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SPORTSHEALING

Protecting the knee for life

What the evidence says, and what I do about it

By the President of the British Knee Society · October 2026 · Reference numbers in square brackets point to the list at the end.

Introduction

I have spent my career inside knees. I have seen what forty years of neglect does to a joint, and I have seen what careful attention preserves. Luck plays a smaller part in which outcome you get than most people assume. Genetics and past injury play their part, but a large share of what I see in clinic is the product of habits, and habits can change.

Knee osteoarthritis is now the most common reason for joint replacement in the UK. Stiff, painful knees trouble more than one in five adults over 50. Yet the condition is not simply wear and tear. The knee is a living organ that adapts to load, nutrition, sleep and the metabolic environment of the whole body. That understanding has changed how I advise patients, and how I live.

This article sets out the evidence under four headings: general health and wellness, biomechanics, managing load, and sleep and rest. In each I describe what the research supports, where it is weaker than people assume, and what I personally do. Where I give an opinion that goes beyond the data, I say so.

Part 1. General health and wellness

Weight is the dominant modifiable risk. The evidence here is as strong as anything in musculoskeletal medicine. A meta-analysis of 14 prospective studies found that being overweight more than doubles the risk of knee osteoarthritis and that obesity raises it more than fourfold. Each 5 kg/m² increase in BMI adds around 35 per cent to the risk, and the effect held across countries, sexes and lengths of follow-up [1].

The mechanism is partly mechanical. Stephen Messier's group at Wake Forest measured knee loading before and after weight loss in older adults with knee arthritis and found that every pound lost removed about four pounds of compressive load from the knee per step [2]. Over a mile of walking that is thousands of pounds of accumulated force spared. A later Danish trial estimated the saving at 2.2 kg of peak knee load for every kilogram lost [3].

Metabolic health is a separate risk, not just a proxy for weight. Fat tissue is metabolically active. It secretes adipokines such as leptin and drives low grade systemic inflammation measured by C-reactive protein. Both reach the joint. Metabolic syndrome has been linked to knee osteoarthritis in a case-control analysis of over 24,000 people after adjusting for BMI, age, sex and activity, and higher CRP tracks with worse pain scores [4]. The strongest clue that this is systemic rather than mechanical is that metabolic syndrome is also associated with osteoarthritis of the hand, a joint that bears no body weight.

There are limits to this. Diabetes on its own does not appear to be an independent risk factor once BMI is accounted for. A 2020 meta-analysis of 31 studies and 295,000 people found no increased odds of osteoarthritis in people with diabetes after that adjustment [5]. So the picture is one of metabolic syndrome as a cluster, with central adiposity and inflammation doing most of the work, rather than blood glucose alone.

Nutrition. The dietary pattern with the best support is Mediterranean. In the Osteoarthritis Initiative cohort, people in the highest quartile of adherence had a knee osteoarthritis prevalence of 25 per cent compared with 34 per cent in the lowest quartile [6]. Over four years of follow-up, high adherence was associated with a 9 per cent lower risk of developing symptomatic knee arthritis and a lower risk of pain worsening [7]. These are observational findings and the effects are modest, but they point in a consistent direction and the diet carries no downside.

Smoking. Smoking rarely gets a mention in articles about knees. It deserves one. For years observational studies hinted that smokers had less osteoarthritis. The finding has not held up. Smokers tend to be lighter, and the early studies drew their non-smoking controls from hospital patients with smoking-related disease, so the apparent protection was an artefact [29]. Better designed studies point the other way. In a 30 month study of men with knee arthritis, current smokers had more than twice the risk of cartilage loss in the inner compartment of the knee and reported more pain [30]. Two 2023 cohorts found no effect on cartilage volume, so the structural picture is mixed [31]. There is no such ambiguity about healing. Nicotine constricts blood vessels and suppresses type 1 collagen synthesis, the protein that tendons, ligaments and menisci are built from. After ACL reconstruction, smokers have more surgical infections, more residual laxity and worse functional scores [32, 33]. The encouraging finding is that patients who stopped at least a month before surgery did as well as those who had never smoked [34]. If you smoke, stopping is the most useful thing you can do for your knees after losing weight.

Supplements with thin evidence. NICE guideline NG226 states that glucosamine should not be offered for osteoarthritis [8], and the Australian national guideline also recommends against chondroitin, omega-3 and diacerein, each on very low quality evidence [9]. The trial data are thin and I do not recommend them routinely. That said, some of my patients report real relief from glucosamine or chondroitin, and since both are safe for most people I do not stop anyone who finds them useful. Turmeric, or its active component curcumin, sits in the same category. It has anti-inflammatory properties and some patients find it eases pain, though it will not change the joint itself. One caution matters here: turmeric has a mild blood thinning effect, so it should not be combined with anticoagulants or antiplatelet drugs such as warfarin, apixaban or

aspirin without medical advice, because the combination raises the risk of bruising and bleeding.

Vitamin D. I take vitamin D and recommend it, and I think the evidence justifies that, provided the claims stay within what the trials actually show. Its role in bone health is settled. Its role in muscle is well supported: vitamin D receptors sit in muscle tissue, deficiency tracks with weakness and sarcopenia, and a 2024 meta-analysis of 2,431 older adults found that active vitamin D compounds improved quadriceps strength and cut falls by 19 per cent [35]. Doses of 700 to 2,000 IU per day are associated with fewer falls in people over 50 [36]. For the knee joint itself the picture is more specific. A meta-analysis of eight randomised trials in 3,077 patients with knee arthritis found that supplementation improved pain and function scores, though not stiffness, and had no effect on cartilage volume or joint space [37]. An earlier analysis found the benefit was clearest at doses above 2,000 IU per day [38]. Vitamin D will not regrow cartilage, whatever the marketing says. What it does is support the bone beneath the cartilage, the muscle that protects the joint and the balance that prevents falls, with a modest effect on symptoms. In a country where most of us are deficient from October to March, that is a good return for a cheap and safe supplement. The NHS advice of 400 IU daily through autumn and winter is a floor, not a ceiling. For most adults I suggest 1,000 to 3,000 IU a day, which sits comfortably below the 4,000 IU safe upper limit. People with darker skin make less vitamin D from British sunlight and should sit at the top of that range.

Collagen. The evidence for collagen is more recent and thinner, but it exists. Collagen peptides are hydrolysed collagen, broken into fragments small enough to be absorbed. A 2023 meta-analysis of four randomised trials in 507 patients with knee arthritis found a moderate reduction in pain compared with placebo, with no excess side effects, and one head to head trial found collagen peptide outperformed glucosamine over 90 days [39]. The strength data are more striking. In 53 men over 65 with sarcopenia, 15 grams of collagen peptides daily alongside 12 weeks of resistance training produced 4.2 kg of fat-free mass against 2.9 kg on placebo, and more than double the gain in quadriceps strength [40]. The same group replicated the finding in 77 premenopausal women, who gained more fat-free mass, grip strength and leg strength than training alone [41]. For tendon and ligament, Keith Baar's laboratory showed that 15 grams of gelatin with vitamin C taken an hour before a short burst of exercise doubled markers of collagen synthesis, and that serum from supplemented volunteers made engineered ligaments measurably stronger [42].

The limits need stating. These are small trials, several from a single research group, often using a manufacturer's product. The tendon work is biomarker and laboratory tissue, not human tendons. The pain benefit in arthritis is moderate and the trials were rated at high risk of bias. Collagen is plausible and promising rather than proven. Since the downside is close to zero, I take it, timed before training.

Other peptides. Patients increasingly ask me about injectable peptides sold online, BPC-157 and TB-500 among them, with claims of tendon and cartilage repair. The data are animal and cell culture only. There are no published human randomised trials, they are not licensed medicines in the UK, and the products are unregulated. I do not use them and I advise against them until human evidence exists.

What I do. I keep my BMI below 25. I eat fish, pulses, vegetables, olive oil and whole grains, and I limit ultra-processed food. I have my blood pressure and lipids checked. I have never smoked. I take 3,000 IU of vitamin D every day, all year. I have Asian skin, which makes less vitamin D from British sunlight, so I sit at the top of the range I recommend to patients. I take 15 grams of collagen peptides with vitamin C about an hour before strength sessions. I take nothing else for my joints.

Part 2. Biomechanics

Muscle is the knee's primary shock absorber. Cartilage is thin, avascular and slow to repair. The quadriceps, hamstrings and gluteal muscles are the structures that absorb impact before it reaches the joint surface. In the Multicenter Osteoarthritis Study, adults in the weakest quartile of knee extensor power had nearly twice the odds of developing frequent knee pain over two years compared with the strongest quartile [10]. In women, quadriceps weakness raised the risk of worsening cartilage damage behind the kneecap by 50 per cent [11]. Weak muscle fatigues sooner, controls the joint less precisely and transmits more load to bone and cartilage.

Leg strength training is therefore the foundation of everything else in this article. Progressive resistance work two or three times a week has the best evidence for both preventing and managing knee pain, and it is the part most of my patients skip.

Depth and the patellofemoral joint. Here I part company with some of the strength and conditioning literature, so I will set out the reasoning. The force across the patellofemoral joint, where the kneecap meets the thigh bone, rises steeply with knee flexion. Biomechanical studies of the squat consistently show that patellofemoral stress increases as depth increases and increases again when external load is added [12]. The same is true of the lunge. Patellofemoral force climbs progressively through flexion, and a short step lunge loads the kneecap more than a long step between 70 and 90 degrees [13].

The counter-argument is that trained lifters squat deep for decades with healthy knees, and that research in that population does not show harm [14]. I accept that. But my clinic is not full of elite powerlifters. It is full of 45 to 70 year olds, many with early cartilage change behind the kneecap that they do not yet know about. For them the balance of risk is different. The occupational evidence supports caution. A dose-response meta-analysis found that each 5,000 cumulative hours of kneeling or squatting at work raised the odds of knee osteoarthritis by 26 per cent [15]. A review of 71 studies covering almost a million people found that jobs involving frequent deep knee flexion carried around 50 per cent higher odds of knee arthritis than sedentary work [16]. Repeated deep flexion under load, accumulated over years, is not benign.

In practice I advise the following. For the typical adult past 40, and for anyone with patellofemoral symptoms, squat to around 90 degrees of knee flexion, no deeper. Use a long step when you lunge and avoid dropping the back knee to the floor under load. Keep the knee tracking over the second toe. You will get almost all of the muscular benefit with a fraction of the patellofemoral stress. If you are a young, well conditioned athlete with no knee history, the evidence does not say you must stop squatting deep. It says you are taking on a load that your joint may or may not

tolerate for life.

Running. Many people are surprised to hear that recreational running is not associated with knee arthritis. The largest meta-analysis, covering 25 studies and over 125,000 people, found hip and knee osteoarthritis in 3.5 per cent of recreational runners compared with 10.2 per cent of non-runners. Competitive runners, at 13.3 per cent, were the group at higher risk [17]. Running at moderate volumes appears to condition the joint. The dose, the surface and the pace matter more than the activity itself.

What I do. Two leg strength sessions a week. Squats to 90 degrees, long step lunges, deadlifts, hamstring curls and step-ups. Form matters more than load. When I squat I watch that the knee does not drift inward into valgus, because that twists the kneecap in its groove. I am careful with isolated leg extensions on a machine, which load the patellofemoral joint heavily through the last part of the movement, and I stop any exercise that produces kneecap pain rather than pushing through it. I run 10 to 15 km a week on mixed surfaces and add another 10 to 15 km of brisk walking with my wife. I do not do loaded deep squats, deep lunges or plyometric box jumps.

Part 3. Managing load

Tissue fails when load changes faster than it adapts. This is the principle that unites most non-contact knee injuries I see, from the runner with a stress reaction to the weekend footballer with a torn meniscus. The best data come from professional sport. A three year study of English Premier League players found that when a player's weekly training load spiked to more than twice their recent four week average, and their chronic conditioning was low, non-contact injury risk rose six to seven fold [18]. The protective finding was just as important. Players who had built a high chronic load over months tolerated spikes far better, because conditioning is what allows a body to absorb a hard week.

I should note that the specific metric used in that research, the acute to chronic workload ratio, has been criticised on statistical grounds [19]. The criticism is fair. But the underlying principle, that sudden unaccustomed load is dangerous and that gradual progression protects, is supported across sports and across decades of clinical experience. It is as true for the person who clears a garden after a sedentary winter as it is for a footballer.

Fatigue changes how the knee moves. When muscles tire, the body lands stiffer and the knee flexes less. Studies of landing mechanics under fatigue show reduced knee flexion, altered hip control and degraded central motor control, all of which shift load onto passive structures like the anterior cruciate ligament and the menisci [20]. ACL injuries cluster in the first and last 15 minutes of matches, when players are either not yet warm or already exhausted [21]. This is why I advise against high impact work, such as box jumps or repeated maximal sprints, at the end of a hard session. The forces through the knee on landing from a box can reach many multiples of body weight. Add fatigued muscle and a tired nervous system and the knee is poorly placed to cope.

Walking is the safest load there is. In the Osteoarthritis Initiative, adults over 50 with knee arthritis who walked for exercise had 40 per cent lower odds of developing new frequent knee pain and 20 per cent lower odds of medial joint space narrowing over four years [22]. The effect was strongest in those not yet in daily pain, which is the group where prevention has most to offer.

Underloading harms too. Cartilage has no blood supply. It is nourished by synovial fluid being pressed in and out of the tissue during movement. Laboratory work shows that long uninterrupted sedentary bouts reduce cartilage hydration and raise friction, even when total daily activity is held constant [23]. In cohort data, more than four hours a day of leisure time sitting and fewer than 6,000 steps a day were both associated with faster structural worsening of knee arthritis [24]. The knee needs the right amount of load, distributed through the day. Too little harms it as surely as too much.

What I do. I never increase running volume by more than about 10 per cent a week. I build strength before speed. I do my most explosive work early in a session, never last. I take the stairs and I stand up every 30 to 45 minutes when I am operating all day or sitting in clinic. I ski about once a year. Before I go I make sure my legs and core are trained for it, I do not ski after drinking alcohol, and I avoid the afternoon when the snow turns to slush and the knee has to cope with unpredictable loads.

Part 4. Sleep and rest

Sleep is when the knee repairs. Growth hormone release, collagen synthesis and the clearance of inflammatory mediators are all concentrated in deep sleep. The epidemiology is consistent. In a prospective study of Division I basketball and volleyball players, each additional hour of sleep was associated with a 32 to 42 per cent lower risk of injury the following day [25]. Adolescent athletes sleeping under eight hours had 1.7 times the injury risk of those sleeping more [26]. A 2025 study of 425 recreational runners found that poor sleep quality or short duration raised injury odds 1.78 fold over a year [27]. The pattern holds from school sport to the professional game.

Poor sleep amplifies pain. This matters for anyone who already has knee arthritis. In a study of 208 participants, those with knee osteoarthritis and insomnia showed the greatest degree of central sensitisation, the process by which the nervous system turns up the gain on pain signals [28]. A bad night therefore makes a sore knee feel worse than the joint itself would justify, and chronic poor sleep can lock that pattern in. Treating sleep is a legitimate part of treating knee pain, and cognitive behavioural therapy for insomnia is now being trialled in this population for exactly that reason.

Rest days are training. Adaptation to load happens during recovery, not during the session. The professional football data above make the point: it is not high load that injures, it is high load without the recovery and conditioning to support it. Rest days, easy weeks and stopping when a joint is swollen are how strength is actually built.

What I do. I am in bed by 10 and up at 5.30, which gives me about seven and a half hours. Before I leave for work at 6.45 I do a short routine of yoga, breathwork and meditation. I do not train hard two days in a row. If a knee is swollen or warm I rest it, and I do not return to impact until it has settled.

Summary

The knee is a living structure that responds to the whole body, not a hinge that simply wears out. The four principles in this article reinforce each other. Good metabolic health reduces inflammation at the joint and the load it carries. Strong muscle protects cartilage from impact. Gradual, well distributed load builds tissue tolerance while sudden spikes and fatigue break it down. Sleep is where all of that adaptation actually happens.

If I could give every patient one piece of advice it would be this: keep your weight and metabolism in order, do not smoke, build the muscles around the knee, change load slowly and never when exhausted, keep the knee out of deep flexion under load, and sleep. None of it is exciting, and all of it works.

References

Links go to open access versions or publisher abstracts.

1. Zheng H, Chen C. Body mass index and risk of knee osteoarthritis: systematic review and meta-analysis of prospective studies. *BMJ Open* 2015;5(12):e007568. [Link](#)
2. Messier SP et al. Weight loss reduces knee-joint loads in overweight and obese older adults with knee osteoarthritis. *Arthritis Rheum* 2005. [Link](#)
3. Aaboe J, Bliddal H, Messier SP, Alkjaer T, Henriksen M. Effects of an intensive weight loss program on knee joint loading in obese adults with knee osteoarthritis. *Osteoarthritis Cartilage* 2011;19(7):822-828. [Link](#)
4. Lambova SN. Knee osteoarthritis: emphasis on metabolic type. *Life* 2023. [Link](#)
5. Khor A et al. Diabetes mellitus is not a risk factor for osteoarthritis. *RMD Open* 2020. [Link](#)
6. Veronese N et al. Adherence to a Mediterranean diet is associated with lower prevalence of osteoarthritis. *Clin Nutr* 2017. [Link](#)
7. Veronese N et al. Mediterranean diet and knee osteoarthritis outcomes: a longitudinal cohort study. *Clin Nutr* 2019. [Link](#)
8. NICE NG226 glucosamine recommendation, summarised in NHS Dorset briefing. [Link](#)
9. RACGP Guideline for the management of knee and hip osteoarthritis: nutraceuticals. [Link](#)
10. Huang CH et al. Effect of knee extensor power on knee pain in adults with or at risk for osteoarthritis: MOST. *J Rheumatol* 2025. [Link](#)
11. Culvenor AG, Segal NA, Guermazi A, Roemer F, Felson DT, Nevitt MC, Lewis CE, Stefanik JJ. Sex-specific influence of quadriceps weakness on worsening patellofemoral and tibiofemoral cartilage damage: a prospective cohort study. *Arthritis Care Res* 2019;71(10):1360-1365. [Link](#)
12. Gheidi N, Kiminski R, Besch M, Ristow A, Wallace B, Kernozek T. Patellofemoral joint stress during front and back squats at two depths. *Applied Sciences* 2025;15(16):8784. [Link](#)
13. Escamilla RF et al. Patellofemoral joint force and stress between a short- and long-step forward lunge. *J Orthop Sports Phys Ther* 2008. [Link](#)
14. Schoenfeld B, Williams M. Are deep squats a safe and viable exercise? *Strength Cond J* 2012. [Link](#)
15. Verbeek J et al. Occupational exposure to knee loading and the risk of osteoarthritis of the knee: dose-response meta-analysis. *Saf Health Work* 2017. [Link](#)
16. Wang X et al. Occupational risk in knee osteoarthritis: systematic review and meta-analysis. *Arthritis Care Res* 2020. [Link](#)
17. Alentorn-Geli E et al. The association of recreational and competitive running with hip and knee osteoarthritis. *J Orthop Sports Phys Ther* 2017. [Link](#)
18. Bowen L et al. Spikes in acute:chronic workload ratio associated with a 5 to 7 times greater injury rate in English Premier League football players. *Br J Sports Med* 2020. [Link](#)
19. Tee J. The acute:chronic workload ratio: science or religion? *Sports Injury Bulletin*. [Link](#)
20. McLean SG, Samorezov JE. Fatigue-induced ACL injury risk stems from a degradation in central control. *Med Sci Sports Exerc* 2009. [Link](#)
21. Woddillah NA et al. Fatigue effect on landing biomechanics after ACL reconstruction: systematic review. *JUMMEC* 2023. [Link](#)
22. Lo GH et al. Association between walking for exercise and symptomatic and structural progression in knee osteoarthritis. *Arthritis Rheumatol* 2022. [Link](#)
23. Graham BT et al. Detrimental effects of long sedentary bouts on the biomechanical response of cartilage to sliding. *Connect Tissue Res* 2020. [Link](#)
24. Voinier D. The effects of physical activity on structural worsening of knee osteoarthritis. University of Delaware 2021. [Link](#)
25. Watson A et al. Sleep and in-season injury in collegiate athletes. *AMSSM* 2019. [Link](#)
26. Milewski MD, Skaggs DL, Bishop GA, Pace JL, Ibrahim DA, Wren TA, Barzdukas A. Chronic lack of sleep is associated with increased sports injuries in adolescent athletes. *J Pediatr Orthop* 2014;34(2):129-133. Linked page is Watson 2020, which cites it. [Link](#)
27. de Jonge J et al. Sleep and injury in recreational runners. *Applied Sciences* 2025. [Link](#)
28. Campbell CM et al. Sleep, pain catastrophizing and central sensitization in knee osteoarthritis patients with and without insomnia. *Arthritis Care Res* 2015. [Link](#)

29. Felson DT, Zhang Y. Smoking and osteoarthritis: a review of the evidence and its implications. *Osteoarthritis Cartilage* 2015. [Link](#)
30. Amin S et al. Cigarette smoking and the risk for cartilage loss and knee pain in men with knee osteoarthritis. *Ann Rheum Dis* 2007. [Link](#)
31. Shen Z et al. Association of smoking with cartilage loss of knee osteoarthritis: data from two longitudinal cohorts. *BMC Musculoskelet Disord* 2023. [Link](#)
32. Ibrahim A, Al-Musabi M, Kabariti R, Kempe-Gowda S, Wade R. The impact of smoking on outcomes following anterior cruciate ligament reconstruction: a systematic review and meta-analysis. *Cureus* 2025;17(11):e96765. [Link](#)
33. Novikov DA, Swensen S, Buza JA, Gidumal RH, Strauss EJ. The effect of smoking on ACL reconstruction: a systematic review. *Phys Sportsmed* 2016;44(4). [Link](#)
34. Kim SJ et al. Effect of cigarette smoking on the clinical outcomes of ACL reconstruction. *J Bone Joint Surg Am* 2014. [Link](#)
35. Xiong A et al. Effects of active vitamin D analogues on muscle strength and falls in elderly people: an updated meta-analysis. *Front Endocrinol* 2024. [Link](#)
36. Wei FL, Li T, Gao QY, Huang Y, Zhou CP, Wang W, Qian JX. Association between vitamin D supplementation and fall prevention. *Front Endocrinol* 2022;13:919839. [Link](#)
37. Wang R et al. Relationship between 25-hydroxy vitamin D and knee osteoarthritis: systematic review and meta-analysis of RCTs. *Front Med* 2023. [Link](#)
38. The effect of vitamin D supplementation on knee osteoarthritis: a meta-analysis of randomized controlled trials. *Int J Surg* 2017 (four RCTs, 1,136 patients). [Link](#)
39. Lin CR et al. Analgesic efficacy of collagen peptide in knee osteoarthritis: a meta-analysis of randomized controlled trials. *J Orthop Surg Res* 2023. [Link](#)
40. Zdzieblik D et al. Collagen peptide supplementation in combination with resistance training improves body composition and increases muscle strength in elderly sarcopenic men. *Br J Nutr* 2015. [Link](#)
41. Jendricke P et al. Specific collagen peptides in combination with resistance training improve body composition and regional muscle strength in premenopausal women. *Nutrients* 2019. [Link](#)
42. Shaw G, Lee-Barthel A, Ross MLR, Wang B, Baar K. Vitamin C-enriched gelatin supplementation before intermittent activity augments collagen synthesis. *Am J Clin Nutr* 2017. [Link](#)