

Osteoarthritis

Clinician Edition

A comprehensive guide to current concepts in diagnosis, pathophysiology and management for MSK Therapists.

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Preface

Osteoarthritis is an extremely common condition affecting millions worldwide. Despite this it remains poorly understood by many clinicians and outdated concepts persist throughout healthcare, the media and in the minds of the people afflicted by the condition.

Working as a Physiotherapist in a variety of roles including Rheumatology, Orthopaedics and private practice I have seen my fair share of patients with Osteoarthritis. Like many clinicians I try to keep abreast of developments in the literature, this is amplified even more so by my decision to teach.

Running courses means that I try to stay ahead of what is commonly accepted knowledge, I theorise and plan for various outcomes as new research is released. As soon as I fall behind, my courses are no longer contemporary and this would clearly be problematic.

I noticed I was being asked more and more about Osteoarthritis as well as Rheumatology conditions and as a consequence I was drawn in to the literature to ask questions of “common knowledge”

This Ebook is part account of my journey and part summary of my understanding of the current state of the understanding in Osteoarthritis. It is written for MSK clinicians such as Physiotherapists, Osteopaths and Chiropractors to try and help to save you time with a summary of the current theories.

This book is my interpretation of ideas, evidence and my reading. It is entirely on my shoulders if I have misunderstood or misrepresented anyone else’s work and I sincerely apologise if I have done so!

There is still a lot to learn and please do get in touch if you would like to discuss any of these concepts, disagree or offer alternative narratives!

I hope you find this book useful and informative.

Enjoy! Jack.

About The Author



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The Rheumatology Physio

Jack is a Physiotherapist who qualified in 2008 from Plymouth University and after rotational posts settled into Rheumatology which he has made his specialty since 2011. He has given seminars, lectures at conferences and courses on Rheumatology subjects mostly covering the topics of Recognition, Investigation and Management. These have been aimed at Allied Health Professionals (Physios, OTs, Nurses...) but have also been attended by Medical Colleagues from GP practices who have also provided positive feedback.

His current roles alongside the provision of CPD include:

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History

Once upon a time, I, like many people, thought of Osteoarthritis (OA) as a simple problem. Much like the tyres of a car, the more your joints were used the more they would wear away. The most common locations to get OA are the knee and the hip, which are overtly high-load joints. Bunions are a Hallux Valgus caused by OA of the first Metacarpal Phalangeal Joint, another known high-load area. This is a pervasive narrative. How many people have you heard over the years say the following?

“Running will wear out your knees”

“I played lots of sports when I was younger so I have arthritis now”

In fact, I ran my own little test and asked my family members (a hopefully biased subgroup) what they think causes Osteoarthritis...

Brother 1 - *“Alien probes or, and this is a complete guess...cell mutations”*

Brother 2 - *“I imagine diet might be a cause? Not enough cartilage-producing vitamins and minerals...”*

Sister-In-Law - *“I’m assuming there are many causes...Is the cause of the pain a lack of cartilage between the bones so you end up with bone rubbing on bone? Erm... Diet? Hypermobility? Overuse (in terms of impact or something along those lines)?”*

Sister - *“I think it’s joint damage - injury, overuse, increased weight bearing due to obesity?”*

Mum - *“Wear and tear of the weight bearing joints”*

This idea that joints have a shelf life or a certain amount of use in them before eventually rubbing away to inevitable *bone-on-bone* with the associated pain, stiffness and lack of range of motion seen in older people is common. It all makes intuitive sense: older people who have used their joints more than younger people get OA and, as the population grows steadily older on average, we are seeing more and more OA diagnosed.

There is also a clear mechanism for those who are overweight being more likely to get OA of course, because with each step more force goes through each joint which may hypothetically speed up the process of degeneration. We can use our car tyre idea again, in that if you carried lots of heavy things in the car you would expect the tyres to wear down faster.

These narratives have taken a bit of a hit in recent times with multiple studies showing a lack of correlation between imaging findings and pain, that runners have lower incidence of symptomatic OA than sedentary people and that obesity causes as much OA in the CarpoMetacarpal Joint as in the knee and hip joints leading to one editorial being brilliantly titled “obese people do not walk on their hands”¹.

This led me personally down a different path and combining this information I reasoned that OA was perhaps not related to load but instead due to inflammatory factors which caused active degradation of the joint. This is built upon a base of understanding gained from working in Rheumatology where I saw inflammation cause structural change to joints in conditions such as Rheumatoid Arthritis (erosions of peripheral joints) and Axial Spondyloarthritis (fusion of Sacroiliac joints and the spine). Returning to the crude analogy, instead of the car tyre wearing away through increased mileage, it was more akin to something picking away at the tyre and removing small pieces so that over time it became damaged regardless of the mileage covered.

I was able to reason a lot with this. We know that obesity causes increased systemic inflammation and joint injury causes high amounts of local inflammation and that even those elite level runners who *did* suffer more OA than recreational runners, well that was because it was an extreme level of running that caused sufficient local inflammation; a threshold that recreational runners did not reach. This is an appealing narrative too because it has some reassuring flavours to it. If load is not the issue then surely more load can't be detrimental to a person's joints so they can be as active as they can tolerate. Keeping active has many benefits but specifically for OA it helps maintain cartilage thickness. This systemic inflammation idea helps make sense of the non-weight bearing joint OA prevalence too. Other pro-inflammatory factors such as smoking, Type 2 diabetes, other inflammatory arthropathies and surgical interventions are things we know can contribute to OA with corticosteroids and NSAIDs being known therapeutic agents in the anti-inflammatory camp.

Of course, as I should have known, replacing one simple explanation with another relatively simple explanation when it comes to humans was a solid error. I have come to learn over the past six months or so that this narrative was probably as wrong as the previous one. Trying my best not to hold on to narratives despite having spent some decent periods of time learning about

them, I plunged once again into trying to get my head around OA and its wonderful complexities. There is absolutely loads we do not know about OA and surprisingly for one of the most common afflictions of humans, it remains highly mysterious to us.

Diagnostics

I hope that that paints a picture of the journey I have been on through my various degrees of understanding of the pathogenesis of OA. What follows is my attempt at explaining where I am at currently, with some immediate caveats! There remain gaps within my knowledge that I struggle to fill for a variety of reasons; a combination of an inability to understand the hard science and gaps in the scientific literature itself. I certainly do not profess to be an expert!

Firstly we need to make a nod to the complexity of humans themselves. Knee joints for example do not exist outside of a person with their unique personality, neurological system, physical, psychological and social history. No two people are the same and no two knees are the same and therefore no presentations/experiences of OA will be the same.

This leads us immediately off-track to a discussion around Symptomatic and Radiological OA.

Put simply, those people who report symptoms from the Osteoarthritic process are in the Symptomatic category but don't necessarily show any clear findings on imaging. Those people who show imaging changes fit into the Radiological category and of course there are those who have both^{2,3,4,5,6}. Let's consider the implications of this.

Some people have OA symptomology but with no radiological changes.

Some people have MRI changes associated with OA but report no symptoms.

How do we know whether the symptoms are related to the changes seen on imaging when some people display these changes with no symptoms? The answer is clinical correlation, which means that when a person attends clinic, we clinically assess their symptoms, form a hypothesis diagnosis (in this case OA) and then request radiological investigations to confirm it. This is a different approach to requesting images and then forming a hypothesis on receipt of the

results. You can go as far as to say, you cannot see pain on imaging alone.

The thing is though, if a clinician thinks the diagnosis is OA and sends for an MRI, some scans will come back with changes and some won't. Does this change the diagnosis in either direction? Probably not! One study⁷ showed that imaging improved GPs' confidence in their diagnosis but did not change their diagnosis or treatment plan compared with when they did not have imaging available. This is further supported by the NICE Clinical Practice Guidelines⁸ which state:

Diagnose Osteoarthritis clinically without investigations if a person:

- Is over 45 years old and
- Has activity-related joint pain and
- Has either no morning joint-related stiffness or morning stiffness that lasts less than 30 minutes

The assessment also of course involves ruling out other clinically important conditions such as other types of arthritis, joint injuries and/or referred pain from elsewhere but I hope this is becoming clear. Clinical reasoning taking into account the symptoms presented is better than imaging.

Imaging when there is a suspicion of OA is therefore best utilised in those who are either younger than 45 (possible early onset OA), when symptoms are progressing rapidly or to rule out other differential diagnoses. It may also be used to monitor changes as correlation has been shown between worsening symptoms and deterioration of joint space narrowing⁹.

An Altered Paradigm

Let's return for a moment to my previous understanding of OA; that it was an active process exaggerated by upregulators of the inflammatory process such as obesity, joint injury, smoking, genetic predisposition and more. I was not far off but as my understanding has evolved there are some subtle but important errors in this reasoning process. For example, it doesn't account for ageing which is the dominant predisposing factor for developing OA.

I was extremely lucky to speak to Professor Tonia Vincent who amongst other things (Rheumatologist, Professor of Musculoskeletal Biology) is the Director for the Centre of Osteoarthritis Pathogenesis. There is a link in the resources section where you can listen to that conversation in full. I learned a LOT from that conversation, one part of which was a reframing of how I thought about the process of joint change in OA.

In OA we are considering the turnover of the cartilaginous matrix, which consists mainly of collagen and proteoglycans. Chondrocytes are the only cells in this matrix and are responsible for maintenance and restoration of the tissue

Rather than an active degradation of the joint structure, Professor Vincent suggested we should think of OA as a **suppression of restoration** during the normal turnover of the tissues. All tissues in the body turnover as cells don't last forever and they are replaced with shiny new ones. To me this immediately makes more sense and solves the puzzle to do with ageing. To elaborate on this further, the cartilage of the joint undergoes this entirely normal restoration process like the rest of the body's structures. As we age this process slows but the demand remains the same, leading over time to less high quality tissue forming the cartilage. This lower quality tissue has a higher percentage water and is easily squashed, eventually becoming thinner. There is a net negative turnover which leads to the joint space narrowing seen on imaging. For a healthy joint, net neutral turnover is desirable. The holy grail of treatment would be to create a net positive turnover so that the volume of tissue increases.

This net turnover can be upregulated and downregulated by various things that happen to the joint either directly or indirectly. If the restoration process is slowed down to below the rate of the cell degradation this results in a net negative turnover. If you stimulate the restoration process to be faster than the cell degradation you will see a net positive turnover.

Things that upregulate the restoration process:

- Loading (exercise)

Things that downregulate the restoration process:

- Increasing age
- Increased systemic inflammation
- Obesity
- Low physical activity levels

Things that increase the rate of matrix degradation:

- Joint injury
- “Shear forces”
- Inflammatory Arthritides

Loading (exercise) causes a compressive effect on the cartilage, which stimulates chondrocyte activity thereby optimising the maintenance of the matrix. Think of it in the same way as exercise for muscles and the adage “use it or lose it”. If the tissues are not used or stimulated then they suffer atrophy. Of course the opposite is true for inactivity – if you load the joints less, you can expect poorer maintenance of the cartilage matrix.

Increased systemic inflammation can be caused by many long-term conditions including obesity. Fat cells (particularly abdominal ones) release adipokines into the system, which are inflammatory upregulators. The inflammatory process stimulates the synovium of the joint, which leads to more synovial fluid in the joint, increased amounts of inflammatory cytokines in the synovial fluid and reduced suppression of osteoclast formation (sorry about the double negative). This low-grade increase in systemic inflammation causes a small increase in activity in the synovium (synovitis), enough to downregulate the restoration process.

We need to eliminate the belief that the joints “wear out” and that obesity increases this through heightened load as this is counter to the evidence and negatively impacts the narrative we want to get across to people, which is to increase activity levels to help manage OA. If a person thinks their joints are “wearing out” with activity, why on earth would they want to do anything that potentially increases that?

Joint injuries can have multiple effects on the joint from an risk point of view. Of course something like a tear will literally disrupt the matrix but other intra-articular injuries have effects too. ACL ruptures for example lead to OA at an alarming rate whether they are repaired or not. It is possible that the large inflammatory reaction at the time of injury, combined with a significant period of decreased physical activity and potential surgery (which restarts the whole cycle of inflammation and restricted physical activity) downregulates the matrix restoration process sufficiently that the joint never recovers. It is also possible that there are an increased amount of shear forces due to the resulting knee instability. As usual, a combination of these things is probably most likely.

The importance of **shear forces** are another big takeaway from my discussion with Professor Vincent. To be honest it is something I am still grappling with so please see this section as a work in progress.

Professor Vincent described a shear force essentially as a force directed in a non-perpendicular direction to the cartilage matrix. In the knee for example, shear forces result from lateral movement or anterior/posterior movement of the joint, which we see more excessively following ligament injuries. Load applied in a perpendicular direction to the matrix in the knee joint would come from axial force, which is considered to be helpful loading. I am unclear at this time if the shear forces themselves reduce the chondrocyte activation or they take some of the force away from the perpendicular force. With either reasoning process this may go some way to explain why we see increased rates of OA in Hypermobility Syndromes but these of course result from a collagen variation and what is cartilage made from...? There is complexity everywhere you look.

Here we start to bounce off the ceiling of my reasoning (and probably intelligence) because joints move and this involves sliding and rotating across the cartilage matrix which creates a shear force. What I don't yet have answers for is what makes for a tolerable level of shear force and what tips the process into overload. We also see a wide variety of movement patterns in humans both globally and locally. What is normal for me is not necessarily normal for you and so determining 'normal' at an individual level is very difficult. Finally, add this to the fact that we can't measure shear forces currently (as far as I am aware) and therefore don't have normative values, never mind the ability to measure for a threshold for risk of developing OA.

What does that mean for Therapists? Again in all honesty, I don't really know at this stage. We should continue to encourage loading and exercise to upregulate the chondrocyte activity. Maybe (and I can't stress how strongly this is purely theory) it would be better to provide closed chain exercises or attempt to restrict the planes of movement the joint can move in. I am thinking cable machines over free-weight or barbell exercises but then how much of someone's daily activity is spent "exercising"? Probably a fairly small percentage and so is that change really going to make much difference? I have no idea but my hunch is that I doubt it.

Other inflammatory arthritides including Rheumatoid Arthritis and Axial Spondyloarthritis have two possible mechanisms for increasing risk both directly and indirectly. The acute synovitis and potential subsequent intra-articular joint damage from these pathologies both increases the inflammatory processes around the joint and directly reduces the cartilage thickness. Indirectly all inflammatory arthritides increase systemic inflammation, which we have already discussed will affect OA risk.

Increasing age is where we begin the conversation about complexity, individualism and confounding factors. We know that with increasing age comes a slowing down of bodily processes. We have already mentioned that some

things will alter the net turnover of the matrix so I would theorise it would be possible to negate the slowing tissue restoration process with loading in order to maintain the cartilage matrix into advanced age. Probably not forever and if you were to live long enough then OA may well be an inevitability. We can use the example of Osteoporosis. Bone density reduces with age but loading holds off this reduction so why wouldn't it be the same in OA?

Now, the complexity with the factor of increased age is that it is traditionally associated with reduced loading through decreased activity levels. People retire, give up hobbies and so on, becoming more sedentary. Maybe this is changing through a societal shift towards a more active older generation. With increased age you have also had more time to sustain injuries in the past, develop other illnesses or comorbidities and undergo surgeries for whatever reason meaning that these risk factors likely feature more as well. I don't think we can really extricate the specific factors from ageing.

As we mentioned earlier these joints don't exist in a vacuum and the development of OA is inevitably multifaceted.

Phenotypes

A Phenotype is an individual's observable traits which are different expressions of the same characteristic. For example, brown, blue and green are different phenotypes of eye colour.

I listened to a podcast on Phenotypes of OA and I have outlined my takeaways from that here. I HIGHLY doubt people will present with purely one phenotype and I also suspect there are more types! I have also read different articles that don't seem to agree on phenotypes and even a consensus statement that showed they were still at the point of deciding how to even research them⁹. I do want to point out specifically here that there is disagreement amongst researchers as to whether phenotypes exist at all. My personal viewpoint is that phenotypes as a factor in OA makes a lot of intuitive sense, can be used to explain differences in presentation to patients and answer some of the questions I have. They should be used with a lot of care in the clinical environment and so avoid absolute certainty.

Possible Phenotypes of Osteoarthritis – and their most common features

- ***Age-related***
 - o Multiple joints affected
 - o Most commonly the knees and hips, then the 1st MTPJs and the CMCJs
 - o Other joints affected variably
 - o Slower onset starting after the age of 60

- ***Injury-related***
 - o Single joint affected
 - o Most likely the knee because of the high incidence of injury and surgical intervention
 - o Potentially faster and younger age of onset
 - o History of injury

- ***Obesity-related*** (could also add other systemic inflammatory issues)
 - o Variable age of onset
 - o Often multiple joints affected
 - o Commonly the CMCJs affected

- ***Joint shape-related***
 - o Younger age of onset
 - o More common in the hips (dysplasia, FAI)
 - o Joint shape changes following fracture of the shoulder
 - o Intra-articular joint changes of the ankle (anterior or posterior impingement) which is likely due to focal loading affecting the ability of the chondrocytes to maintain that area of matrix sufficiently

Genetics/Family History

Genetics plays an important role across the spectrum of what we have already discussed. It is likely that there are genetic variations of both predisposing and

protective nature for almost all of the risk factors. My reading and understanding is that genetics is not hugely relevant to us in clinical practice for a variety of reasons, top of the pile being the volume of confounding factors, different phenotypes and other individual factors such as psychosocial variables.

There are other societal changes at play as well. Smoking was previously much more widespread but now pollution is more prevalent. Diets have changed in some ways for the better and in other ways for the worse. Can we use even family history to suppose risk? Perhaps in siblings, for example if someone's brother or sister has a diagnosis of OA without significant joint injury, obesity or other confounding factor.

Genetic predisposition could work both to increase the risk of developing OA but also to reduce the risk. Possible variances include an increased response to loading exercise by way of upregulating the chondrocyte function or a more resilient cartilaginous matrix. The opposite is also true, where people are predisposed to a less resilient matrix and less reactive chondrocytes. There are also likely genetic variances in an individual's likelihood of experiencing symptoms of OA. Unfortunately, I think we are a long way from using this in the clinic in any way.

Symptomatic Presentation

As we discussed, some people have symptomatic OA with no radiological changes, others have Radiological OA and some have both. It is well outside the scope of this eBook to delve fully into factors that drive pain perception but there are certain things that make a person more likely to experience symptoms from their joint. These things are "*associated*" with increased symptom severity and so having one or more doesn't mean that symptoms will definitely be experienced and having none doesn't mean symptoms won't be experienced. Here is a non-exhaustive list:

- Anxiety
- Depression
- Other painful areas or previous high pain experiences
- Low job satisfaction or unemployment
- Poor sleep
- Poor diet

There does not appear to be an association between the amount of inflammation or joint changes and reported pain but increased joint space narrowing of the knee is associated with deteriorating symptoms. Osteophytes in the knee are not associated with reported pain and osteoarthritic changes on MRI of the spine have also been seen in asymptomatic populations and don't seem to be associated with symptom severity^{2,3,4}.

There is also a varied presentation of symptoms such as pain intensity, swelling, degree of stiffness or response of symptoms to activity, which will differ between individuals. Symptoms can also be transient in the early stages, varying their severity over the course of days, weeks or months. This is all of an individual nature and is one reason that the NICE guidelines have such a broad diagnostic criteria⁸.

Pain

There are disagreements amongst researchers as to the pain-generating structures in OA. We see a disease of the whole joint affecting the synovium, cartilage and bone and of course we can't extricate the joint from the human. Professor Vincent argues that we see nerve innervation into the cartilage in OA making cartilage a possible pain generator where it was previously argued that it could not be due to not being innervated. We have seen that Bone Marrow Lesions (BMLs) in hand OA are not associated with pain alone, whereas synovitis is and the combination of the two is associated with the most pain⁶. BMLs are seen more frequently in painful knees than non-painful knees and the severity of the synovitis also appears correlated¹⁰.

To me this clearly indicates a combination of factors combining to be pain generators. As with the rest of the contributors we have discussed in this book, there is no way of isolating these structural factors from the person and too narrow of a focus will only lead to partially answered questions. Consideration of psychosocial factors is extremely important but not always accounted for in these mechanistic studies and reviews.

Flare Ups

It is clear that many people experience periodic increased symptoms related to their OA, which are variably referred to as flare ups, exacerbations or periods of worsened symptoms in the literature¹⁷. We see this clinically too. People seek help because their symptoms are worse. Sometimes this is because of an initiating trigger like increased activity or an incident (fall, injury) and sometimes it is insidious. Regardless, these episodes can be severe, debilitating and of concern for the individual that their pain has suddenly deteriorated significantly.

Most of the time these flares do not represent deterioration of the joint and resolve over time¹⁸. There is however an interesting lack of evidence regarding non-pharmacological management of these episodes¹⁷. Best treatment currently for flare-ups is to modify activity as deemed appropriate, apply symptom modification modalities if possible, practicable and safe for the individual and most of all educate/reassure that the episode will most likely improve over time.

Exercise and activity remains safe and can be encouraged at the person's tolerance level but at present we cannot say for certain that it will be beneficial for pain in the short term although it might be helpful for symptoms of stiffness.

If symptoms are significantly affecting function then a medication review with the person's GP is an option.

Stratification

Exercise seems to be beneficial for treating OA but we don't really see obvious differences in benefits with different types of activity or doses. As we discussed, the load bearing aspect would upregulate the chondrocytes to maintain the cellular matrix of the cartilage but there are also wider systemic effects of exercise on the inflammatory system, psychological system, neurological system and overall function to consider. Exercise as a short term analgesic seems unlikely but might be possible in some cases, though it seems more likely that the impact would be in the longer term. The primary effects of exercise include increased stability of the joint and increased tolerance of the joint tissues to load and secondary effects incorporate decreased systemic inflammation, weight loss, psychological feelings of robustness, and social interaction if exercising in groups.

The variable responses may well be due to an inability to stratify by phenotype or to account for the individual nature of humans. Let's consider a joint shape-related OA. Loading exercise may have benefits for increased strength and cardiovascular fitness but may be detrimental for others like pain levels.

Personally, if I was to speculate, this is something we need to account for if we are going to improve outcomes with treatments. Directed treatments based on tailored assessments of who would benefit from them the most is likely to be the holy grail of OA management. How we see this progress will be fascinating and complex due to the confounding factors.

Exercise and Activity

NICE guidance states that this is one of the three core treatments for OA (alongside education and weight loss) and goes on to be a bit more specific saying this should incorporate "local strengthening and general cardiovascular exercise"⁸.

Research seems a little inconclusive about the specifics and there seems to be a trend towards improvement in outcomes but how to achieve this is rather all over the place¹⁶. This may be an issue with the nature of Randomised Controlled Trials not catering to individuals and exercise being a multifactorial intervention. Imagine some of your cohort "don't like" the exercises for whatever reason, which could have an impact on the outcome. Individualising exercise taking into account patient preference or enjoyment may enhance the outcomes that are difficult to account for in RCTs. The other issue which we mentioned previously might be the inability to stratify these patients appropriately to phenotype.

There is of course a final possibility that once someone has clinical signs of OA, exercise is only modestly effective. There seems to be a lot more work to be done in this space.

My current assessment of the evidence surrounding exercise and activity is that without the overt difference between exercise/activity types then a patient-led approach is the best option. Supporting the patient to make choices around increasing their activity levels and promoting exercise regimes that incorporate strengthening, aerobic and range of motion components would seem safe options. Educating the person to adapt their exercise regimen for both when they have increased symptoms or indeed increased capacity is a valuable skill.

Dietary Supplements

All manner of supplements have been reported as beneficial for OA both anecdotally by patients (personally I have heard everything from tomatoes to cider vinegar to Brie...) and of course the widely touted Glucosamine and Chondroitin.

This meta-analysis¹² in 2018 wasn't impressed by the literature quality but there might be some short-term benefits with some supplements but even in these low quality trials this didn't extend to the long-term. Glucosamine and Chondroitin were shown to be either ineffective or if they did demonstrate benefit it was clinically insignificant. NICE guidance specifies not to offer these supplements⁸.

The conclusion is if someone finds benefit and can afford the supplements then they are of low harm, otherwise they should save their money.

Weight Loss

Weight loss as a specific treatment for OA appears effective to both reduce pain and improve physical function for those patients who are overweight. Clinical differences in pain are seen with weight loss starting at approximately 6% of body weight with physical function improvements seen at 15%. It does seem of more benefit to both pain and physical function to lose 10-20% over the long-term and 25% weightloss has been shown to improve outcome measures by 50%^{13,14,15}.

When you combine weight loss with an exercise program it appears that the amount of improvement in pain remains the same but there are greater improvements in physical function¹⁴.

It is outside the scope of this eBook to discuss weight loss protocols but care should be taken to do this in an appropriate, healthy and compassionate manner that promotes long-term maintenance of the weight loss. It should also be noted that the cause of obesity is a multifactorial and it may require specialist input.

Conclusions

OA is a complex disease of the entire joint and advances in understanding in joint models (usually in mice) are difficult to apply in clinical practice. The overlapping nature of these phenotypes means that stratifying patients to different care groups is a massive challenge, which may well be why we see such variation in treatment responses.


Just because it is difficult to understand does not mean we shouldn't try and I hope I have provided a narrative here that clinicians can use to reassure patients that their joints are not going to wear away over time.

There remain many unknowns in the research, phenotypes being a classic example. Do they even exist? Accounting for the individual nature of humans remains a challenge, Randomised Controlled Trials can suffer with having to sacrifice internal validity to gain external validity and vice versa. This commonly leads to losing the individual in the process and affects our ability to apply research to clinical practice.


Clinical assessments should mostly centre around ruling out other clinically important differential diagnoses and the person's function. Investigations should be limited to again ruling out other conditions, younger patients and those with rapidly progressing symptoms.

Individualising management plans with the person to incorporate their preferences is likely to be far more effective than a didactic attempt to design the perfect exercise program. Include strengthening, aerobic and range of motion components in any given programme and advise on weight management, general health (sleep, activity, smoking cessation, mental health etc) within your skill base.


Further Resources


[The Physio Matters Podcast Session 87 - Wear Are We With Osteoarthritis with Professor Tonia Vincent](#) 

[The Joint Action Podcast](#) 


[Greg Lehman's website – OA Optimism](#) 

More From Jack


[Rheumatology.Physio](#) contains a wealth of learning resources concentrating on Rheumatology topics which are free to access. There are also booklets including the AtAGlance Series (Rheumatology, Spinal Masqueraders, The Hand and The Hip), a clinical scenarios EBook and an Audiobook. 


Jack also runs Continuing Education Courses for MSK Therapists, online and in-person courses are available to book and bespoke departmental courses on application. There is also a 6 hour pre-recorded course covering Rheumatology recognition and management in great depth. [Find all of these here](#) 


The Rheumatology.Physio Podcast channel contains the much loved RheumMates podcasts and weekly blog reads. It is available on all podcast channels, [you can find your favourite here](#) 

Jack sees Rheumatology patients via virtual consultation for second opinions, management advice and guidance. [More information and booking here](#) 

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Jack runs on a fuel of coffee and there is none better than [UWhoLifestyle Coffee](#). Delivered to your door, ad hoc or on subscription and environmentally friendly packaging. 

[Physio-Matters.com](#) is frankly the best place to get CPD with all the TherapyLive conference recordings and a whole lot more on offer for an absolute bargain price. 

[HMDG](#) created and manage the Rheumatology.Physio website and Jack would not trust anyone else to work with for websites or Marketing. You will never regret a call with them to see how they can bring you amazing results. 

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