2. Pain and pathology. Implications for clinicians

Understanding pain and nociception

The four key questions that patients are interested in are the following:

a. What is wrong with me?
b. How long will it take?
c. What can you do about it?
d. And what can I do about it?

It is critical that we understand the difference between pain and nociception. The first is a feeling produced when the brain decides to protect some body part, when the weight of evidence of the brain is that the body is under threat. Consequently, patients will experience pain as an output. Because of this, pain should be considered a neurotag. Thus, when a certain number of neurons in your brain light up at the same time, you feel pain. This feeling is comparable to the feeling of smelling fresh bread, when the same phenomenon of a lot of neurons in your brain lighting up at the same time occurs; this is how you recognize the smell of fresh bread.

Nociception, on the other hand, is an input, which consists of information coming from the tissue. It is your tendon giving you information, resulting in a powerful contributor to pain. However, it is not the only contributor; and here lies the difference between the two concepts.

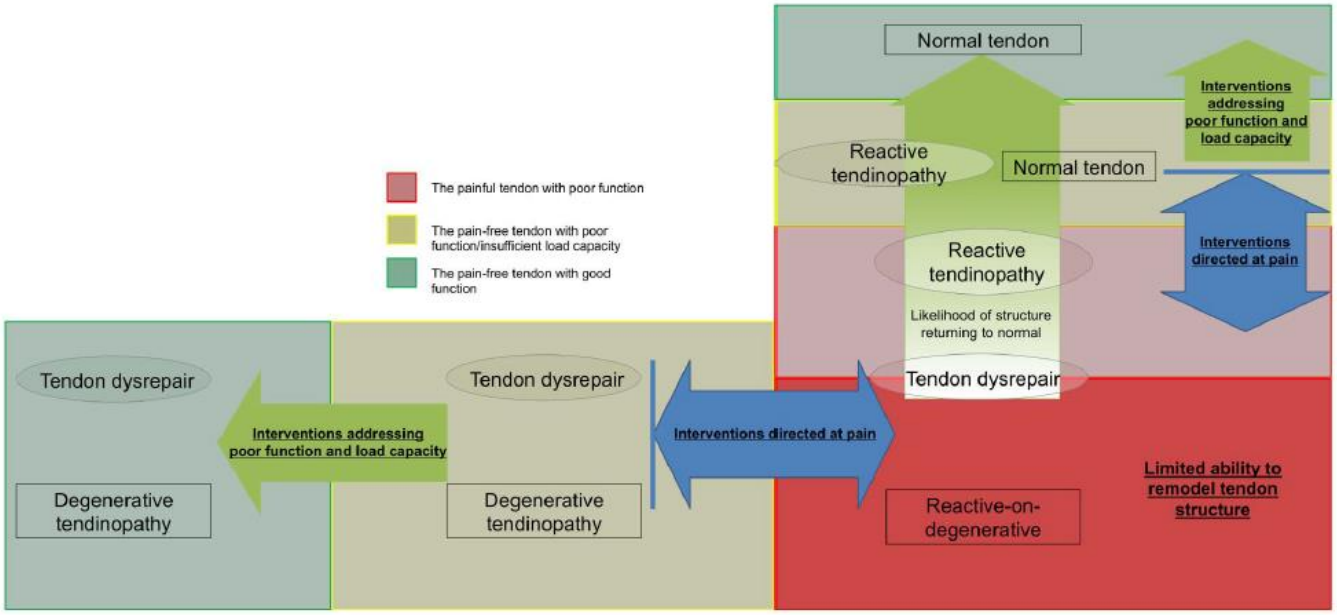
According to Cook and Purdam (2009) pain is the only clinical feature that the clinician seeks to change, and pain response to treatment is a key indicator of treatment success. If pain levels and response to load are considered together with the stage of pathology, treatment progression and outcomes may improve.

Loading a painful tendon perpetuates nociceptive stimuli, and it is plausible that secondary hyperalgesia in tendinopathy is a response to ongoing nociception. (Cook, Rio, Purdam, Docking, 2016).

According to Cook, et al. (2016), it is important to note that interventions directed solely at pain will not drive the tendon to a positive outcome, as they do not address dysfunction, such as motor inhibition, strength and power deficits, or tendon load capacity. Interventions that target structure may improve tendon structure and direct the tendon ‘upwards along the

continuum'. However, it will not address functional deficits (effect on pain is inconclusive) or load capacity and may leave the tendon vulnerable to reinjury (figure 1).

Figure 1: Schematic representation of how we may phenotype patients with tendinopathy in relation to the continuum and target treatments



(Cook, et al., 2016. p. 4)

The biopsychosocial model and tendinopathy

It is important that we understand how terms used by practitioners affect patients' perceptions. Every time language is used, there is an updating of what people think is wrong, and what they think they need to do to heal their tendon. In 2017, a systematic review showed that if you reported an injury as a *broken bone*, 56% of people thought they needed a cast, whereas if you had the same injury, but reported it as a *crack in the bone*, only 13% thought they needed a cast. This review turns out to be important when we consider the terms we use to talk about tendons-related matters, such as tendonitis and tendon tear, since they have implications for the biopsychosocial model.

The biopsychosocial model, on the one hand, is composed of the biology -the tissue health and the overall homeostasis of the body, the muscles, the bones and the whole system-. On the other hand, the psychosocial aspect corresponds to the person's context. When it comes

to our biology, there are a lot of potential or suggested drivers of nociception. But it is important that we consider them on the basis of the context of the clinical presentation. So, just to remind you, tendon pain is very well localized and very well related to load, and this relationship implies that it is a predominantly nociceptive driven condition, regardless of for how long people feel pain. It has a very on-off pattern associated with load. In fact, location of the pain can even determine what area of the tendon is suffering the injury (figure 2).

Figure 2: Localization of Achilles tendinopathy A) midportion and B) insertional

(Silbernagel & Crossley, 2015)

And again, we do not see a lot of spreading of pain or pain at rest. So, if we consider what being a potential nociceptive driver is, we actually know more about what does not drive nociception. Strange as it may sound, I am going to explain how this is still helpful. First, we said that tendon pain is completely unrelated to structure. You can have completely varied sonographic findings. Asymptomatic pathology is incredibly common and it seems to be more a marker of load rather than be linked with tendon pain. In fact, there is not a single musculoskeletal condition where pain and pathology marry up at all, and tendon pain is not different. We cannot image people and determine that it is the tendon that is really driving their symptoms. There is also no classic inflammatory process and, therefore, we should not

be using terms like 'tendinitis'. Considering that basic inflammatory response therapies are inflammatory cytokines, they turn out to be important in communication. The innovation patterns do not change with pathology. So, we might see an ingrowth of nerves and vessels into the tendon as a marker of pathology in stage pathology. But the nerves appear to be associated with the vessels. So they are associated with vessel diameter as opposed to being sensory or nociceptive, and that is why we see that neovascularization or these new vessels are inconsistently linked with pain. You can have a tendon that lights up like a Christmas tree and the person can be completely pain-free. We have a number of other candidates in tendon pain, including, you know, the cells, substances produced by the cells. In ummary, we actually do not know what drives nociception in tendinopathy.

In relation to this, Silbernagel and Crossley (2015) stated that Achilles tendinopathy is a painful overuse injury that affects an athlete's ability to be physically active. During the early stages of the injury, the patient might be able to continue with regular activities and sports. However, as the injury progresses, the patient's ability to be physically active is progressively impaired. Repetitive overloading of the Achilles tendon and training errors, such as rapidly increasing training intensity or duration, are reported to be contributing factors in 60-80% of those who develop Achilles tendinopathy.

This is a clear example of the following idea: Even in a nociceptive condition, it is never one or the other that we are talking about, regarding the tendon or the person. This can be described as a changing clump of cells. Therefore, it is important to teach patients that they are bioplastic: every single cell in their body is capable of changing until the day it dies. Thus, it is our role to modify the clumps of cells for the better. A good way to do that is to meet the patients and explain their imaging; you cannot say to people 'don't worry about your imaging' because they're very concerned about it, and often they will have a report that describes a lot of different changes. It is about understanding and explaining what those changes are.

According to Cook and Purdam:

> Pain can occur at any point in this pathological model, thus supporting the wellknown dissociation between pain and pathology in tendinopathy. Even tendons that appear normal on imaging can be painful. Conversely, two thirds of tendons degenerative enough to rupture have been reported to be pain-free before rupture. The source of pain has been associated with neurovascular ingrowth, seen in this model at the late tendon dysrepair/ degenerative phase. However, the fact that tendons can be painful or pain-free anywhere in this model suggests another or supplementary cause of

> pain that has so far proved elusive. (Cook & Purdam, 2009 https://bjsm.bmj.com/content/bjsports/43/6/409.full.pdf)

We know that pathology and pain are not equal, that we can have a variety of pathologies and no pain. In fact, pain can be load-related change. We also know that our bodies and brains are clever, so, what we see in tendons are load-appropriate changes. Therefore, when a patient visits the doctor with their imaging and a report of an increase in anterior-posterior diameter, we can reassure them and tell them that the tendon has adapted. We can explain them that they have this area of load-related change, where the tendon has increased in size, it is increasing cross-sectional area, and that they have a load-bearing tendon. We should explain patients that they have this amazing adaptive tendon. This is an example of taking biology while considering the context.

Language can promotes self-efficacy, address fee and promote an active recovery. There is also some biology in reducing catastrophizing. As opposed to those patients who feel fear after their imaging, Rather than making people fearful for their imaging, we should encourage them to make progress. Real relationship in the immune system is not mediated by negative beliefs.

Language and terminology

"Capacity is a helpful term in clinical practice to indicate to clients that they (and more importantly their musculoskeletal tissues) are either able or unable to complete a task or participate in physical activity." (Cook & Docking, 2015 https://doi.org/10.1136/bjsports-2015-094849)

> Beliefs about pain and pathology may influence the development and management of unresponsive symptoms. Some athletes may have been told that "tears" and "degeneration" have caused permanent tendon "weakening," increasing the risk of rupture. Patellar tendon rupture (in the absence of systemic disease) in sport is rare. Some athletes may develop fear-avoidance behavior, which has been associated with poorer functional outcomes in individuals with lower-limb tendinopathy. Education about pain and realistic time frames for rehabilitation are important. This includes education regarding the potential link between psychosocial factors and pain. Athletes need to be aware that pain is not necessarily equal to harm, and some pain during rehabilitation is acceptable. It is important to educate

> patients regarding the concept of load tolerance as defined in this article, so that they are eventually able to self-manage based on symptom response to load. (Malliaras et al. 2018 https://doi.org/10.2519/jospt.2015.5987)

I would like to demonstrate how what we know about nociception can actually influence the language we use with patients. The first thing I will present here is the term 'tendon tear'. We have a very poor ability or low accuracy to diagnose tears on imaging. In fact, there are a number of studies that show that when some patients undergo surgery, we are no better than flipping a coin or a 50% chance to tell whether or not the patient has a true tear or tendinopathy. We also know that these tens or the area of letting go is happening in a pair in an area of non-load-bearing tendon, and so it does not change our clinical practice. However, the clinical implication is huge. If you say to anyone that they have a tear in your tendon, they will probably think that they need to rest or be immobilized. They will certainly not expect to be given exercises by their physiotherapist. This has huge implications on what people think they need to do to get better. The second term that I would like to talk about is 'tendonitis'. We do not let anyone use the term 'tendinitis'; only specialists, use the term tendinitis. The reason is that people think that there is something inflamed, so they will probably not want to load it, but specialists know the best evidence for tendons is actually load. If people continue using a term that implies inflammation, again, they will think of passive treatments where they think they need rest or ice or antiinflammatory medications, and we know that such treatments are ineffective. In fact, we know now from some basic science studies that unloading a tendon can be as detrimental as overload. So, it is highly important that our language reflects our best practice. And best practice for tendons is exercise.

The following terms are those that we need to truly understand and know when they are appropriate to be used. They are terms that are commonly used by people in general, by the media and even by our medical colleagues. The first term is "tendinopathy", and it refers to the clinical presentation of pain and dysfunction. It is the most appropriate term to use because we actually know very little about the nurse, deceptive driver or the relevant historic pathology. What we actually have is the patient in front of us with pain and a dysfunction. Besides, in a recent international consensus this is the term that was voted for to refer to this clinical presentation. The second term relates to pathology: 'Tendon pathology' means that it is changes that we can actually see on imaging. As you will learn after this course, that does not correlate with pain. There are lots of examples of a symptomatic pathology, which probably relates to a load-related change. In the case of young basketball athletes, for instance, if they have played a lot during their development, they are likely to present changes in their patella tendon, but very few of them will actually develop patellar tendinopathy. 'Paratendonitis' is also a pathological term. It is linked to changes, which we

see on imaging, and has no clinical importance for us. The paratendonitis really refers to pathology of the shapes. As you may remember from our four types of tendon load, we can irritate the sheath or the surrounding structures of the tendon when we are doing low-load repetition. An example of this could be the case of the Achilles tendon when we swim: we kick our feet a lot and we get much shear and friction load, thus irritating the sheath or the power attendant of the Achilles over the tendon itself, but we are not using the Achilles tendon like a spring. Another good example is cycling: we might go through a repeated plan of flexion and Dorsiflexion, but we are not using our Achilles tendon like a spring. The importance of all this when we get to management is that we actually manage these people differently. This is where using specific terminology is important: tendinopathy has different provocative loads and there is a different management strategy to paratendinitis, which has shear and friction, provocative loads and a different management strategy. Finally, the last word is 'banned'. 'Tendinitis' is a term that has been used commonly in the past. The issue retention, tendonitis as the term is, it really implies an underpinning of an inflammatory process and that is clinically unhelpful. There is lot of debate in basic science about the process of tendon pathology. However, the term 'tendinitis' is clinically unhelpful, because people think they need a passive approach to control their pain. And that is not true.

Adaptation and bioplasticity – *kinetic chain.*

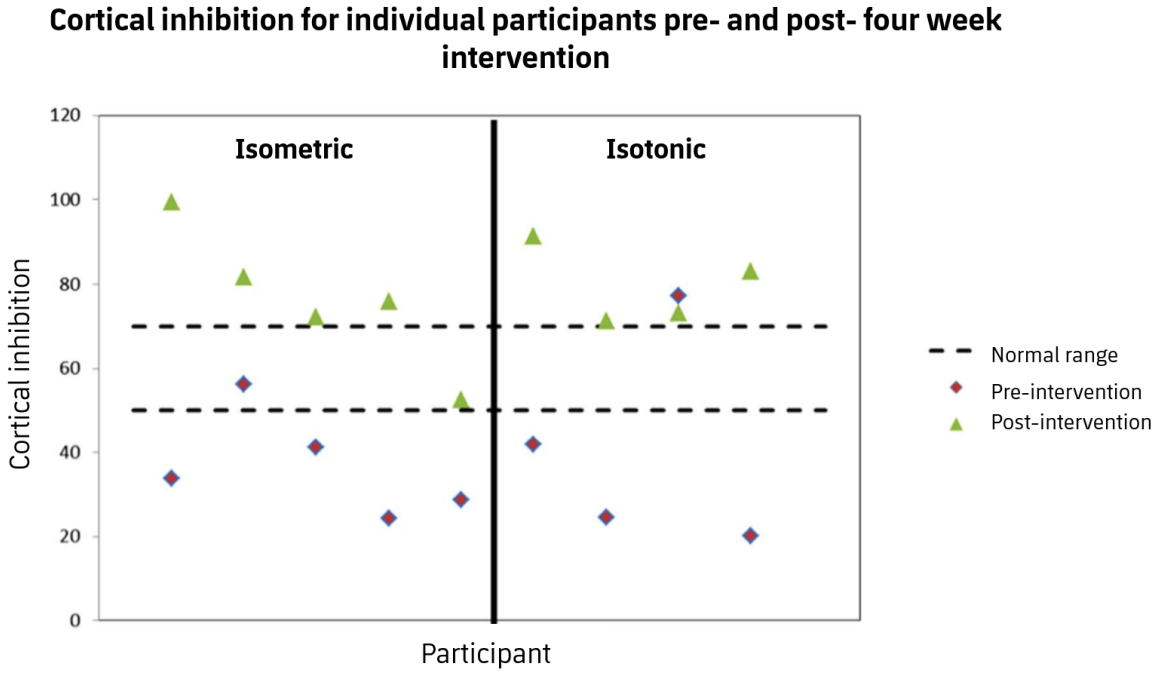
What we see with tendon pain are a number of adaptations and also maladaptation, particularly as people have had tendon pain for a long time and it can be quite persistent. At a tendon level, we can see pathology in the tendon. We appear to have this amazing adaptation that occurs in a thickening of the tendon as we move our way out. We see a reduction in cross-sectional area, but also our capacity or strength, endurance or mechanical properties of our muscle. Likewise, we also see our maladaptation in the remaining kinetic chain. Clinically, this means that we have a poorer strength in the associated muscle tendon unit, but we also have a poorer strength often in the capacity of the whole kinetic chain, its ability to function as a spring. This can lead to other injuries that can mean an overloading or a change in people's performance. And in fact, clinically, what people say is that tendon pain takes away what they are good at, so it takes away their spring, same thing happens to muscle capacity.

Adaptation and bioplasticity of the whole system – *do not forget about the brain and the spinal cord!*

> Jumping and landing mechanics will be influenced by motor control changes that include both peripheral and central contributions to lower limb activation. People with patellar tendon abnormality (pathologically observable on ultrasound) have demonstrated landing patterns different from those of controls and importantly, have less variability in movement than healthy controls. Movement variability is thought to minimize load accumulation in a specific region and reduced variability may be observed in, for example, reduced coefficient of variation in knee joint angles during a landing task. The invariable motor pattern implies that corticospinal control is altered in some way and may be due to protective strategies underpinned by evaluative processes relating to the consequence or meaning of the motor task. In the case of patellar tendon abnormality, invariable motor pattern may reflect a strategy to avoid pain during jumping (consequence) as well as the competing desire for optimal performance (meaning). (Rio, et al. 2016 https://doi.org/10.1136/bjsports-2015-095215)

Do not forget about the brain and spinal cord. Tendon pain is localized and load-related, but it is persistent. We have to remember that the brain and the spinal cord are aware that there is this persistent pain condition. This means that there is evidence from motor and sensory changes in a number of tendons, including the lateral elbow and the rotator cuff. And our research is predominantly in the lower limb. We were interested in the motor cortex or the drive or the control of the quadriceps muscle in people with Patellar tendinopathy, and in order to investigate these, we used noninvasive brain stimulation, called 'transcranial magnetic stimulation'. This is where you set a motor. You place a magnetic coil over the motor cortex and in the area that is responsible for leg extension; then you put surface electrodes on the quadriceps muscle, on the femoris rectus muscle, so you can record the twitch to stimulate the motor cortex in the region. And this signal travels down the cortex spinal tract and it is recorded as a twitch in the target muscle. Now, with this equipment, we can look at both the cortical spinal excitability, and the inhibition of this system. Metaphorically, the cortical spinal excitability and inhibition are best thought of as the accelerator and the brake of a car.

Figure 3: Individual cortical inhibition responses to tendon neuroplastic training by muscle contraction type



(Rio, et al., 2016 p.6)

Every time we make a movement, there is a balance between that excitability and that inhibition. And what we see in people with patellar tendinopathy is a profound change in their motor drive, or they are breaking their accelerator. In this research, we first looked at their accelerated cortical spinal excitability and we found that people with patella tendinopathy had no ability to modulate their excitability; they had an increased excitability. In a different study, we looked at their inhibition, and it was like their brake was stuck on their brain and the brain was saying 'don't use your quadriceps'. It was as if these people had one foot stuck on the accelerator and one foot stuck on the brake. Interestingly, this motor profile was quite different in people that had focal so inferior pole localized pain compared to people with diffused knee pain. People with diffuse pain had a similar cortico spinal excitability to the controls, which makes us believe that the brain appears to care about the nociceptive driver that had quite a different motor profile.

Now I am going to talk about what strategies we can use to change our broken accelerator. We found out that is quite a difference in the motor profile in the brain, I was interested in what techniques we might have to change the motor drive, and so

I looked through the neuroscience literature. We know from lots of tendon and muscle research that load is a really effective way of changing pain and our most effective strategy for changing strength, capacity and function. But we were interested in what strategies we might have to change the motor drive, so as to hit the bull's eye of attacking all three of our deficits, andin order to do so, we need to understand the neuroscience literature. The first thing to do is a visual motor skill, which consists of copying a movement displayed on a screen. This is effective to train tempo, time and attention. We see a ton of plasticity in your brain. You obtain changes in both your braking and your accelerator. If you do a self-paced arm strength exercise in the gym and you start complaining about the movement, even though you can be very focused, you can also get easily distracted by listening to music or thinking about what you are having for dinner. You will get stronger during the exercises because you will get changes in your muscle and some of the substrates, but you actually do not change your braking and your accelerator. On the other hand, if we pair your gym exercise with some sort of external pacing that you are listening to at preempting, like a metronome, we can guarantee not only good principles of strength training, but also your time and your attention. We can also promote plasticity and change your braking and accelerator because we have many neurons between the different parts of our brain that really promote that change. This is a way of applying some neuroscience to strength training and tendon principles.

It is important to remember that repetitive, unskilled movements do not result in changes in the motor cortex and in tendinopathy. We appear to have profound changes in the motor cortex. For this reason, it you can put into context the different interventions for tendinopathy, including adjuncts. For example, if we are going to take a passive tendon intervention, it is not going to have any effect on our strength nor on our motor drive changes. Therefore, it is unsurprising that there is no ´magic bulleť for tendinopathy. We have actually failed to address the key things that people present us with. This also creates an ongoing narrative to focus on the pathology.

What if we perform some sort of strength-based intervention but fail to consider the motor cortex? This is probably our current clinical challenge: tendons that are challenging to treat. They can be persistent. You can have a high recurrence, or you can see the problem from the other side and it can potentially come down to this motor cortex change. And what if we ignore any load-based principles and we just focus on the brain? The problem here is that we do not give people the capacity to perform their task. We know that people will need to be incredibly strong and withstand many times the ground reaction force to do things like jumping and running. If we only worry about the motor cortex and do not respect biology enough, people will end up having an inability to perform the task. In sum, the ideal scenario is that our interventions be load-based and progressive, but that we also start to

consider the complexity and the motor cortex changes and also some of the strategies that we have to optimize all levels of dysfunction.

> Current rehabilitation may not adequately address motor control issues as self-paced strength training (the mainstay of the treatment) does not alter corticospinal drive to the muscle—this may contribute to recalcitrance and recurrence of tendinopathy. Tendon neuroplastic training proposes a concept of strength-based loading that is an important stimulus for tendon and muscle, but with strategies known to optimize neuroplasticity of the motor cortex and drive to the muscle. It needs to be tested in a wide range of anatomical locations as it is unclear whether findings for the patellar tendon are applicable to all tendinopathies. (Rio, et al. 2016 https://doi.org/10.1136/bjsports-2015-095215)

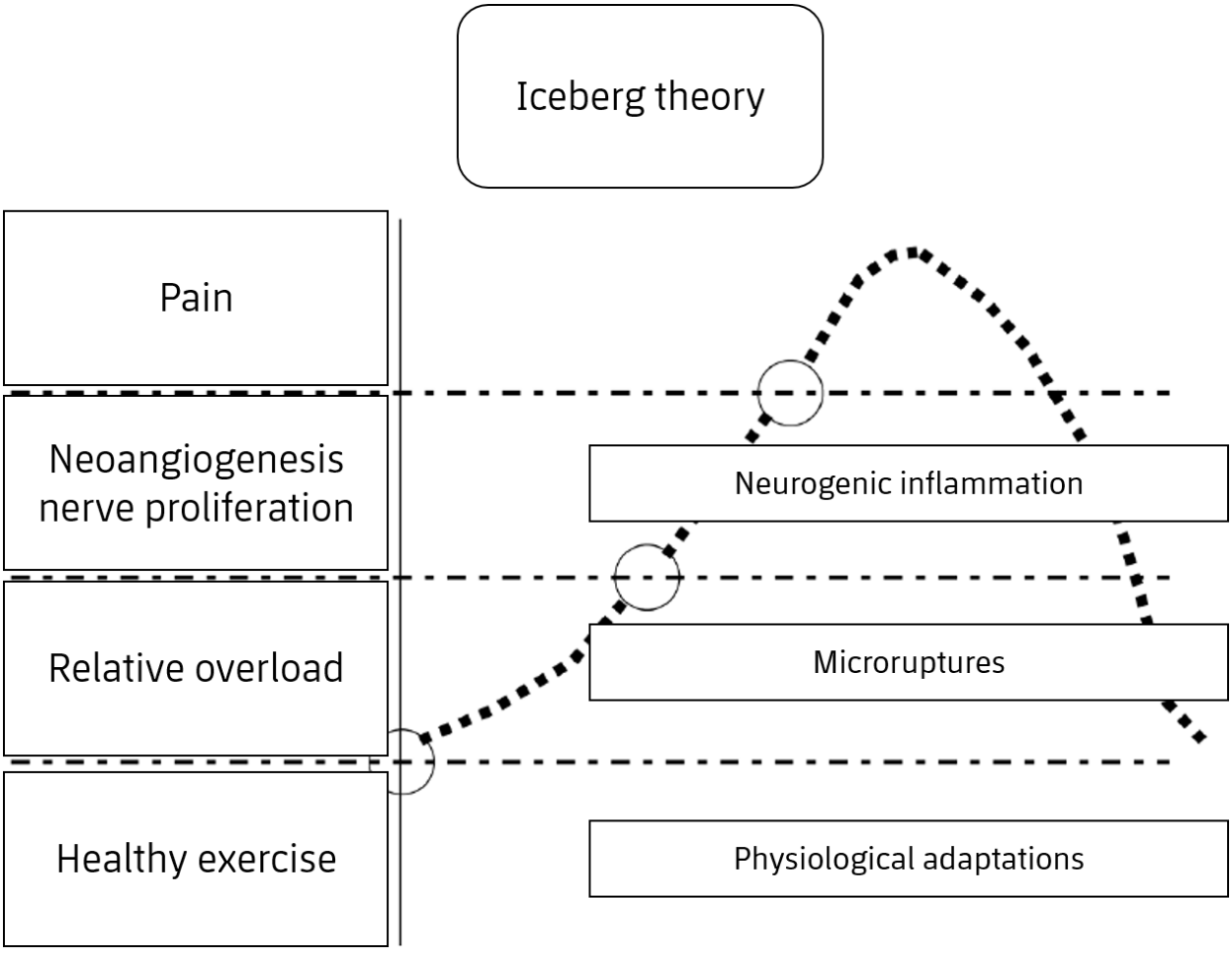
Putting it all together – *relating loads, structure, function and pain*

We have talked a lot about tendon pathology and about tendon pain. But one of the things we tend to forget about is how both affect function and how load is interrelated to that. And we have talked about structure not being very important because the pathology actually does not predict whether you are going to have symptoms or not. And changing pathology is not an important part of reducing symptoms. Pain is much more complex. We have also talked about how pain does not relate to imaging, to structure or to many other factors that we actually look at clinically. But what we do know by examining people with tendon pain is that function is the thing that seems to be the most important. Function is one of the key things that we can assess and we can change; it seems to be affected mostly by pain. Structure is very complex; as we said when we talked about tendon pathology, it is not reversible once it gets to the degenerative stage. We will leave that aside for a moment. But as clinicians, what we do is to intervene from a function perspective. When clinicians treat somebody with tendon pain, what they do is to restore the tendon completely for free function. In the case of athletes, what we know is that if we can return that person to full function and performance, sometimes the pain does not reduce to zero. We know that this structure does not change, but the ability to perform repeatedly at the level that the patient wants is the key thing that we have as objective when we treat these patients. The important thing is to understand how tendon pain is quite different from other pains. There is no direct relationship between the load placed on a structure and the pain. That is, if you have low back pain, it might hurt when you are sitting or when you

bend or stand up, but sometimes such movements do not produce any pain. The difference with tendon pain is that it hurts every single time you load it. So every time you step off a stair, every time you change direction, every time you change to run, your tendon will give you pain. Such direct relationship between load and pain is the thing that seems to make tendon pain quite particular and it absolutely and utterly destroys function. We see athletes who have suffered many injuries. They have fractured their fingers, sprained their ankle, suffered from back pain or hamstring strains, etcetera. What they report is that they can manage the problem; they can tape their fingers or take their ankles, they say they can control the pain with a bit of stretching to play with their low back pain. But the tendon pain impedes me training and playing fully because every time they try to load the tendon, they "get into trouble". Tendon pain seems to restrict capacity for function very quickly. When we assess people, make the key clinical questions, and interpret the examination findings, we observe profound changes in function. Even in athletes who are able to try and play fully at an elite level, when their ability to load their tendon is broken down, it is always diminished and often profoundly diminished.

This is why we say that this direct relationship between tendon load and tendon pain is utterly destructive to function. One of the mantras that we usually say is that an athlete with poor function will always have pain and an athlete with good function is protected from pain. Therefore, our role as clinicians is to improve the capacity of the tendon, the muscle, the kinetic chain in the brain, that is, to restore function. This usually allows us to either relieve or control pain or pain to the level that the athlete can still perform. So one of the key things that we say to our patients is if you. This is. Let me do that again. One of the key things that can make this obvious to patients is this idea that if you hop or jump very poorly and you have any sort of passive intervention, it never changes your ability to hop or jump. So it does not really matter what interventions you have from an injection; a passive perspective will always end up in the same poor hopping and jumping capacity. The only thing that will change that capacity to hop and jump, that will change the direction, and that will allow you to do all the athletic activities that you want is an active intervention. For those patients who have had a lot of passive interventions, all this can make much sense. Through an active intervention, clinicians are interested not only in restoring the functions, but also in controlling the patient's pain.

Figure 4: The Iceberg theory



(Abate, et al., 2009. p. 10)

Clinical examples

We see many patients that had previous treatments that were unsuccessful, and there are many reasons why these treatments are unsuccessful. The first example I want to give you is that of a gentleman who is a trial runner. Trial runners run 150, sometimes 160 kilometers for an event, and what they need from their tendon, muscle, kinetic chain and brain is an amazing amount of strength and endurance. This trial runner previously had treatment for his Achilles tendon. Although he had successfully run ultramarathons or trial runs for many years without any problem, he had had five or six treatments to modify his running technique. However, he had never had any management of his strength and capacity within his tendon, within his muscle and his kinetic chain. So, when we first examined him, he had a really appalling function.

That is, his ability to hop and even jump was restricted by his tendon pain, strength, endurance and power within his limbs. Therefore, he was placed on a long term program. We could not change this gentleman very quickly because he had suffered tendon pain for about nine months and he had lost function considerably. So he spent 12 weeks exercising muscular strength, the calf muscle, the quads hamstrings glute muscle. Then, we progressively added hopping, jumping energy storage type loads to his tendon and to his kinetic chain, and then, gradually, we reintroduced running as an intervention, or is it as a treatment strategy? He must always go on with his strength program. Our next step for him is to introduce some hills and some uneven surfaces, because both of these can influence the load on the tendon. And then, of course, the next step is to increase the distance he runs. If he intends to run a 100 km race in October next year, I think during the period of this year and the following , we have to restore the capacity of that tendon muscle kinetic chain to actually take massive loads repeatedly over a 24 hour period.

The second clinical example I want to give you has to do with the importance of language. One of the patients that we treated some number of years ago suffered from Achilles tendon pain. She was wearing a cam walker boots and she had consulted a number of specialists, had undergone imaging of her Achilles tendon, weekly, which gave us a pile of imaging reports. She was so fearful of the changes to her tendon that she was incredibly worried about loading it. The interventions that she had been offered were passive: a number of blood injections and immobilization. By the time we saw her, she was very afraid of the situation in her tendons, and very worried about her capacity to load. This is a clear example to understand how much we need to educate patients prior to even start a lighting program.

References

Abate, M., Silbernagel, K. G., Siljeholm, C., Di Iorio, A., De Amicis, D., Salini, V., ... Paganelli, R. (2009). Pathogenesis of tendinopathies: inflammation or degeneration? *Arthritis Research & Therapy*, *11*(3), 235. Retrieved from https://doi.org/10.1186/ar2723

Alfredson, H., Pietilä, T., Jonsson, P., & Lorentzon, R. (1998). Heavy-load eccentric calf muscle training for the treatment of chronic achilles tendinosis. *American Journal of Sports Medicine*. Retrieved from https://doi.org/10.1177/03635465980260030301

Bayona, N. A., Bitensky, J., Salter, K., & Teasell, R. (2005). The Role of Task-Specific Training in Rehabilitation Therapies. *Topics in Stroke Rehabilitation*. Retrieved from https://doi.org/10.1310/bqm5-6ygb-mvj5-wvcr

Beyer, R., Kongsgaard, M., Hougs Kjær, B., Øhlenschlæger, T., Kjær, M., & Magnusson, S. P. (2015). Heavy slow resistance versus eccentric training as treatment for achilles tendinopathy: A randomized controlled trial. *American Journal of Sports Medicine*. Retrieved from https://doi.org/10.1177/0363546515584760

Cook, J. L., & Khan, K. M. (2001). What is the most appropriate treatment for patellar tendinopathy? *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsm.35.5.291

Cook, J. L., & Purdam, C. (2012). Is compressive load a factor in the development of tendinopathy? *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsports-2011-090414

Cook JL & Purdam C. (2009). Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. *British Journal of Sports Medicine* 2009;43:409-416. Retrieved from https://bjsm.bmj.com/content/bjsports/43/6/409.full.pdf

Cook, J. L., Rio, E., Purdam, C. R., & Docking, S. I. (2016). Revisiting the continuum model of tendon pathology: What is its merit in clinical practice and research? *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsports-2015-095422

Cook, J. L., Khan, K. M., Kiss, Z. S., & Griffiths, L. (2000). Patellar tendinopathy in junior basketball players: A controlled clinical and ultrasonographic study of 268 patellar

tendons in players aged 14-18 years. *Scandinavian Journal of Medicine and Science in Sports*. Retrieved from https://doi.org/10.1034/j.1600-0838.2000.010004216.x

Cook, J., & Docking, S. (2015). "Rehabilitation will increase the 'capacity' of your ...insert musculoskeletal tissue here...." Defining 'tissue capacity': a core concept for clinicians. *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsports-2015-094849

Coombes, B. K., Bisset, L., & Vicenzino, B. (2012). Elbow flexor and extensor muscle weakness in lateral epicondylalgia. *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsm.2011.083949

Edwards, S., Steele, J. R., Cook, J. L., Purdam, C. R., McGhee, D. E., & Munro, B. J. (2012). Characterizing patellar tendon loading during the landing phases of a stop-jump task. *Scandinavian Journal of Medicine and Science in Sports*. Retrieved from https://doi.org/10.1111/j.1600-0838.2010.01119.x

Edwards, S., Steele, J. R., Cook, J. L., Purdam, C. R., McGhee, D. E., & Munro, B. J. (2012). Characterizing patellar tendon loading during the landing phases of a stop-jump task. *Scandinavian Journal of Medicine and Science in Sports*. Retrieved from https://doi.org/10.1111/j.1600-0838.2010.01119.x

Esculier, J. F., Bouyer, L. J., Dubois, B., Leblond, J., Brisson, M., Chau, L., & Roy, J. S. (2018). Predictors of clinical success in runners with patellofemoral pain: Secondary analyses of a randomized clinical trial. *Journal of Science and Medicine in Sport*. Retrieved from https://doi.org/10.1016/j.jsams.2018.01.006

Fallon, K., Purdam, C., Cook, J., & Lovell, G. (2008). A "polypill" for acute tendon pain in athletes with tendinopathy? *Journal of Science and Medicine in Sport*. Retrieved from https://doi.org/10.1016/j.jsams.2007.09.002

Gaida, J. E., Cook, J. L., Bass, S. L., Austen, S., & Kiss, Z. S. (2004). Are unilateral and bilateral patellar tendinopathy distinguished by differences in anthropometry, body composition, or muscle strength in elite female basketball players? *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsm.2003.006015

Ganderton, C., Pizzari, T., Cook, J., & Semciw, A. (2017). Gluteus Minimus and Gluteus Medius Muscle Activity During Common Rehabilitation Exercises in Healthy Postmenopausal Women. *Journal of Orthopaedic & Sports Physical Therapy*, *47*(12), 914–922. Retrieved from https://doi.org/10.2519/jospt.2017.7229

Grävare Silbernagel, K., & Crossley, K. M. (2015). A Proposed Return-to-Sport Program for Patients With Midportion Achilles Tendinopathy: Rationale and Implementation. *Journal of Orthopaedic & Sports Physical Therapy*. Retrieved from https://doi.org/10.2519/jospt.2015.5885

Hagglund, M., Walden, M., Zwerver, J., & Ekstrand, J. (2011). Epidemiology of patellar tendon injury in elite male soccer players. *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsm.2011.084038.41

Hortobágyi, T., Lambert, N. J., & Hill, J. P. (1997). Greater cross education following training with muscle lengthening than shortening. *Medicine and Science in Sports and Exercise*. Retrieved from https://doi.org/10.1097/00005768-199701000-00015

Jack, K., McLean, S. M., Moffett, J. K., & Gardiner, E. (2010). Barriers to treatment adherence in physiotherapy outpatient clinics: a systematic review. *Manual Therapy*. Retrieved from https://doi.org/10.1016/j.math.2009.12.004

Janssen, I., Steele, J., Munro, B., & Brown, N. (2014). Jump height is the critical factor affecting between-sex differences in patellar tendon loading during landing in Volleyball. *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsports-2014-093494.138

Kidgell, D. J., Frazer, A. K., Rantalainen, T., Ruotsalainen, I., Ahtiainen, J., Avela, J., & Howatson, G. (2015). Increased cross-education of muscle strength and reduced corticospinal inhibition following eccentric strength training. *Neuroscience*. Retrieved from https://doi.org/10.1016/j.neuroscience.2015.05.057

Kongsgaard, M., Qvortrup, K., Larsen, J., Aagaard, P., Doessing, S., Hansen, P., … Magnusson, S. P. (2010). Fibril Morphology and Tendon Mechanical Properties in Patellar Tendinopathy. *American Journal of Sports Medicine*. Retrieved from https://doi.org/10.1177/0363546509350915

Lee, S. E. K., Lira, C. A. B. de, Nouailhetas, V. L. A., Vancini, R. L., & Andrade, M. S. (2018). Do isometric, isotonic and/or isokinetic strength trainings produce different strength outcomes? *Journal of Bodywork and Movement Therapies*, *22*(2), 430–437. Retrieved from https://doi.org/10.1016/j.jbmt.2017.08.001

Leung, K. S., Chong, W. S., Chow, D. H. K., Zhang, P., Cheung, W. H., Wong, M. W. N., & Qin, L. (2015). A Comparative Study on the Biomechanical and Histological Properties of Bone-to-Bone, Bone-to-Tendon, and Tendon-to-Tendon Healing: An Achilles Tendon-

Calcaneus Model in Goats. *American Journal of Sports Medicine*. Retrieved from https://doi.org/10.1177/0363546515576904

Lian, Ø. B., Engebretsen, L., & Bahr, R. (2005). Prevalence of jumper's knee among elite athletes from different sports: A cross-sectional study. *American Journal of Sports Medicine*. Retrieved from https://doi.org/10.1177/0363546504270454

Lian, Ø., Refsnes, P. E., Engebretsen, L., & Bahr, R. (2003). Performance characteristics of volleyball players with patellar tendinopathy. *American Journal of Sports Medicine*. Retrieved from https://doi.org/10.1177/0363546503030310031401

Malliaras, P., Cook, J., Purdam, C., & Rio, E. (2015). Patellar Tendinopathy: Clinical Diagnosis, Load Management, and Advice for Challenging Case Presentations. *Journal of Orthopaedic & Sports Physical Therapy*. Retrieved from https://doi.org/10.2519/jospt.2015.5987

Ngomo, S., Mercier, C., Bouyer, L. J., Savoie, A., & Roy, J. S. (2015). Alterations in central motor representation increase over time in individuals with rotator cuff tendinopathy. *Clinical Neurophysiology*. Retrieved from https://doi.org/10.1016/j.clinph.2014.05.035

Nickel, B., Barratt, A., Copp, T., Moynihan, R., & McCaffery, K. (2017). Words do matter: A systematic review on how different terminology for the same condition influences management preferences. *BMJ Open*. Retrieved from https://doi.org/10.1136/bmjopen-2016-014129

Pelletier-Galarneau, M., Martineau, P., Gaudreault, M., & Pham, X. (2015). Review of running injuries of the foot and ankle: clinical presentation and SPECT-CT imaging patterns. *American Journal of Nuclear Medicine and Molecular Imaging*. Retrieved from

Peters, J. A., Zwerver, J., Diercks, R. L., Elferink-Gemser, M. T., & van den Akker-Scheek, I. (2016). Preventive interventions for tendinopathy: A systematic review. *Journal of Science and Medicine in Sport*. Retrieved from https://doi.org/10.1016/j.jsams.2015.03.008

Reeves, N. D., Maganaris, C. N., & Narici, M. V. (2003). Effect of strength training on human patella tendon mechanical properties of older individuals. *Journal of Physiology*. Retrieved from https://doi.org/10.1113/jphysiol.2002.035576

Revak, A., Diers, K., Kernozek, T., Gheidi, N., & Olbrantz, C. (2017). Achilles Tendon Loading During Heel-Raising and Lowering Exercises.PDF. *Journal of Athletic Training*, *52*(2), 89–96. Retrieved from

Rio, E., Kidgell, D., Lorimer Moseley, G., Gaida, J., Docking, S., Purdam, C., & Cook, J. (2016). Tendon neuroplastic training: Changing the way we think about tendon rehabilitation: A narrative review. *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsports-2015-095215

Rio, E., Purdam, C., Girdwood, M., & Cook, J. (2019). Isometric Exercise to Reduce Pain in Patellar Tendinopathy In-Season: Is It Effective "on the Road"? *Clinical Journal of Sport Medicine : Official Journal of the Canadian Academy of Sport Medicine*. Retrieved from https://doi.org/10.1097/JSM.0000000000000549

Silbernagel, K, Crossley, K, (2015). Proposed return to sport program for patients with midportion Achilles tendinopathy: rationale and implementation. Journal of Orthopaedic & Sports Physical Therapy®. Downloaded from www.jospt.org at Central Michigan University on September 22, 2015. For personal use only. Copyright © Journal of Orthopaedic & Sports Physical Therapy®. All rights reserved.

Soslowsky, L. J., Thomopoulos, S., Esmail, A., Flanagan, C. L., Iannotti, J. P., Williamson, J. D., & Carpenter, J. E. (2002). Rotator cuff tendinosis in an animal model: Role of extrinsic and overuse factors. *Annals of Biomedical Engineering*. Retrieved from https://doi.org/10.1114/1.1509765

Tompra, N., Van Dieën, J. H., & Coppieters, M. W. (2016). Central pain processing is altered in people with Achilles tendinopathy. *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsports-2015-095476

Visnes, H., Aandahl, H. Å., & Bahr, R. (2013). Jumper's knee paradox - Jumping ability is a risk factor for developing jumper's knee: A 5-year prospective study. *British Journal of Sports Medicine*. Retrieved from https://doi.org/10.1136/bjsports-2012-091385

Zwerver, J., Bredeweg, S. W., & Van Den Akker-Scheek, I. (2011). Prevalence of jumper's knee among nonelite athletes from different sports: A cross-sectional survey. *American Journal of Sports Medicine*. Retrieved from https://doi.org/10.1177/0363546511413370