treede[ac], Takahiro Ushida[ad], Owen D. Williamson[ae], Paul W. Hodges[a,*]

More later…

22

How is pain related to joint dysfunction?

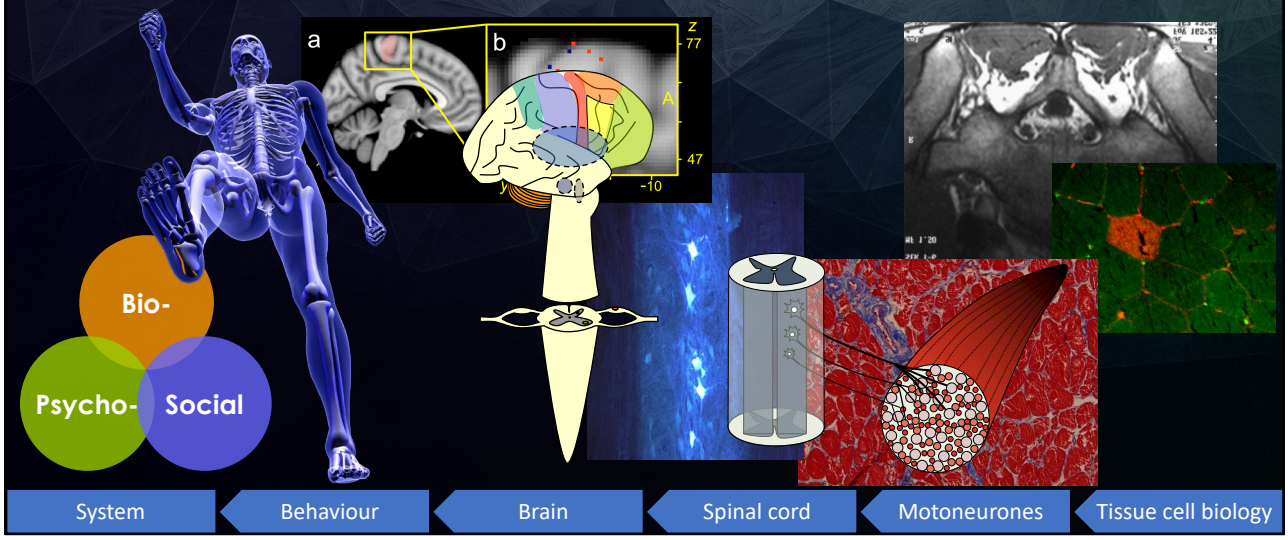
What is pain?

What mechanisms explain the relationship between pain, movement and joint dysfunction?

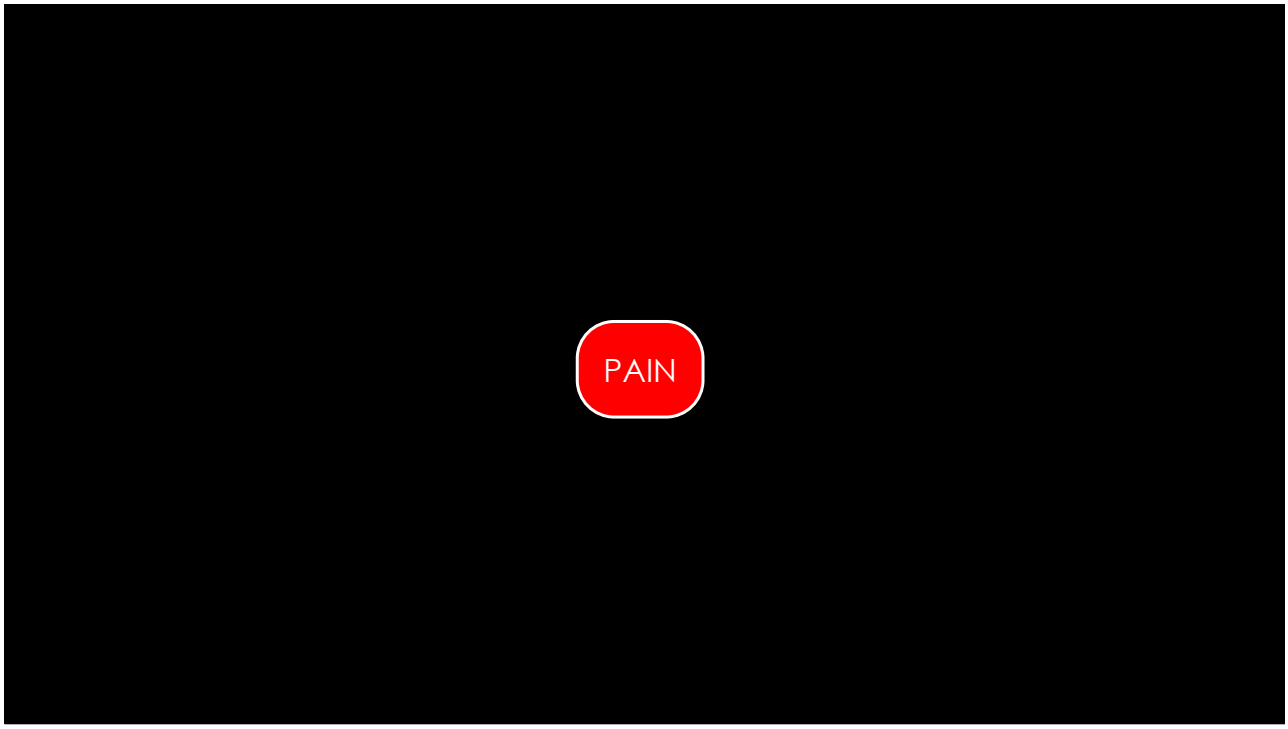
What are the clinical implications?

23

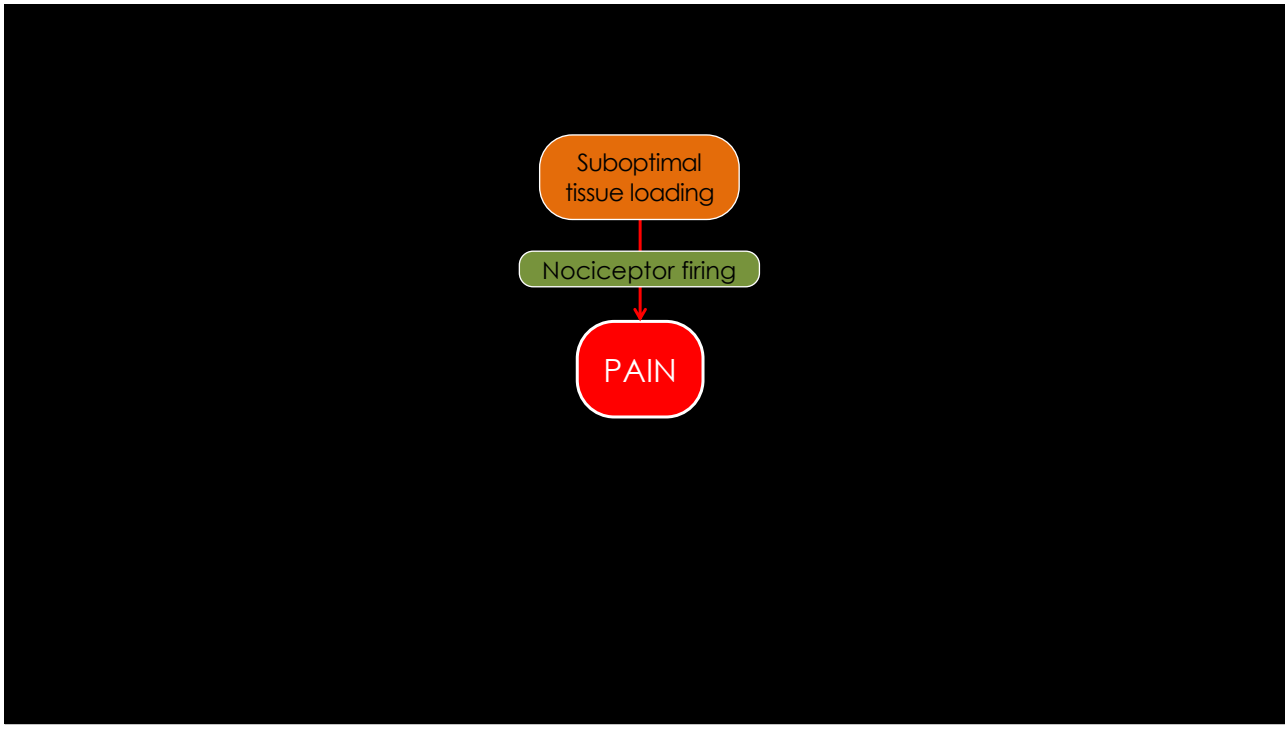
Requires understanding from cells to systems



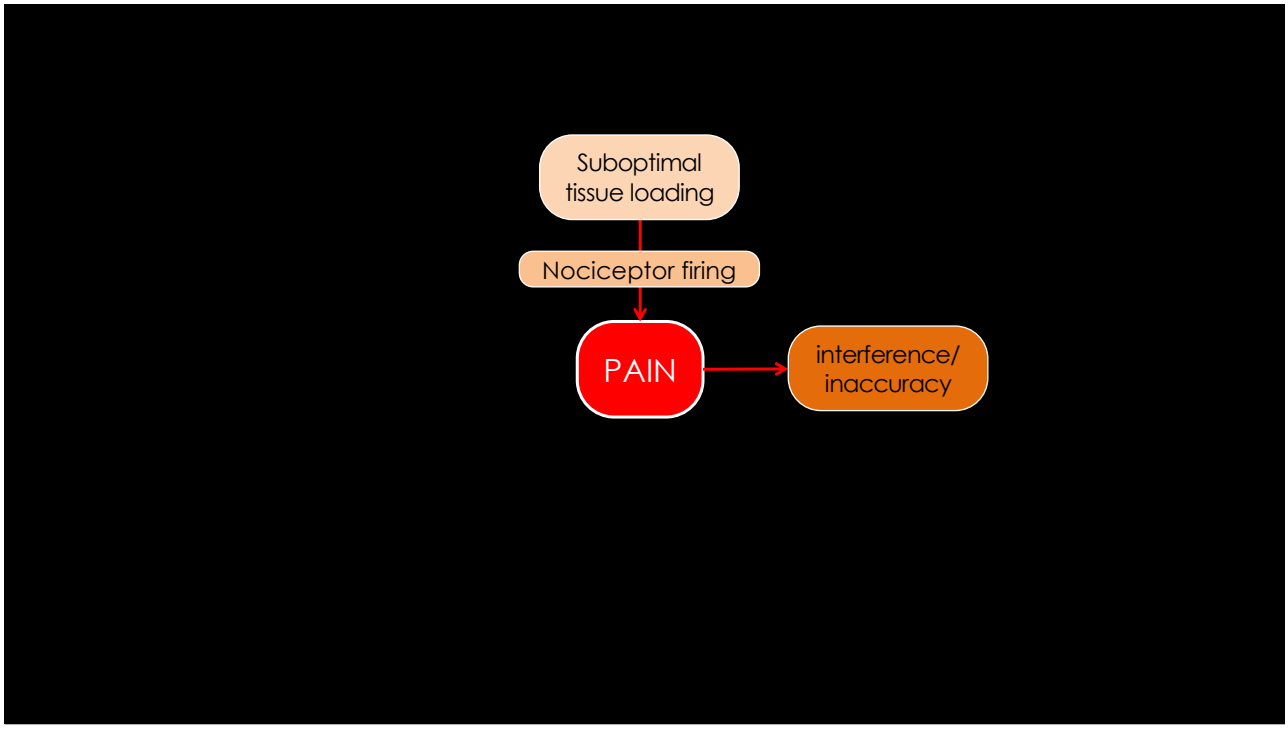
24



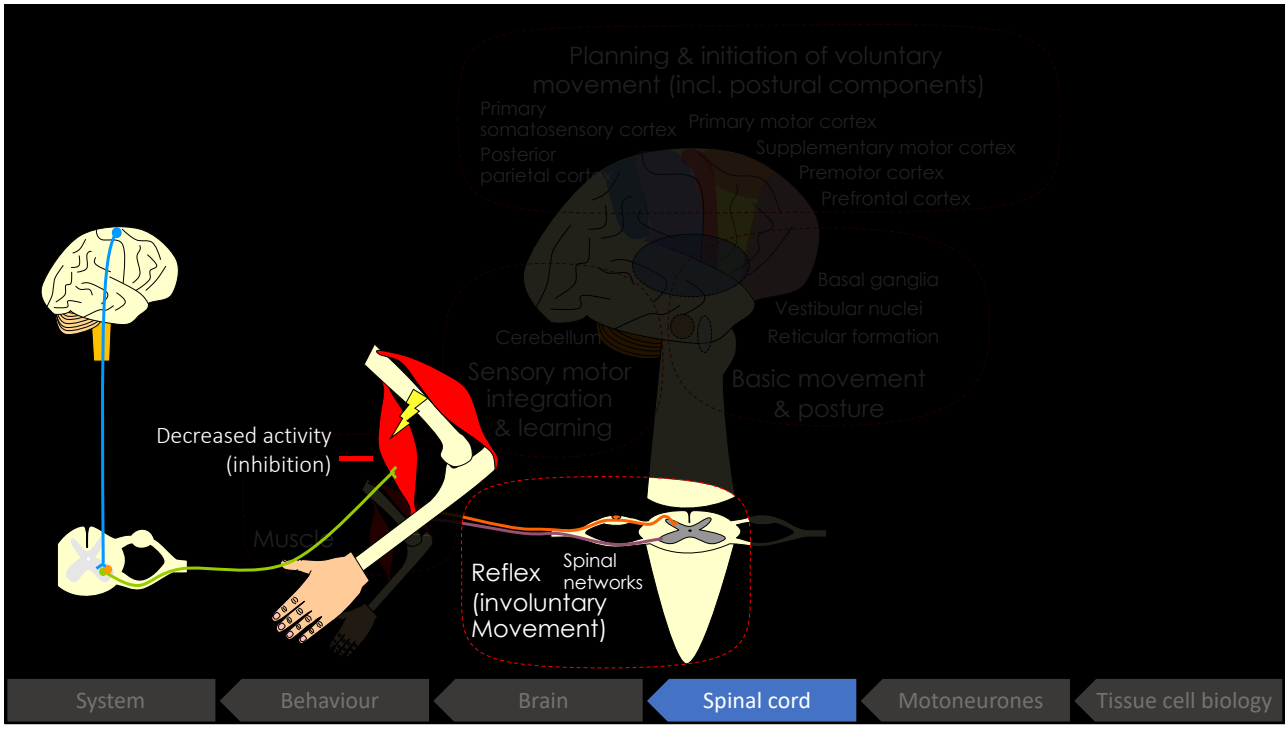
25



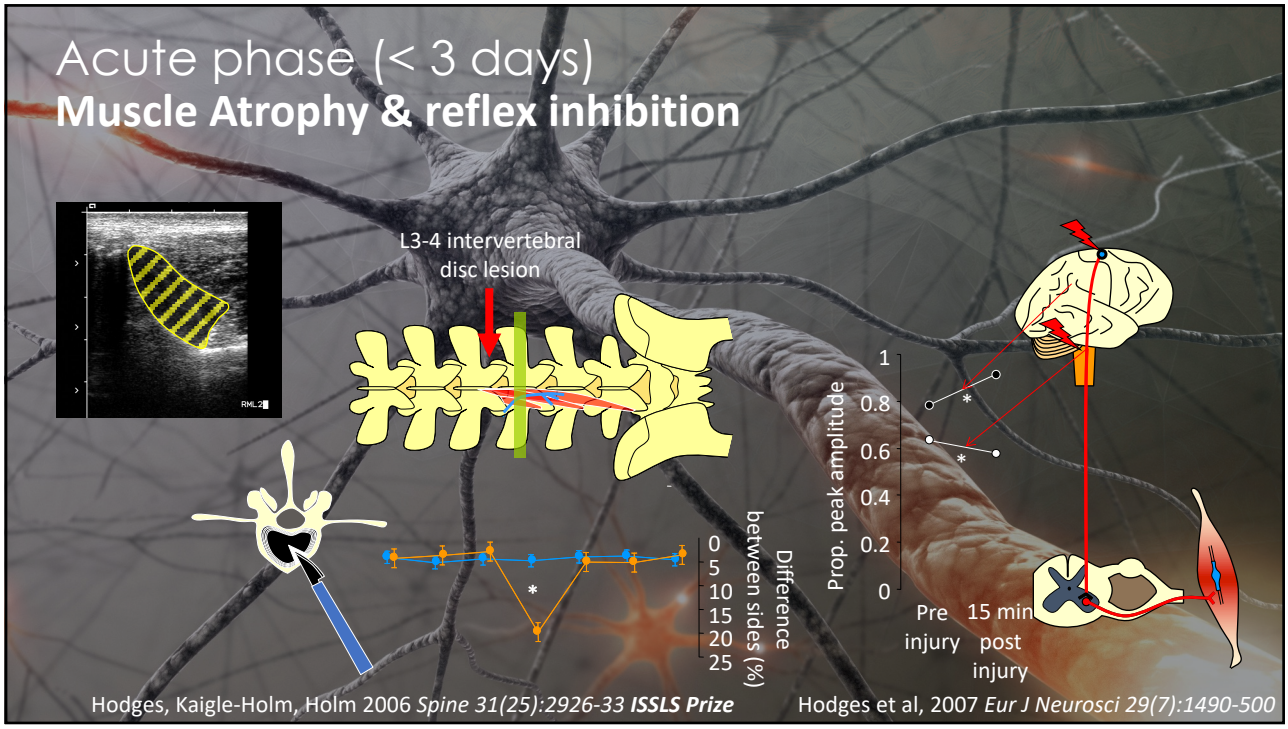
26


Suboptimal tissue loading
Nociceptor firing
PAIN
interference/ inaccuracy

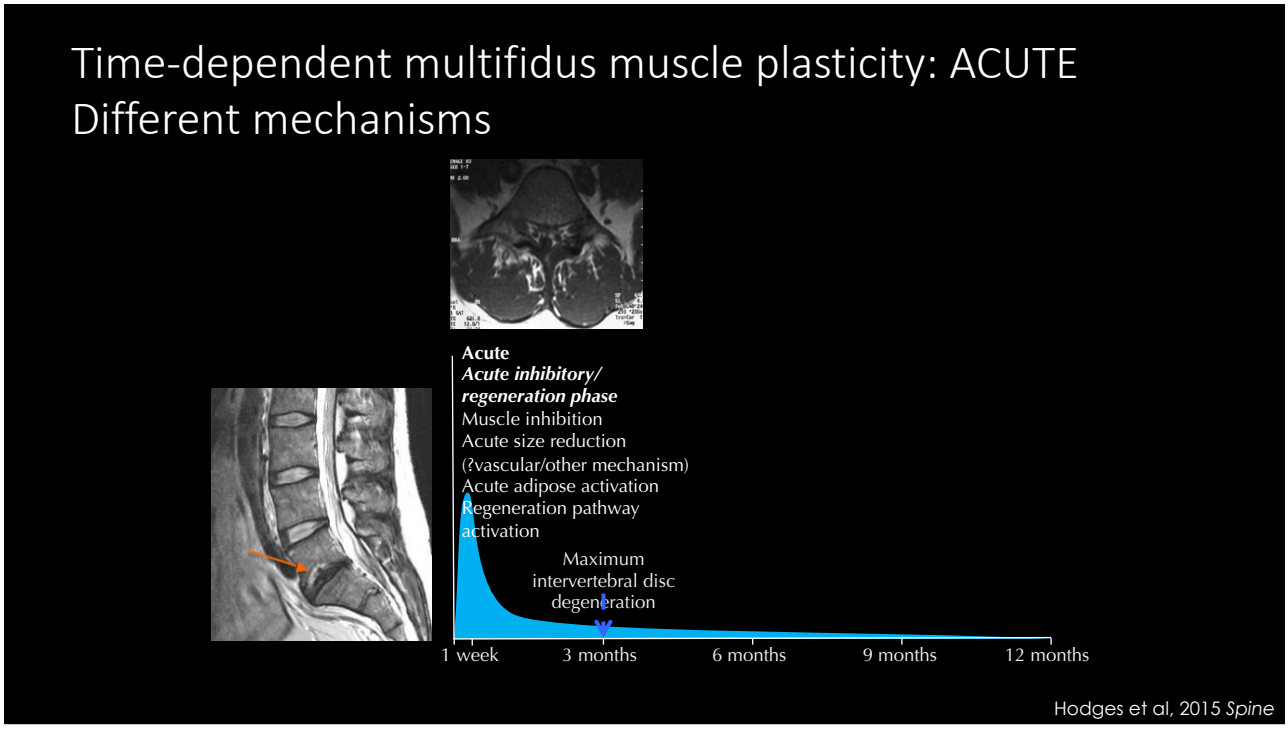
27


Planning & initiation of voluntary movement (incl. postural components)
Primary somatosensory cortex
Posterior parietal cortex
Primary motor cortex
Supplementary motor cortex
Premotor cortex
Prefrontal cortex
Basal ganglia
Vestibular nuclei
Reticular formation
Cerebellum
Sensory motor integration & learning
Basic movement & posture
Decreased activity (inhibition)
Muscle
Reflex (involuntary Movement)
Spinal networks
System
Behaviour
Brain
Spinal cord
Motoneurones
Tissue cell biology

28

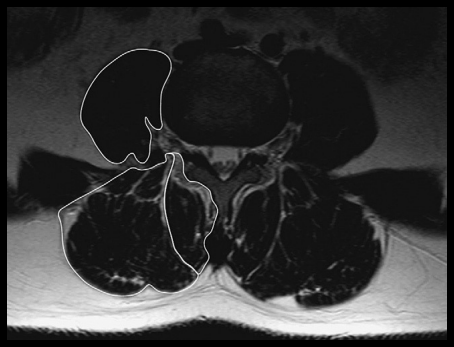

Acute phase (< 3 days)
Muscle Atrophy & reflex inhibition
L3-4 intervertebral disc lesion
Difference between sides (%)
0
5
10
15
20
25
Prop. peak amplitude
1
0.8
0.6
0.4
0.2
0
Pre injury
15 min post injury
Hodges, Kaigle-Holm, Holm 2006 *Spine 31(25):2926-33* ***ISSLS Prize***
Hodges et al, 2007 *Eur J Neurosci 29(7):1490-500*

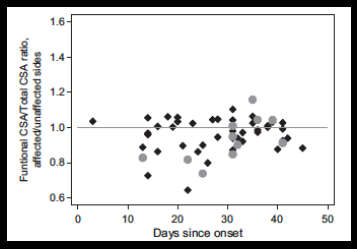
29


Time-dependent multifidus muscle plasticity: ACUTE
Different mechanisms
Acute
Acute inhibitory/ regeneration phase
Muscle inhibition
Acute size reduction
(?vascular/other mechanism)
Acute adipose activation
Regeneration pathway activation
Maximum intervertebral disc degeneration
1 week
3 months
6 months
9 months
12 months
Hodges et al, 2015 *Spine*

30

Subacute phase

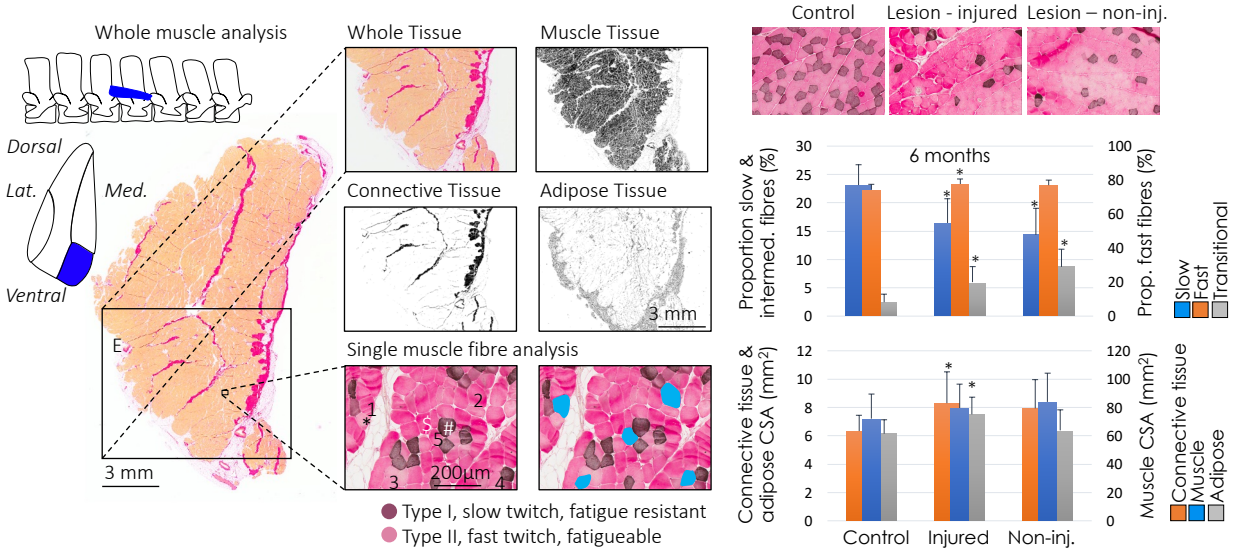




Battie, Niemelainen, Gibbons, Dhillon 2012 *Spine J*

31

Sub-acute phase
Counterintuitive degradation of muscle structure



Hodges, James, Blomster, Hall, Schmid, Shu, Little, Melrose, 2015 *Spine*

32

Possible role of inflammatory response in muscle changes

- Inflammation is increasingly recognized as a mechanism underlying accumulation connective tissue and fat in muscle and other tissues

  Tendon (Barbe et al. 2013)

  Liver, kidney, lung & heart (Weiskirchen et al. 2018)
- TNF expression increased after intervertebral disk disease

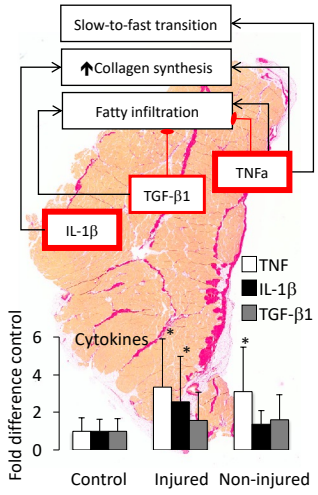
  (Olmarker et al. 1998; Burke et al. 2002)
- Role in muscle remodeling

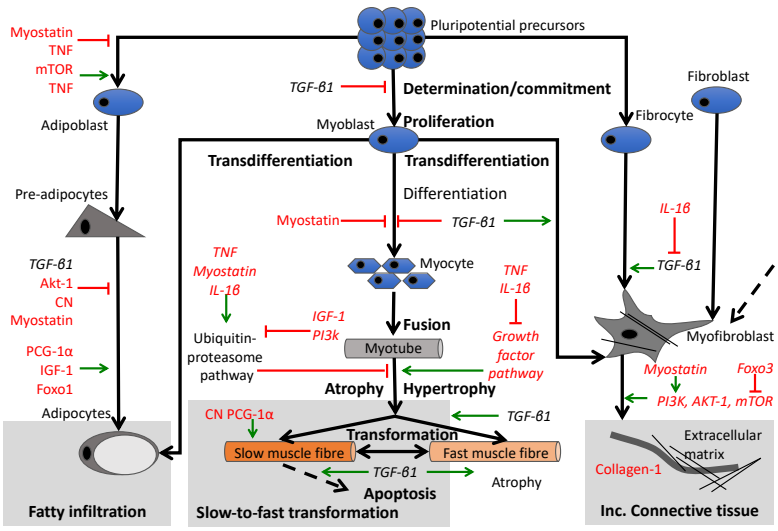
  (Phillips et al 2005; Li et al. 2001)
- Connective tissue and fat are also a source of inflammatory cytokines

  (James et al. 2018)

33

Actively regulated by muscle inflammatory mechanisms



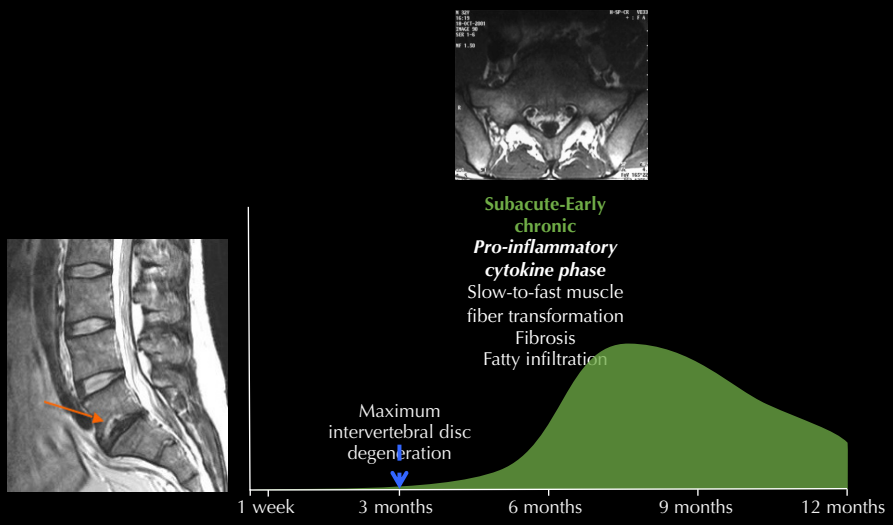


Hodges, James, Blomster, et al, 2014 *Spine 39(13):1010-7*

Hodges, James, Blomster, et al, 2015 *Spine 40(14):1057-71*

34

Time-dependent multifidus muscle plasticity: SUBACUTE Different mechanisms



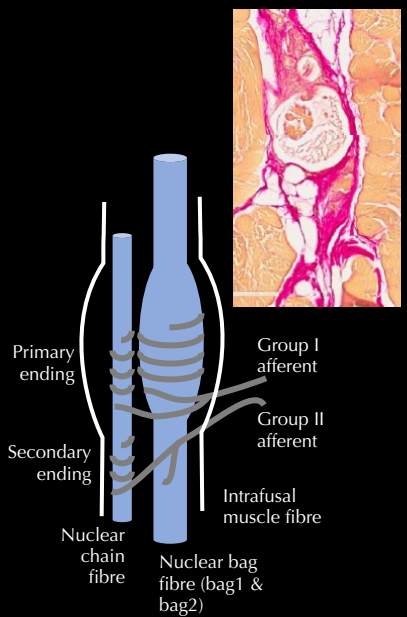
Hodges et al, 2015 *Spine*

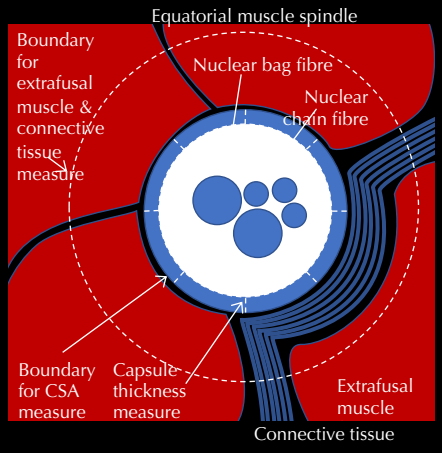
35

Are muscle spindles affected by inflammation?

Afferent information regarding length change & rate of length change

- Structure
  - Intrafusal muscle fibres
  - Connective tissue capsule

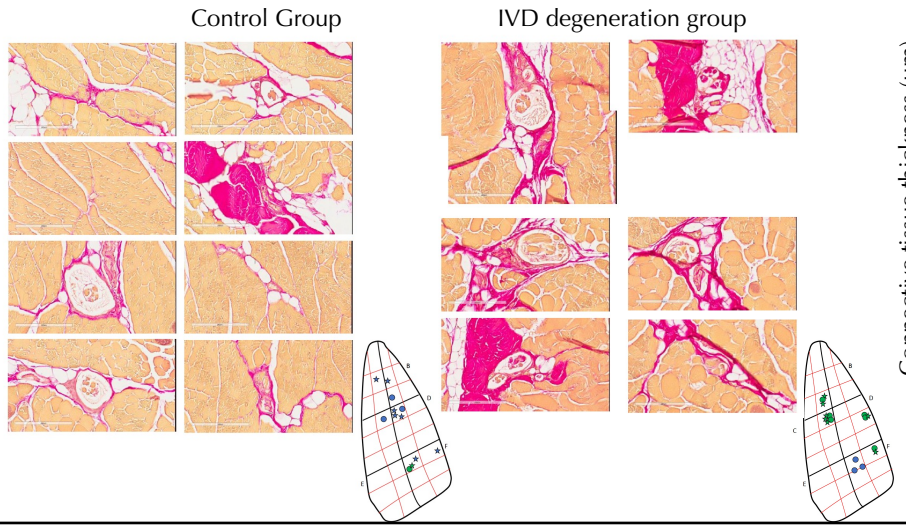




James et al, 2022

36

Muscle spindle morphology – Greater connective tissue thickness after IVD degeneration



37

Implications for muscle spindle function

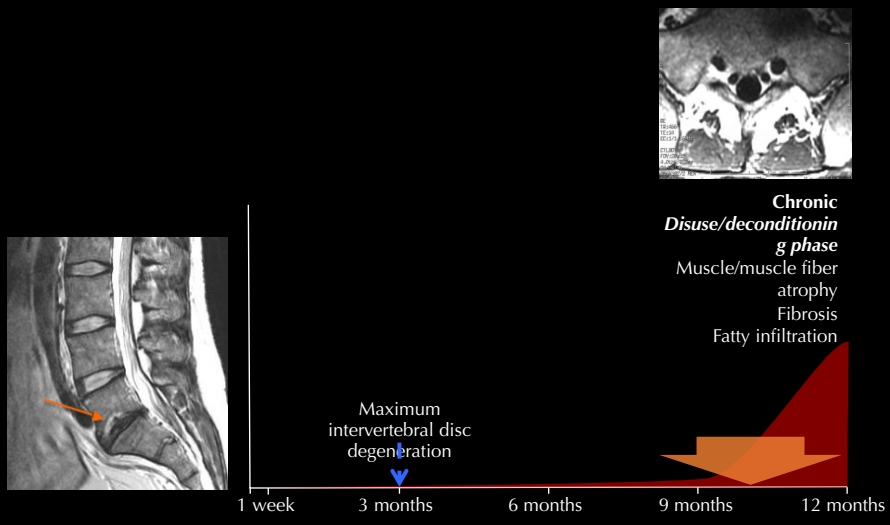
- Increased connective tissue & collagen expression of the muscle spindle capsule
  - Potential impact on mechanical properties
  - Modify transmission of length change to muscle spindles
  - Modify transduction of sensory information

→Might explain some of the proprioceptive deficits identified with low back pain

James et al, 2022 European *Spine Journal*

38

Time-dependent multifidus muscle plasticity: CHRONIC Different mechanisms



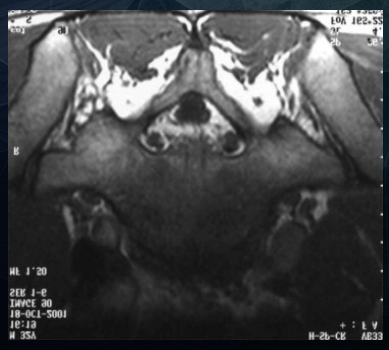
Hodges et al, 2015 *Spine*

39

Does the inflammatory process also occur in humans?

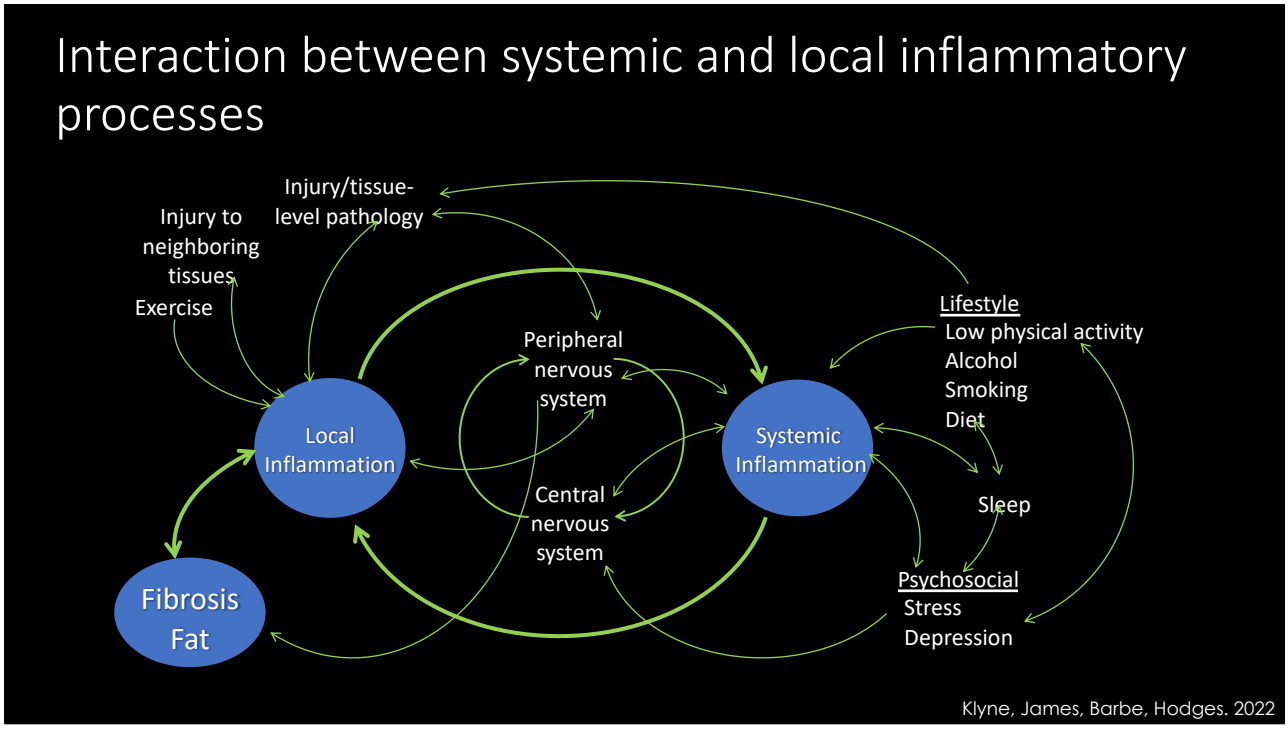
Human IDD: Relationship between fat & expression of TNF

- Multifidus muscle harvested at surgery (n=24)
- TNF expression in multifidus is greater in individuals with higher clinical grade of fat (Kjaer)

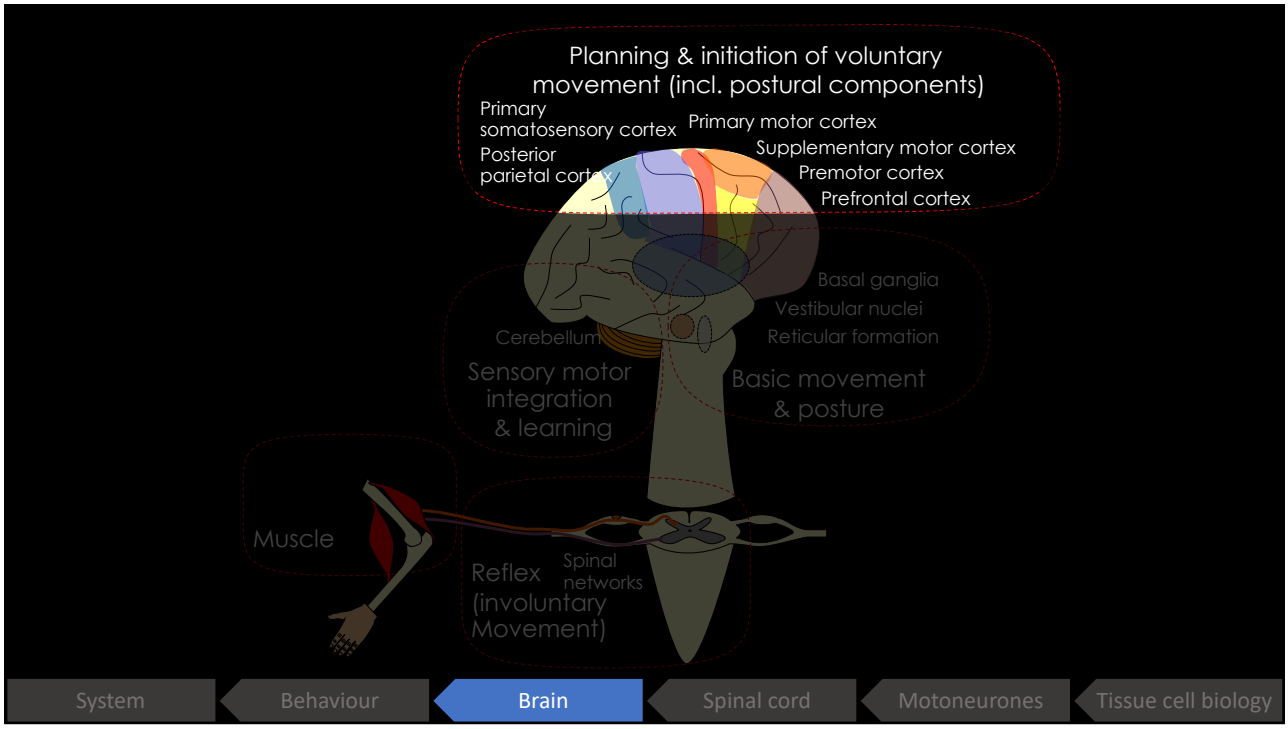


James, Chen, Diwan, Hodges, et al. 2019

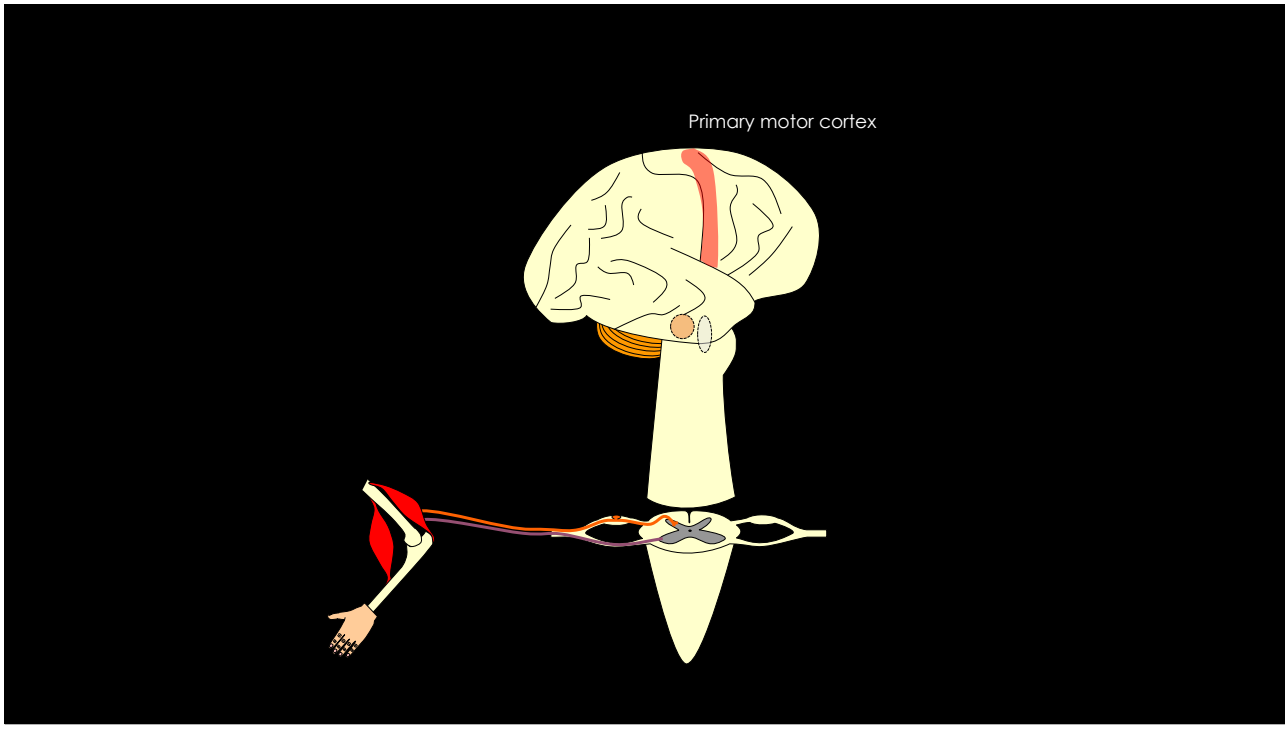
40


Interaction between systemic and local inflammatory processes
Injury/tissue-level pathology
Injury to neighboring tissues
Exercise
Peripheral nervous system
Local Inflammation
Systemic Inflammation
Central nervous system
Fibrosis Fat
Lifestyle
Low physical activity
Alcohol
Smoking
Diet
Sleep
Psychosocial
Stress
Depression
Klyne, James, Barbe, Hodges. 2022

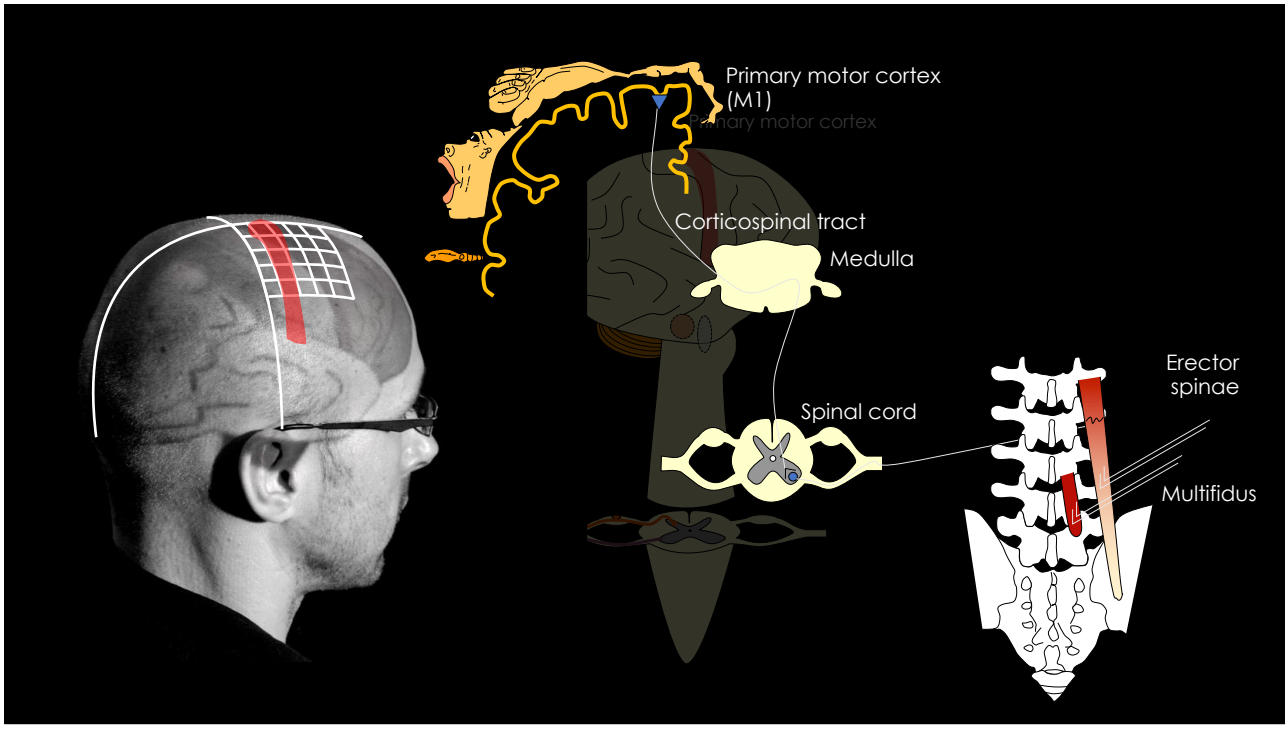
41


Planning & initiation of voluntary movement (incl. postural components)
Primary somatosensory cortex
Primary motor cortex
Supplementary motor cortex
Premotor cortex
Prefrontal cortex
Basal ganglia
Vestibular nuclei
Reticular formation
Cerebellum
Sensory motor integration & learning
Basic movement & posture
Muscle
Reflex (involuntary Movement)
Spinal networks
System
Behaviour
Brain
Spinal cord
Motoneurones
Tissue cell biology

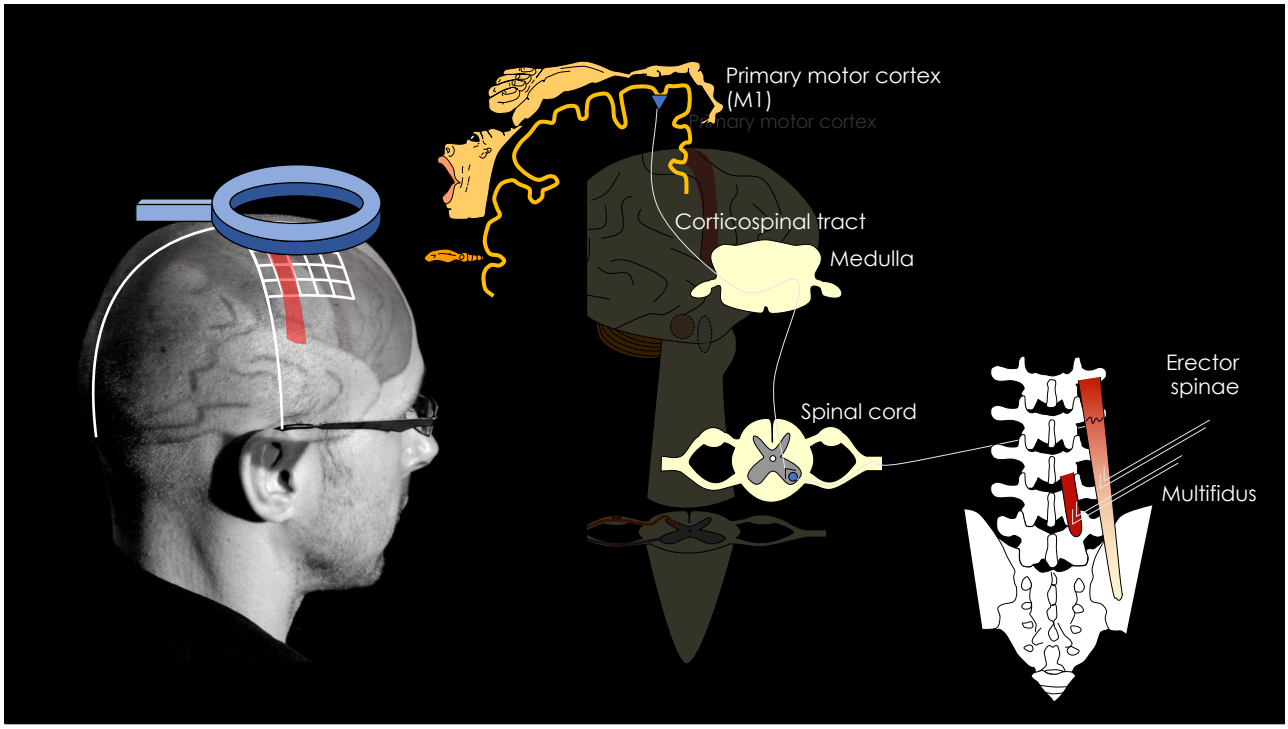
42


Primary motor cortex

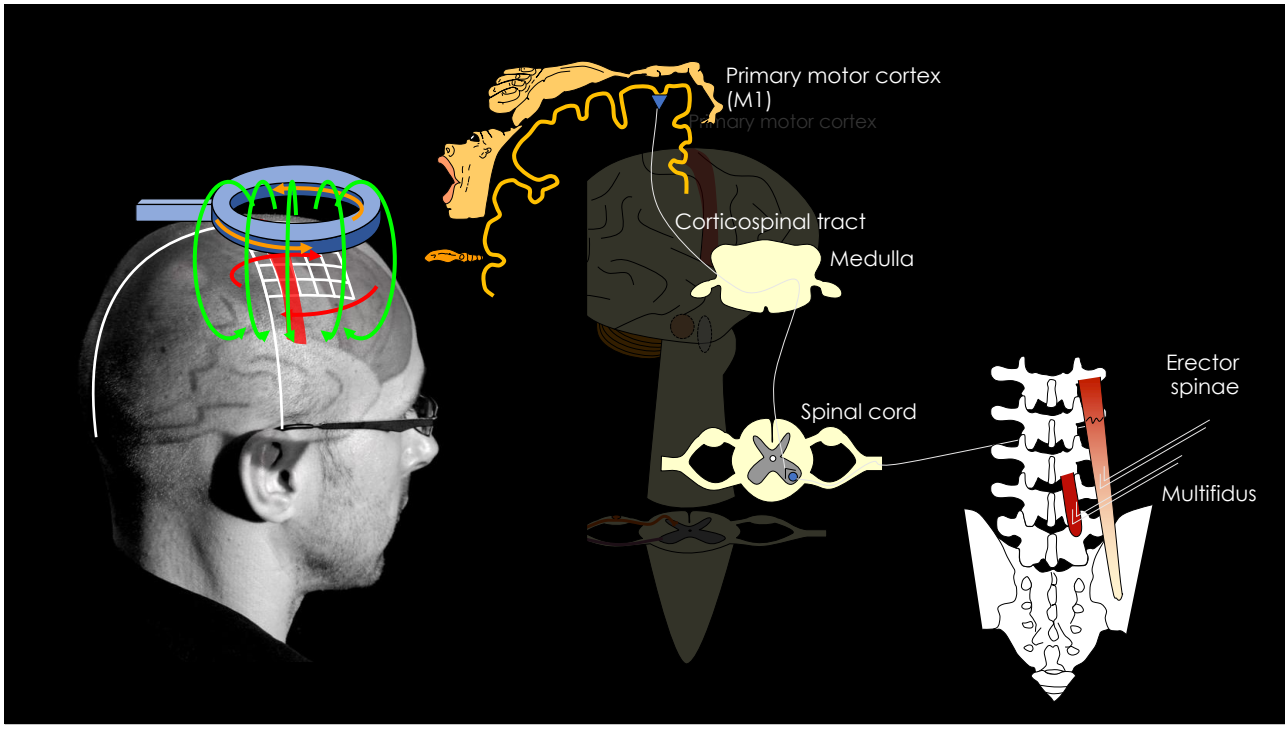
43


Primary motor cortex
(M1)
Corticospinal tract
Medulla
Spinal cord
Erector
spinae
Multifidus

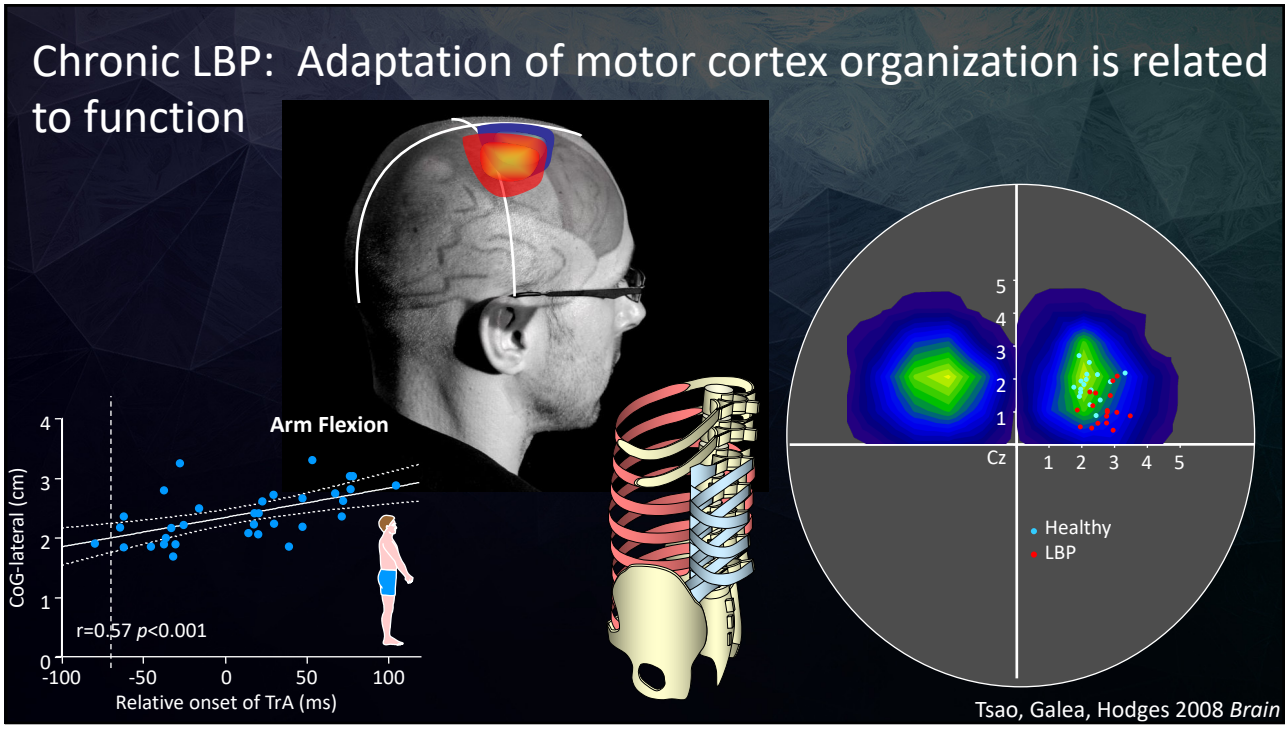
44


Primary motor cortex
(M1)
Corticospinal tract
Medulla
Spinal cord
Erector
spinae
Multifidus

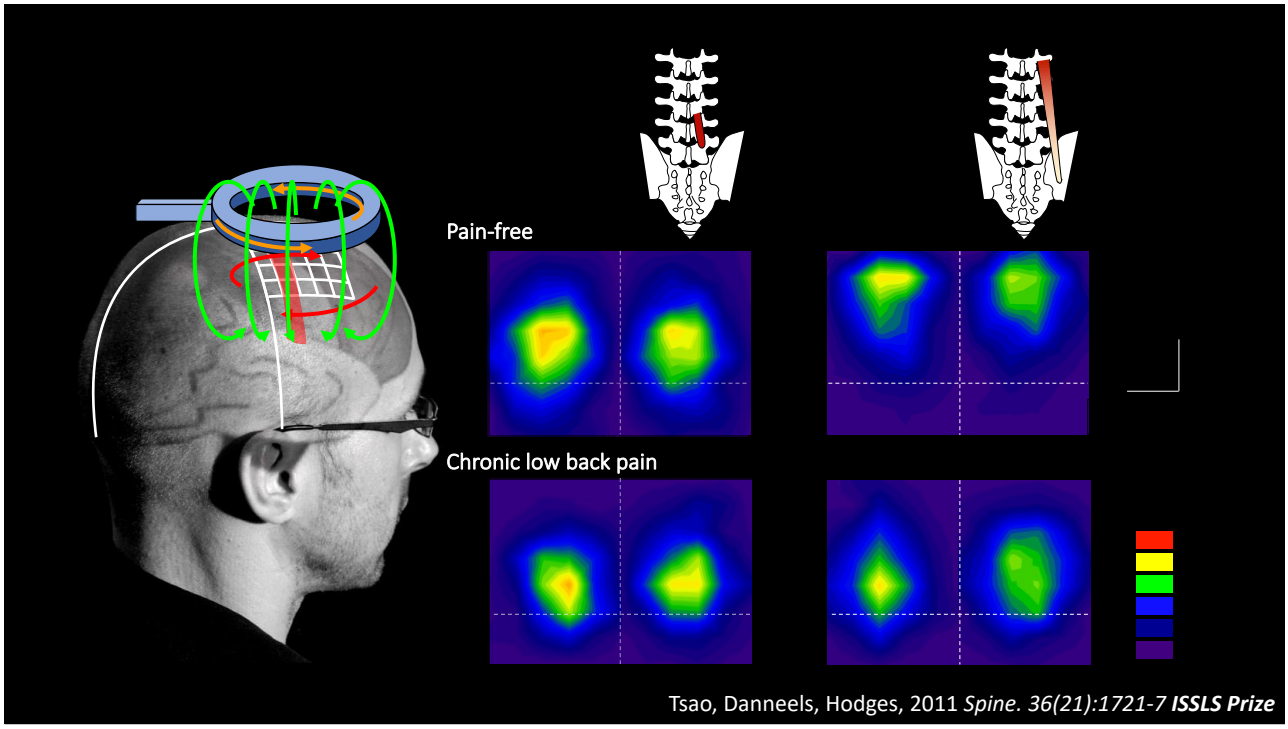
45


Primary motor cortex
(M1)
Corticospinal tract
Medulla
Spinal cord
Erector
spinae
Multifidus

46

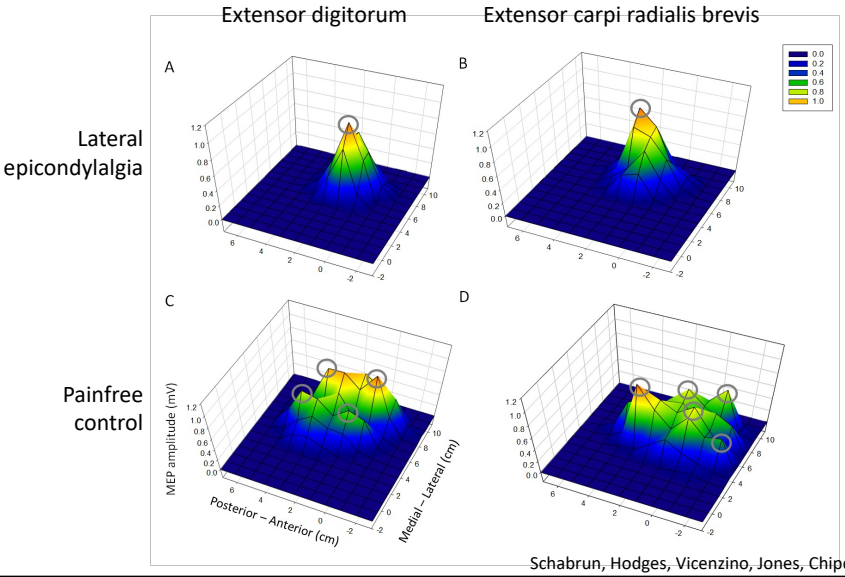

Chronic LBP: Adaptation of motor cortex organization is related to function
Arm Flexion
CoG-lateral (cm)
Relative onset of TrA (ms)
r=0.57 p<0.001
Cz
Healthy
LBP
Tsao, Galea, Hodges 2008 Brain

47


Pain-free
Chronic low back pain
Tsao, Danneels, Hodges, 2011 Spine. 36(21):1721-7 ISSLS Prize

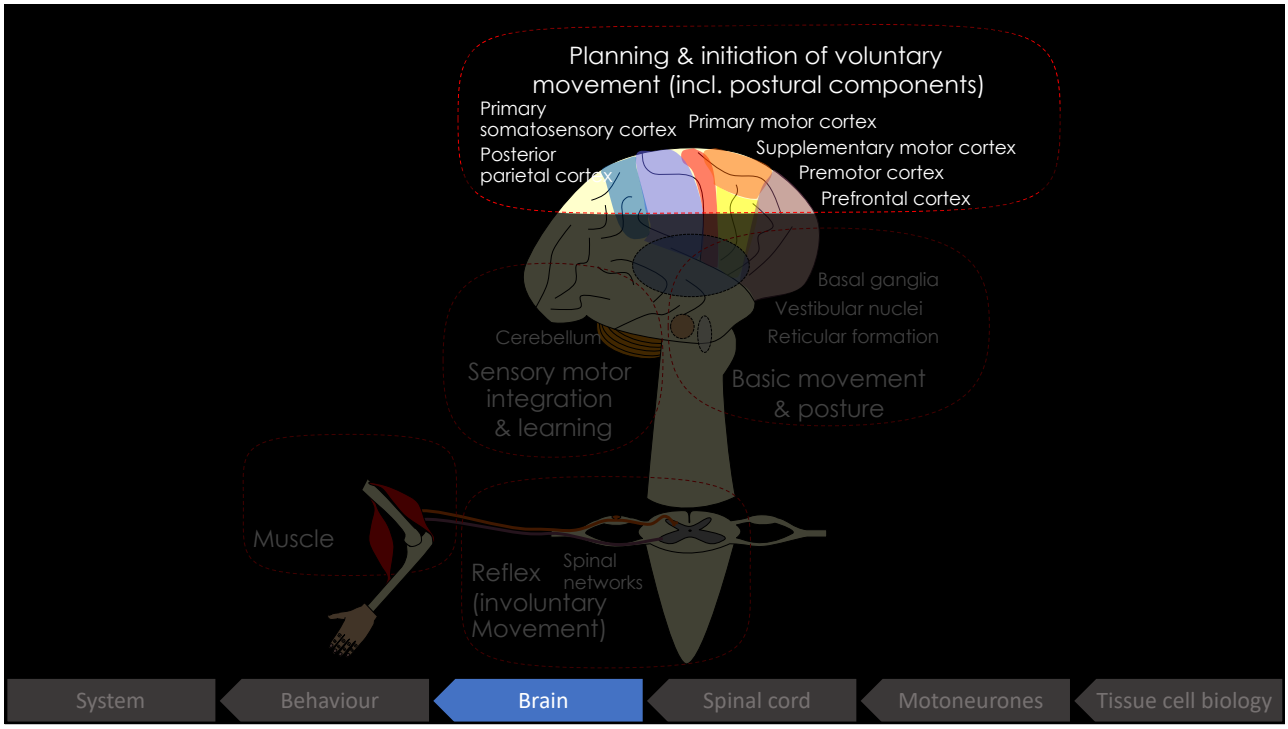
48

Different organisation of the motor cortex in tennis elbow

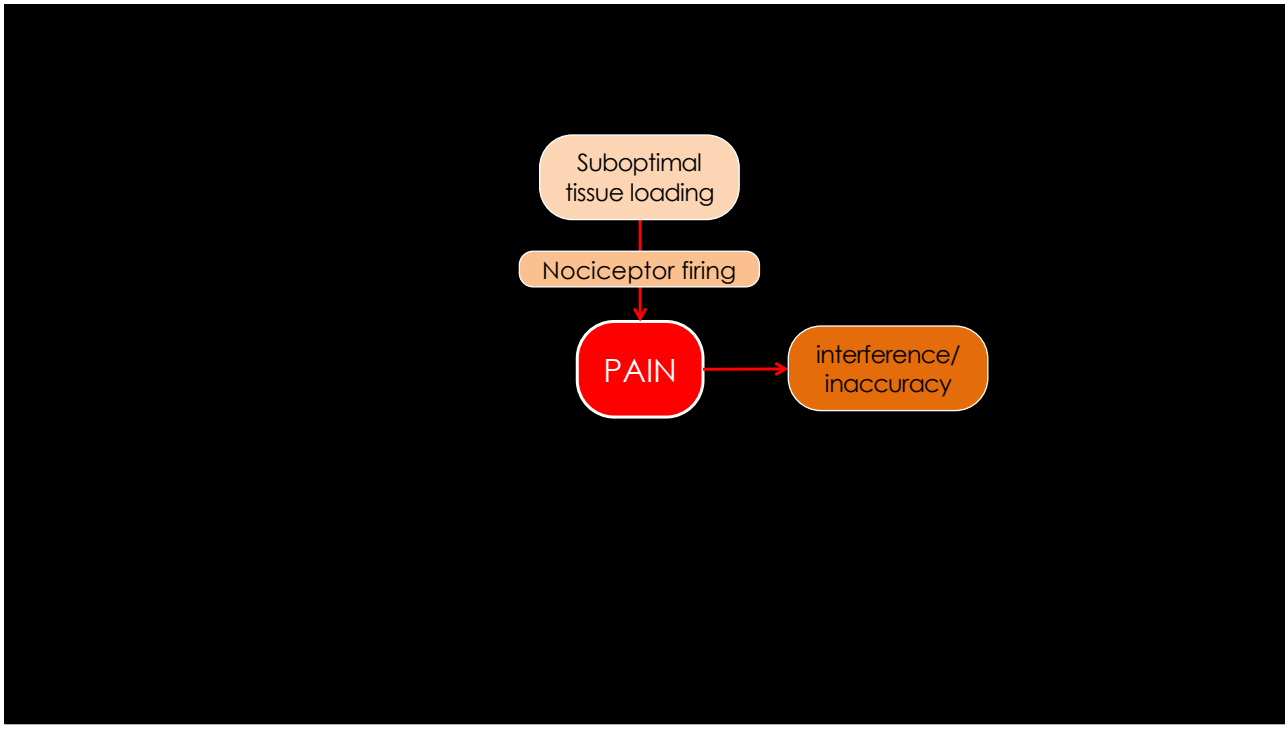


Schabrun, Hodges, Vicenzino, Jones, Chipchase (2015) *Med Sci Sports Exerc*

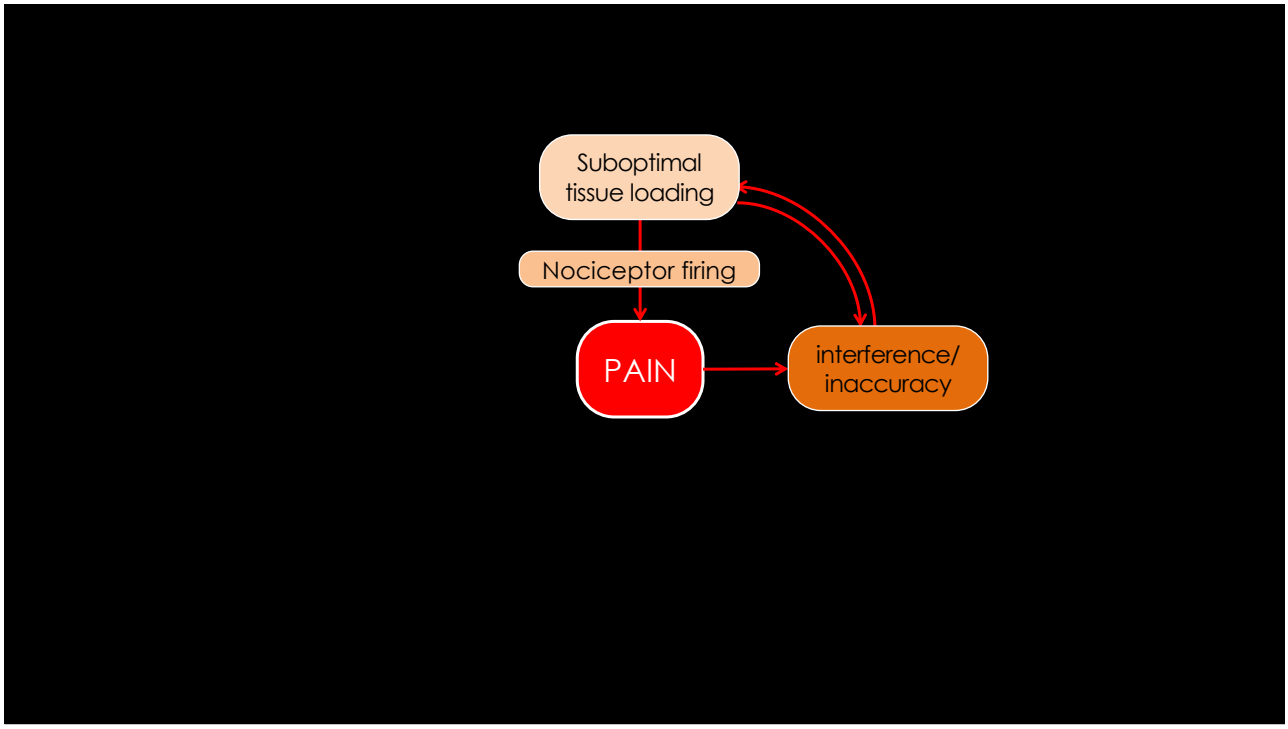
49



50


Suboptimal tissue loading
Nociceptor firing
PAIN
interference/ inaccuracy

51

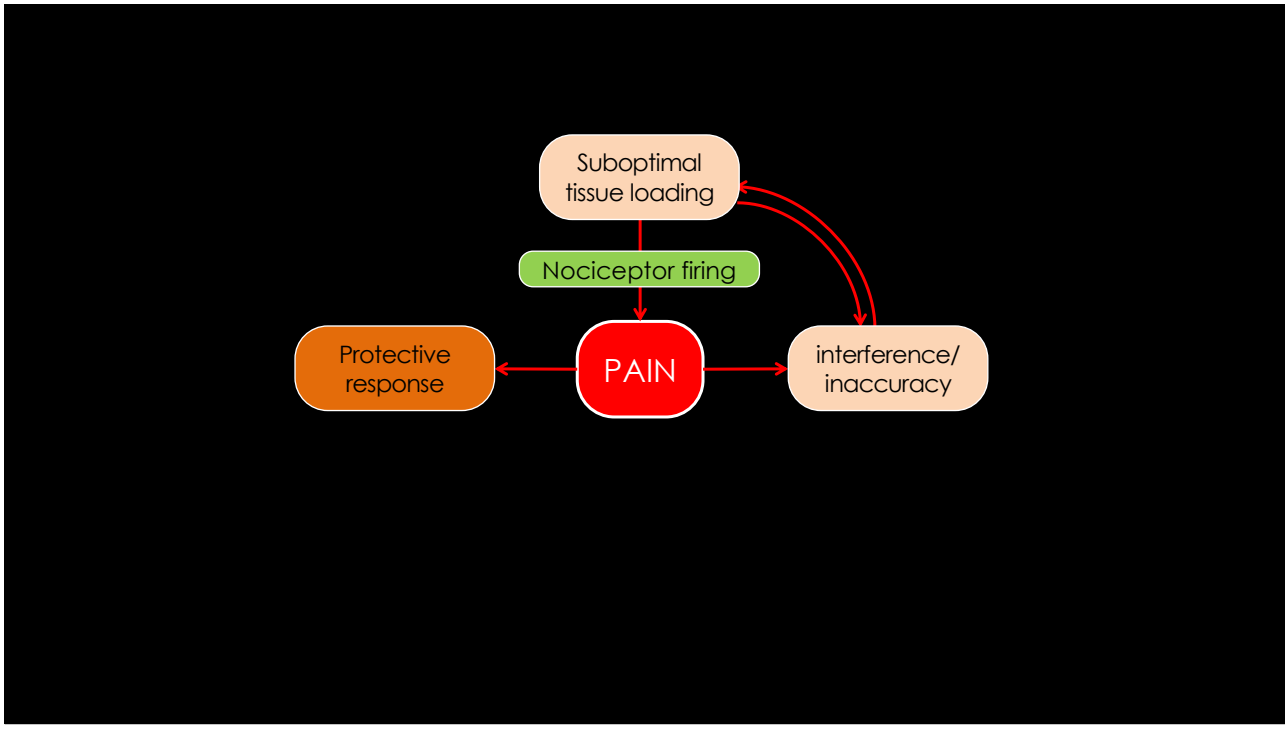

Suboptimal tissue loading
Nociceptor firing
PAIN
interference/ inaccuracy

52

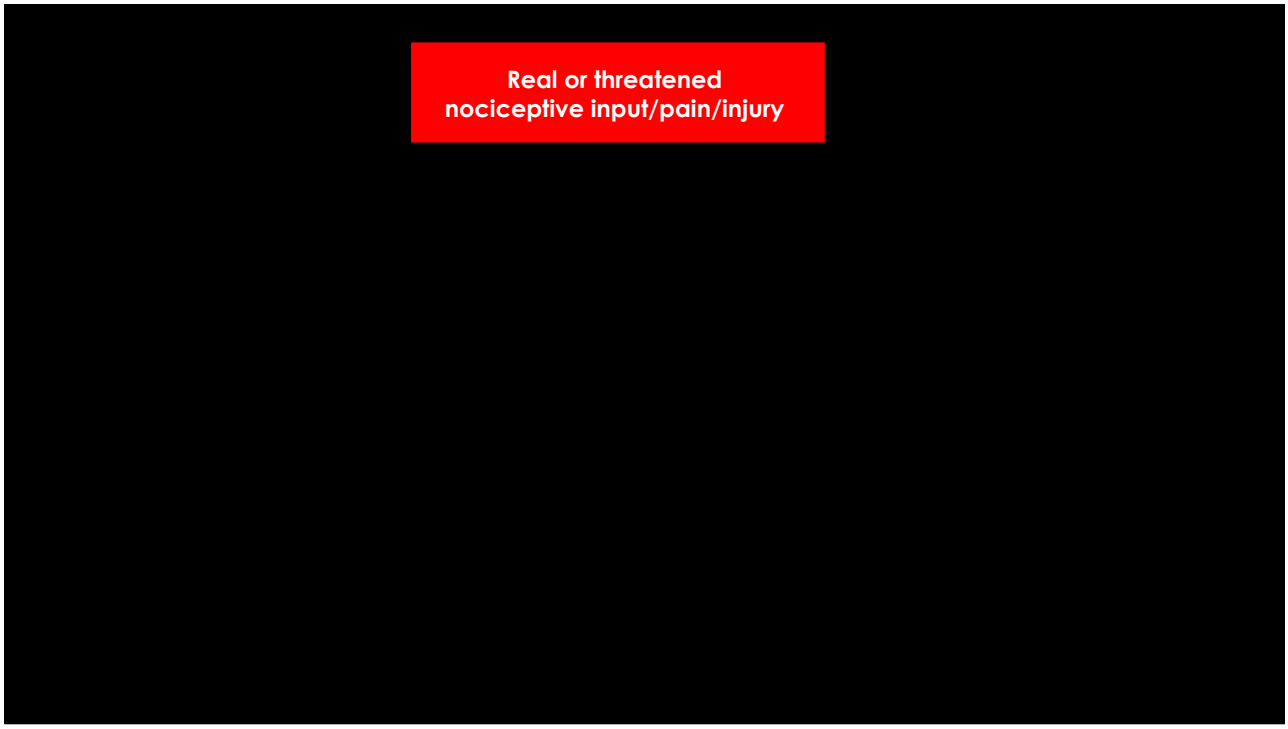
Implication for rehabilitation

- Restoration of **motor control** may contribute to recovery of low back pain – if **suboptimal loading** of tissue contributes to symptoms
- This may require **motor learning** interventions

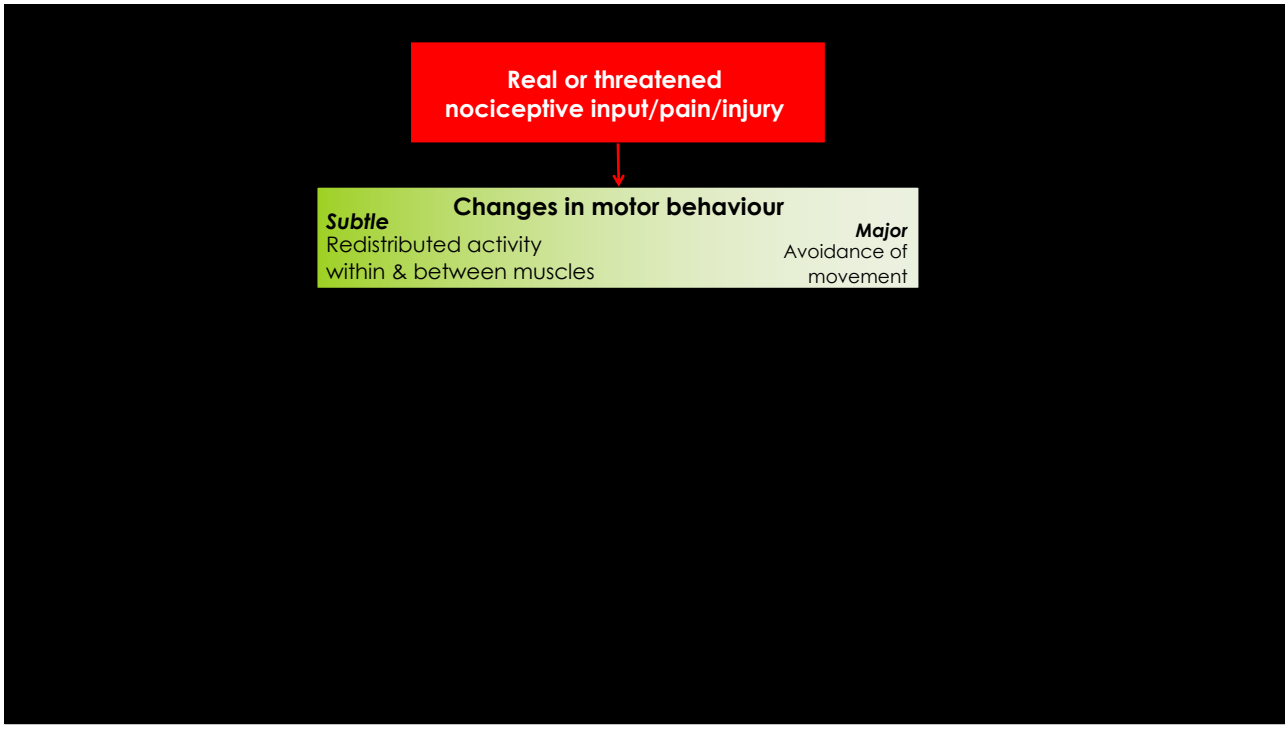
53



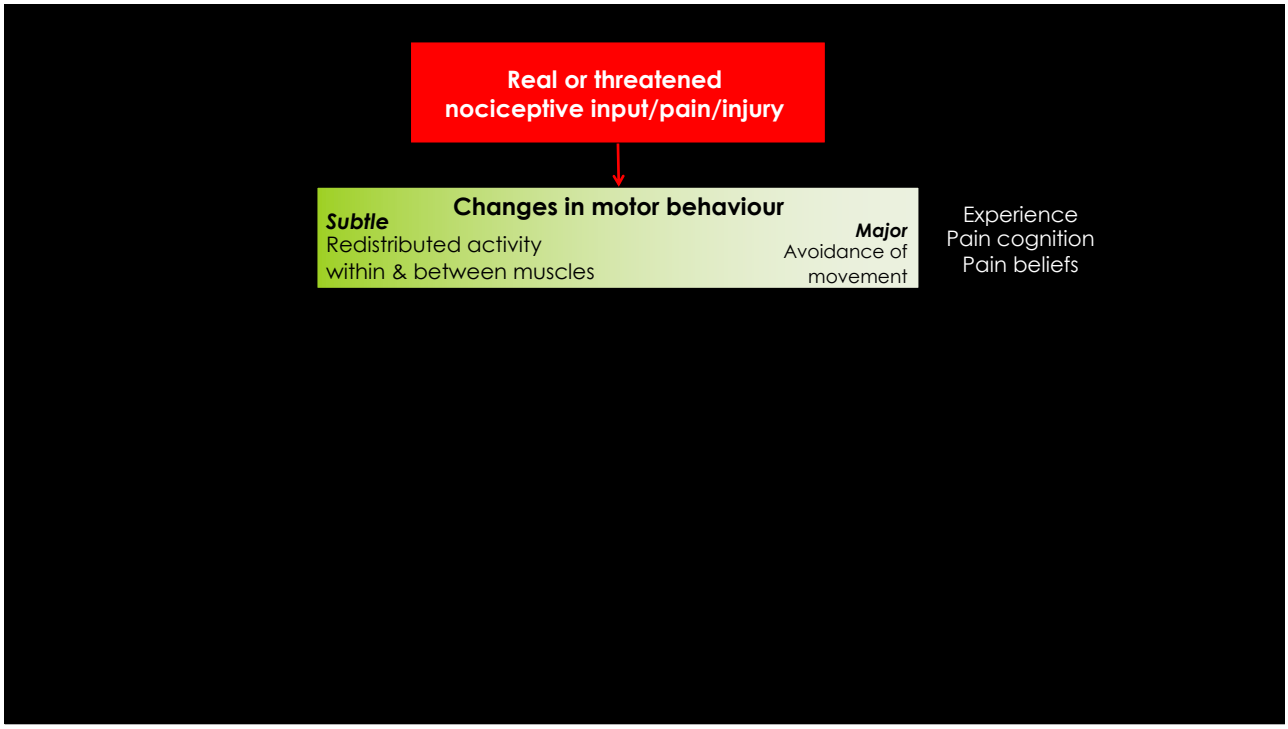
54


Real or threatened
nociceptive input/pain/injury

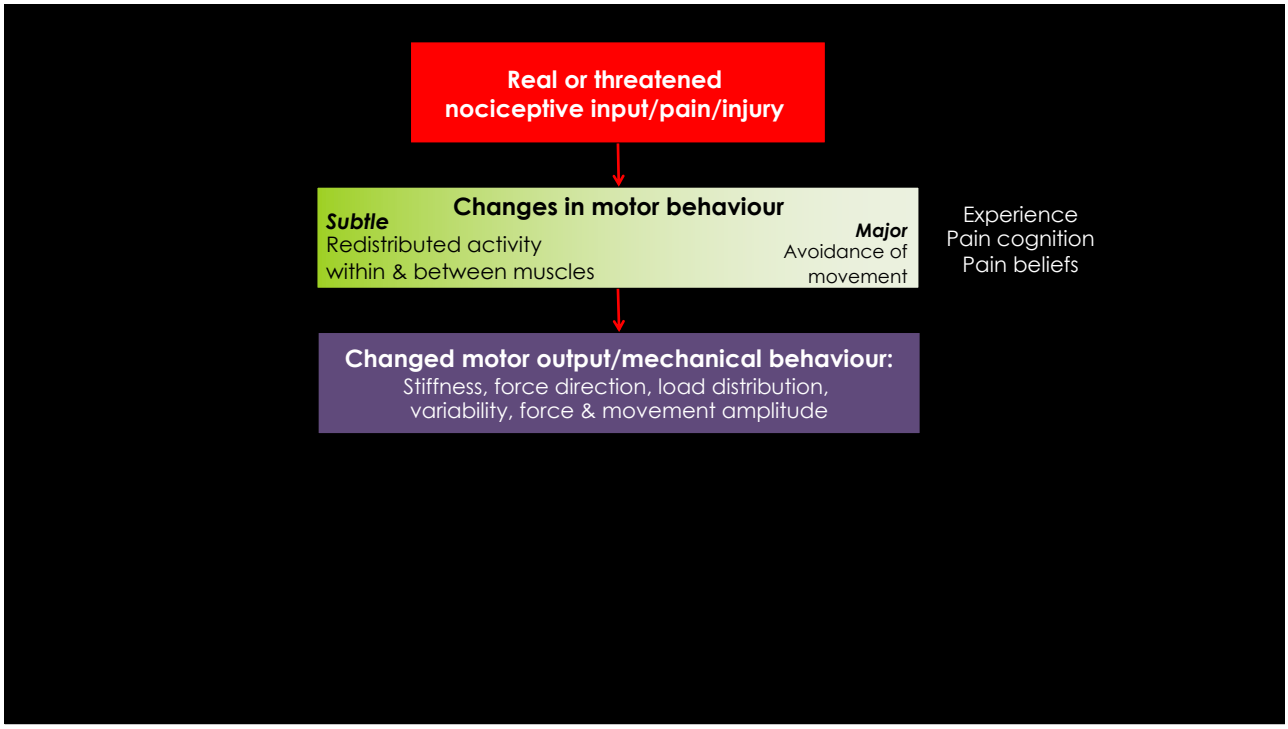
55


Real or threatened
nociceptive input/pain/injury
Changes in motor behaviour
Subtle
Redistributed activity
within & between muscles
Major
Avoidance of
movement

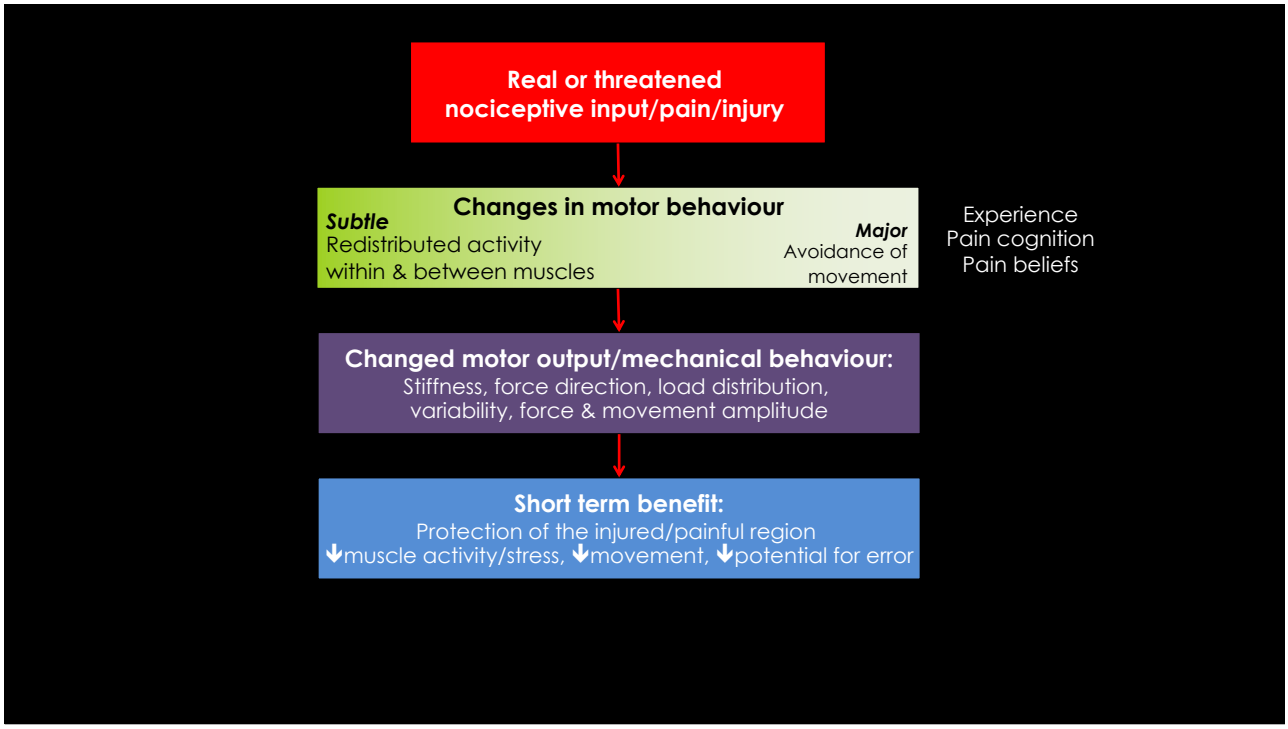
56


Real or threatened
nociceptive input/pain/injury
Changes in motor behaviour
Subtle
Redistributed activity
within & between muscles
Major
Avoidance of
movement
Experience
Pain cognition
Pain beliefs

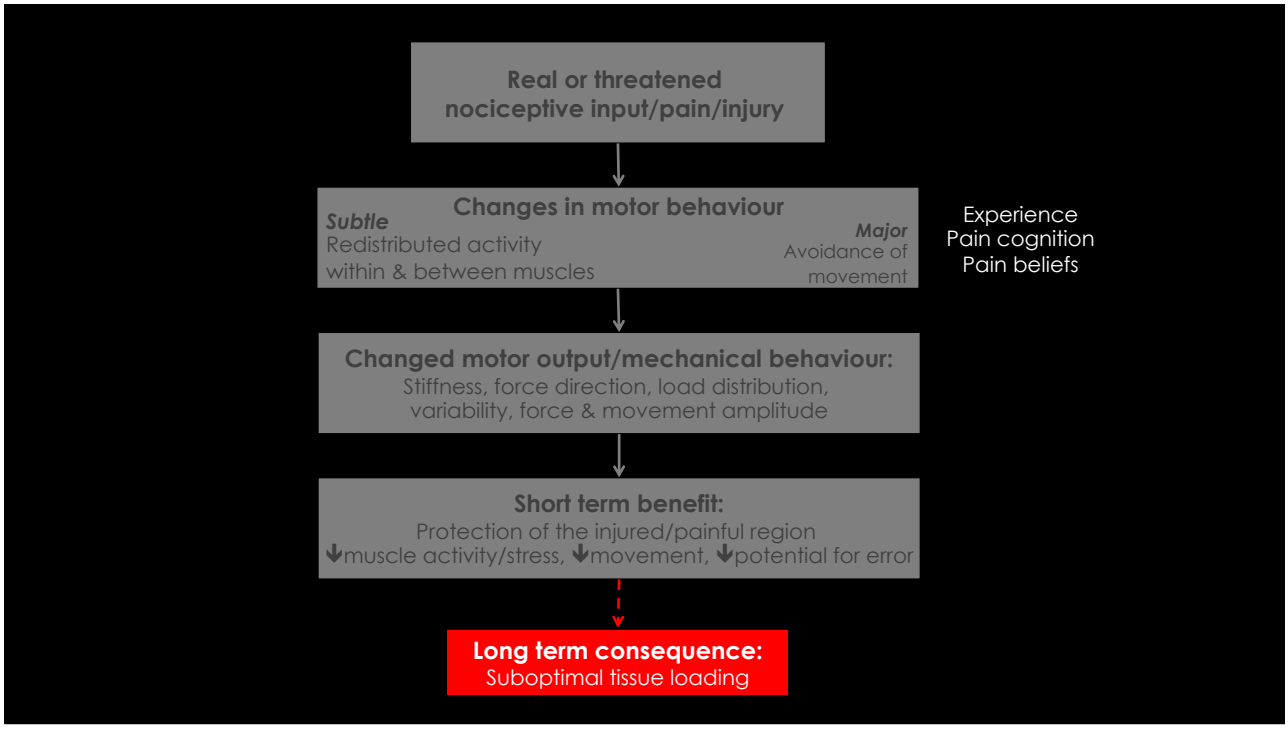
57


Real or threatened
nociceptive input/pain/injury
Changes in motor behaviour
Subtle
Redistributed activity
within & between muscles
Major
Avoidance of
movement
Experience
Pain cognition
Pain beliefs
Changed motor output/mechanical behaviour:
Stiffness, force direction, load distribution,
variability, force & movement amplitude

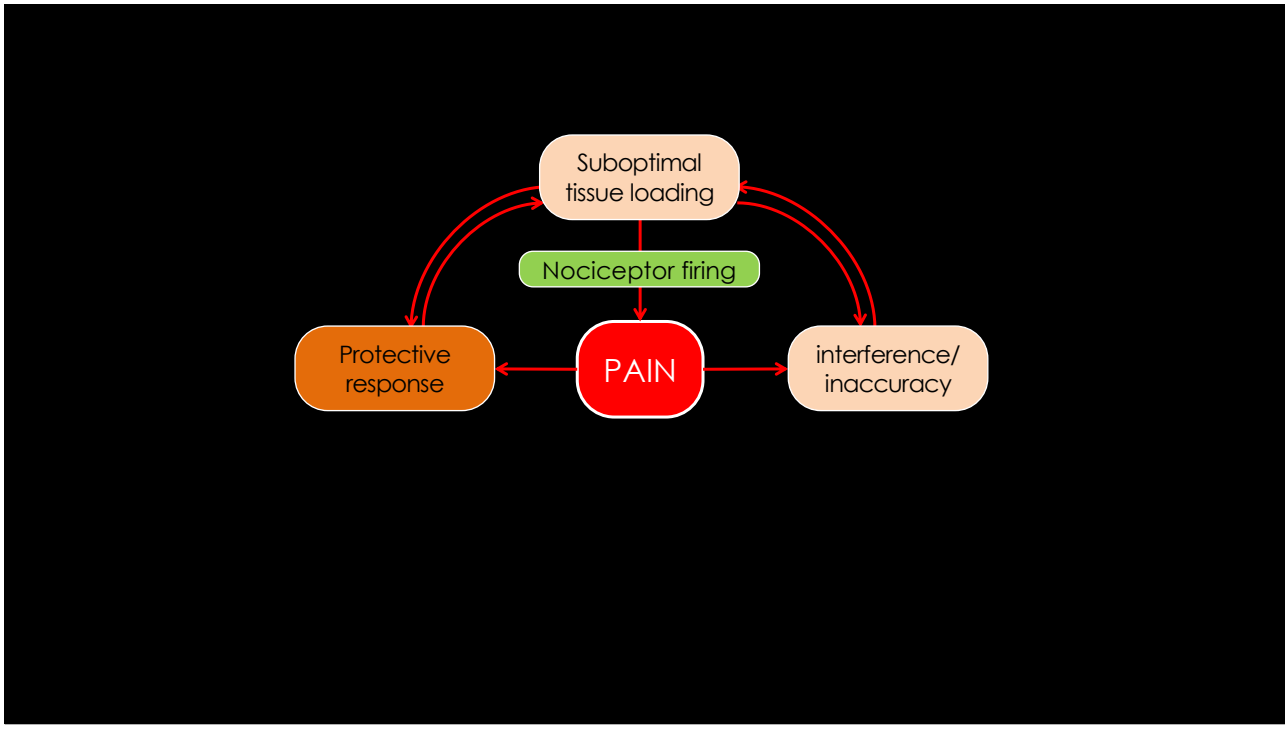
58


Real or threatened nociceptive input/pain/injury
Changes in motor behaviour
Subtle
Redistributed activity within & between muscles
Major
Avoidance of movement
Experience
Pain cognition
Pain beliefs
Changed motor output/mechanical behaviour:
Stiffness, force direction, load distribution, variability, force & movement amplitude
Short term benefit:
Protection of the injured/painful region
↓muscle activity/stress, ↓movement, ↓potential for error

59


Real or threatened nociceptive input/pain/injury
Changes in motor behaviour
Subtle
Redistributed activity within & between muscles
Major
Avoidance of movement
Experience
Pain cognition
Pain beliefs
Changed motor output/mechanical behaviour:
Stiffness, force direction, load distribution, variability, force & movement amplitude
Short term benefit:
Protection of the injured/painful region
↓muscle activity/stress, ↓movement, ↓potential for error
Long term consequence:
Suboptimal tissue loading

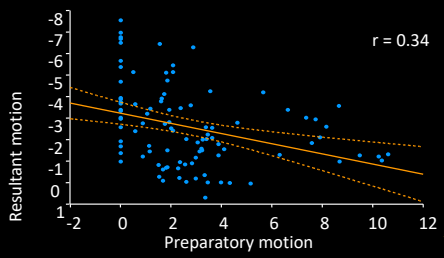
60



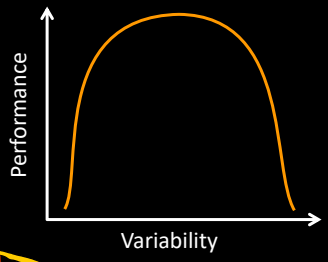
61

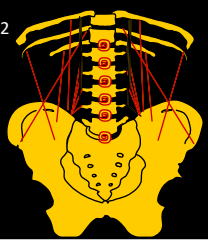
Long term consequences

Reduces movement which is important for dissipation of forces



Reduced movement variability

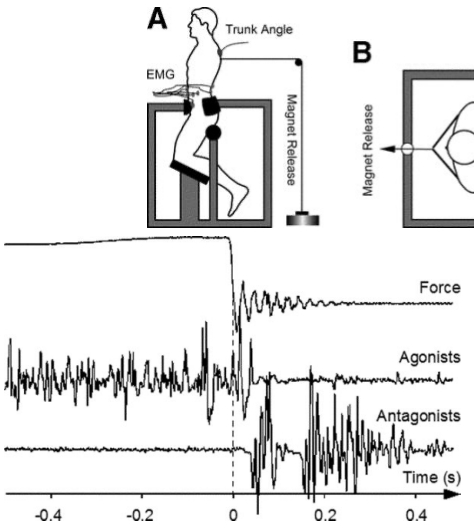




Increases joint loading

62

Delayed "offset" of trunk muscle activity predicts future LBP



N=292 college athletes

Episode of LBP
Longer delay: 77 [36] vs. 63 [31] ms
Fewer muscles off: 0.8[1] vs. 1.3[1.1]

Odds of low back pain ↑ 3% for each 1 ms change

Cholewicki, Stifles, Shah, et al., 2005 **ISSLS Prize** *Spine*

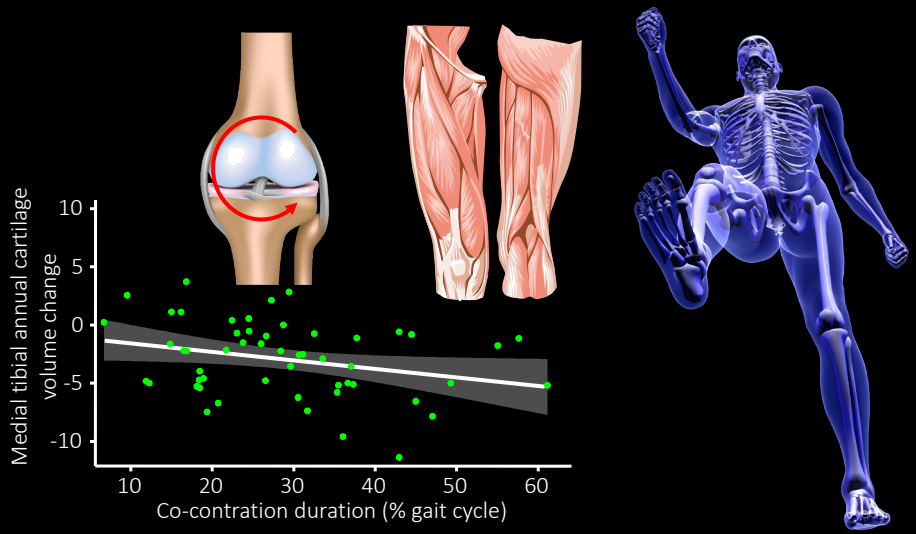
63

Protective adaptation can become part of the problem

Knee muscle co-contraction

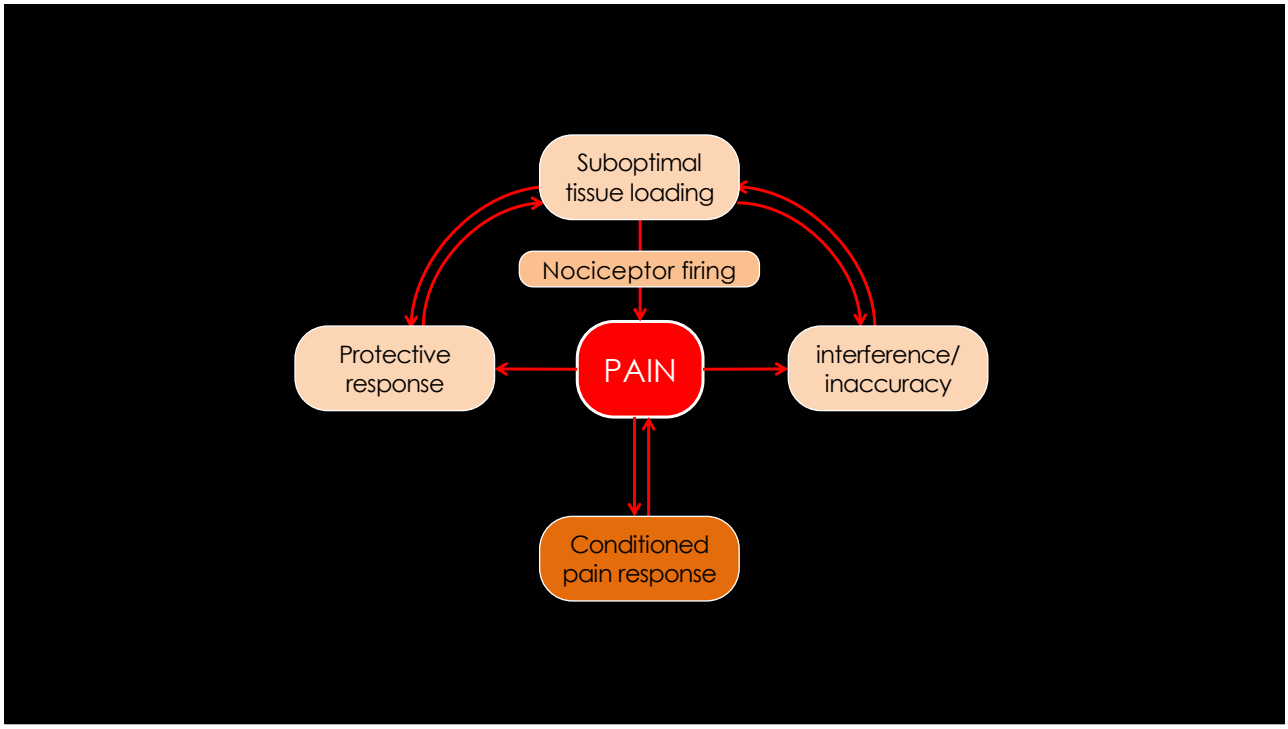
Short term
Protection

Long term
↑Cartilage loss

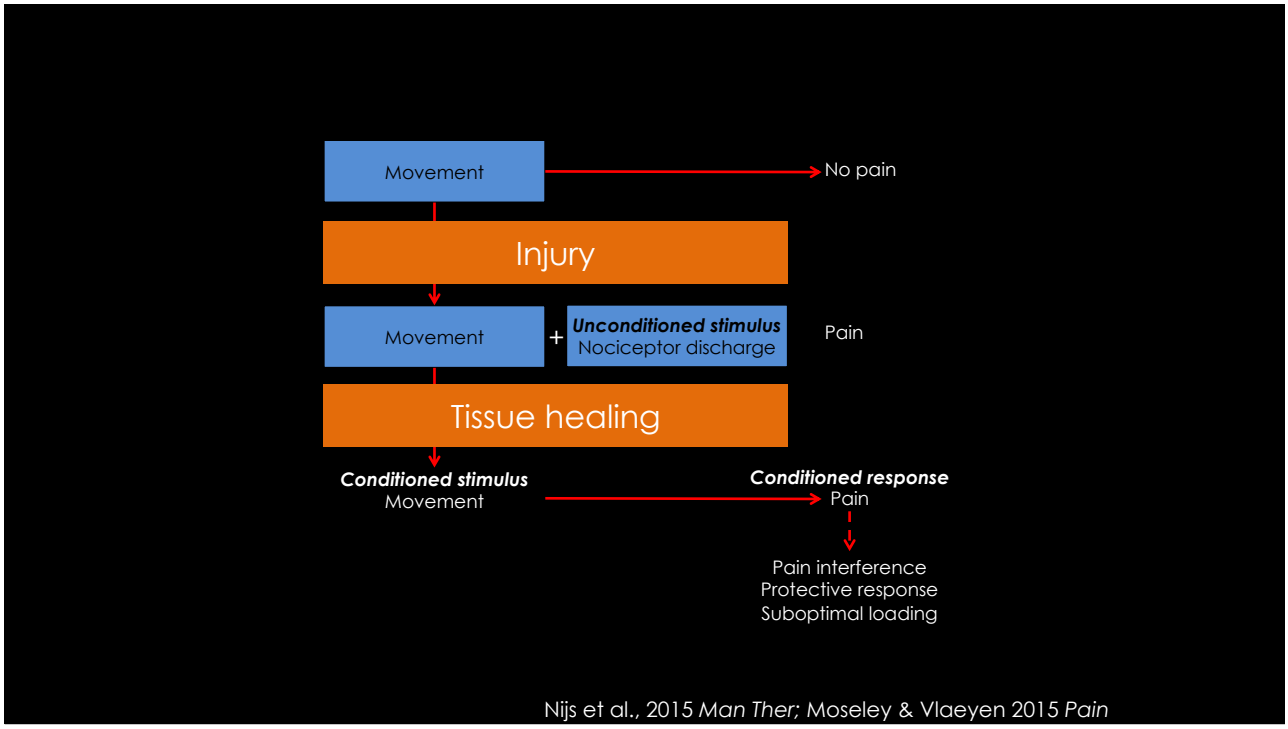


Hodges, Bennell, Wrigley, van den Hoorn (2015) *Man Ther 21:151-8*

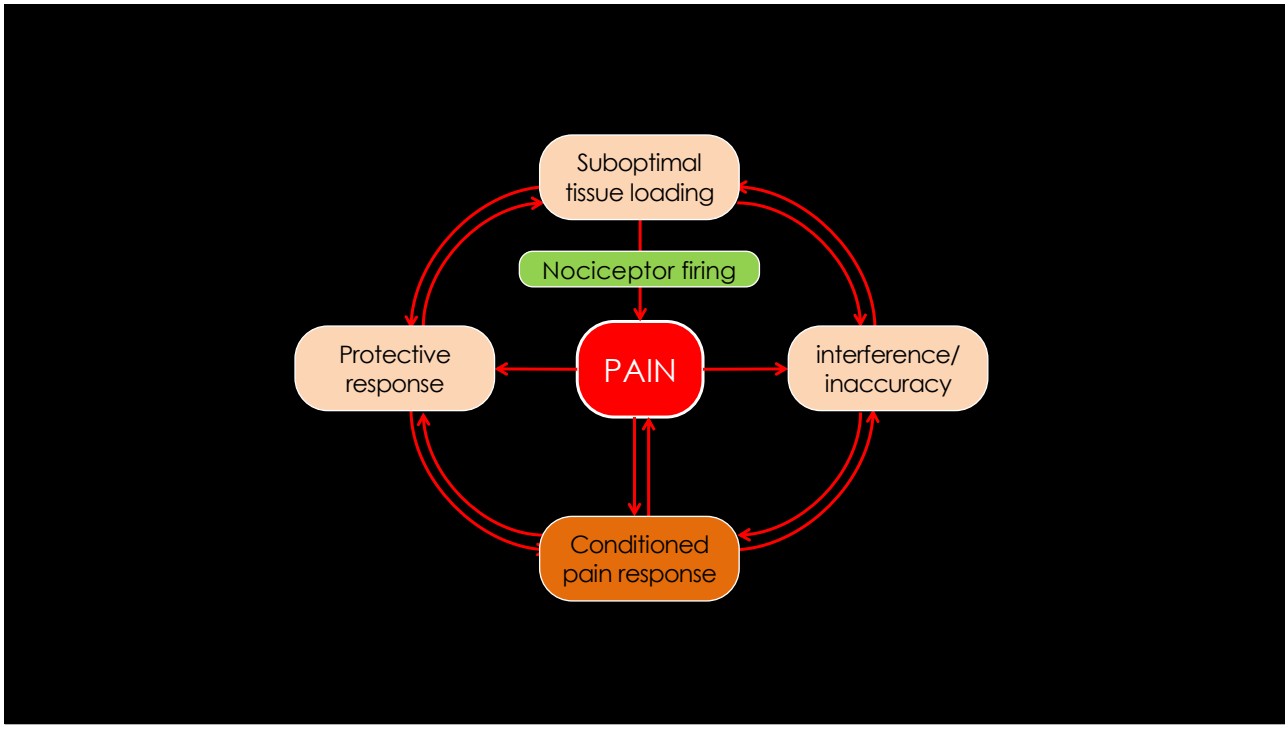
64


Suboptimal tissue loading
Nociceptor firing
Protective response
PAIN
interference/ inaccuracy
Conditioned pain response

65


Movement
No pain
Injury
Movement
+
Unconditioned stimulus
Nociceptor discharge
Pain
Tissue healing
Conditioned stimulus
Movement
Conditioned response
Pain
Pain interference
Protective response
Suboptimal loading
Nijs et al., 2015 Man Ther; Moseley & Vlaeyen 2015 Pain

66



67

How is pain related to joint dysfunction?

What is pain?

What mechanisms explain the relationship between pain, movement and joint dysfunction?

What are the clinical implications?

68

Implication for rehabilitation

- Identify the likely pain mechanism
- If suboptimal joint loading is relevant - Identify and address suboptimal loading
  - Restore joint function
  - Optimise motor control – movement, posture, muscle activation

69

How can we identify pain mechanisms in clinical practice?

Pain features

QST

Questionnaire

History

70

How can we identify pain mechanisms in clinical practice?

	Nociceptive pain
Pain features	Predictable Inc/dec with movement/posture Proportional Localised
QST	Normal sensitivity or local hyperalgesia
Questionnaire	Generally low psychosocial features – multiple questionnaires available to assess specific features
History	Relevant injury

Smart2012a, b, c Man Ther; Nijs et al. 2015 Pain Physician; Chimenti et al 2018 PhysTher; Shraim, Massé-Alarie & Hodges 2021 Pain.

71

How can we identify pain mechanisms in clinical practice?

	Nociplastic pain
Pain features	Unpredictable Inconsistent inc/dec Disproportionate Broad area/multiple area/changing area
QST	Hyperalgesia - ↓ pressure/ temp threshold – local & distant areas
Questionnaire	Central Sensitization Inventory Various Psychological Qs – Pain Catastrophizing Scale; Fear Avoidance, Pain Self Efficacy, etc
History	Mismatch between pain & history/mechanism

Smart2012a, b, c Man Ther; Nijs et al. 2015 Pain Physician; Chimenti et al 2018 PhysTher; Shraim, Massé-Alarie & Hodges 2021 Pain.

72

How can we identify pain mechanisms in clinical practice?

	Neuropathic pain
Pain features	"Electric" Dermatomal distribution/ribbon Pins & needles Numbness +/- positive neurodynamic signs
QST	Can be sensitized – Hyperalgesia - ↓ pressure/ temp threshold
Questionnaire	PainDETECT, LANSS (not specific to neuropathic)
History	History of nerve damage/ dysfunction

Smart2012a, b, c Man Ther; Nijs et al. 2015 Pain Physician; Chimenti et al 2018 PhysTher; Shraim, Massé-Alarie & Hodges 2021 Pain.

73

How can we identify pain mechanisms in clinical practice?

	Nociceptive pain	Neuropathic pain	Nociplastic pain
Pain features	Predictable Inc/dec with movement/posture Proportional Localised	"Electric" Dermatomal distribution/ribbon Pins & needles Numbness +/- positive neurodynamic signs	Unpredictable Inconsistent inc/dec Disproportionate Broad area/multiple area/changing area
QST	Normal sensitivity or local hyperalgesia	Can be sensitized – Hyperalgesia - ↓ pressure/ temp threshold	Hyperalgesia - ↓ pressure/ temp threshold – local & distant areas
Questionnaire	Generally low psychosocial features – multiple questionnaires available to assess specific features	PainDETECT, LANSS (not specific to neuropathic)	Central Sensitization Inventory Various Psychological Qs – Pain Catastrophizing Scale; Fear Avoidance, Pain Self Efficacy, etc
History	Relevant injury	History of nerve damage/ dysfunction	Mismatch between pain & history/mechanism

Smart2012a, b, c Man Ther; Nijs et al. 2015 Pain Physician; Chimenti et al 2018 PhysTher; Shraim, Massé-Alarie & Hodges 2021 Pain.

74


Australian Government
National Health and Medical Research Council
Australian Government
Australian Research Council
Arthritis AUSTRALIA
medibank
Queensland Government
Queensland Health
Physiotherapy Research Foundation
Vetenskapsrådet
Rebecca L. Cooper
Medical Research Foundation
RAMACIOTTI
SUPPORTING BIOMEDICAL RESEARCH
Lung Foundation Australia
when you can't breathe... nothing else matters
FNSNF
Swiss National Science Foundation
Program Grant Team
Prof Bill Vicenzino
Prof Kim Bennell
Prof David Hunter
Post-docs
Dr Natalie Collins
Dr Leanne Hall
Dr Greg James
Dr David Klyne
Dr Rachel Park
Dr Sauro Salomoni
Dr Jenny Setchell
Dr Ryan Stafford
Dr Wolbert van den Hoorn
Research staff
Markus Kiel
UQ Collaborators
A/Prof Sandy Brauer
Dr Kylie Tucker
Dr Andrew Claus
Dr Michelle Smith
Doctoral students (Current)
Rafeef Aljuraifani
Mansour Al Shami
Manuela Besomi
Catharina Bexander
Nathalia Costa
Peter Lawrenson
Liam Maclachlan
Muath Shraim
Louise Tier
Viana Vuvan
Prof Simon Gandevia, Dr Jane Butler, Dr Siobhan Schabrun, NeuRA, Sydney
Prof Chris Maher, Prof Chris Little, Sydney University
Prof Lorimer Moseley, Uni South Australia
Doctoral students (Past)
Kim Allison
Michael Bergin
Andrew Briggs
Angela Chang
Andrew Chapman
Andrew Claus
Sallie Cowan
Anna Dawson
Laura Diamond
Edith Elgueta-Cancino
Alessio Gallina
Jan Gildea
Janine Gray
Allison Greig
Leanne Hall
Luke Heales
Nick Karayannis
David Klyne
Jo Knox
Linda-Joy Lee
David Macdonald
Mary Massery
Rebecca Mellor
Nicola Mok
Lorimer Moseley
Rachel Park
Peter Poortvliet
Ruth Sapsford
Steven Saunders
Annina Schmid
Camille Shanahan
Michelle Smith
Natalie Spearing
Ryan Stafford
Ann Katrin Stensdotter
Narelle Stubbs
Henry Tsao
Donna Urquhart
Wolbert van den Hoorn
Peter Window
Richard Yang
Prof Thomas Graven-Nielsen, Centre for Sensorimotor Interaction, Aalborg, Denmark
Prof Alf Thorstensson, Karolinska Institutet, Stockholm, Sweden
Prof Jacek Cholewicki, Dr John Popovic, Michigan State University, USA
Dr Mary Massery, Chicago USA
Dr Paul Cordo, Dr Fay Horak, Oregon Health Sciences University, Portland, OR, USA
Prof Victor Gurfinkel, Russian Institute of Information Transmission Problems, Moscow, Russia
Prof Sten Holm & Dr Allison Kaigle, Sahlgrenska University Hospital, Göteborg, Sweden
Dr Anne Mannion, Schultes Klinik, Zurich Switzerland
Prof Jaap van Dieen, Vrije University, Amsterdam, Netherlands
A/Prof Paulo Ferreira, a/Prof Manuela Ferreira, Universidade Federal de Minas Gerais, Brazil
Prof. Shinn-Zong Lin, Tzu Chi University, Taiwan
Prof Lieven Dannees, Gent University, Belgium
Prof Simon Brumagne & Prof Sabine Verschueren, University of Leuven, Belgium
Prof Francois Hug, Nantes University, France
Prof Jayne Garland, Uni. British Columbia
Prof Laurent Bouyer, Uni Laval, Quebec, Canada
Dr Janine Gray, University of Cape Town, South Africa

75