

Spinal health and common sense

Nick Kruger 2026

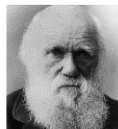


Ageing spines are part of the human condition and most of us can expect spinal pain during our lifetimes. Thankfully, these episodes are mostly self-limiting, and the vast majority (90%) will improve over 6-8 weeks.

So why do we start getting spinal pain in our early to Middle Ages: Is there a design fault that culminates in back and neck pain in adults who are entering the prime of their productive lives? Humans expect to live to 8 decades, and it is decidedly inconvenient that our spines are so poorly designed that they fail in our 40's.

Evolution has not missed a trick. Our spines are very well designed to give service for roughly 35 years, thereafter, we are redundant in terms of our species as longevity decreases evolvability. Humans are adult at 18 and able to raise a family. Once our offspring are mature, parents, having served their purpose, become non-essential in evolutionary terms. This occurs at around 35-40 years of age, which, unsurprisingly, is when our backs and neck tend to give trouble.

Average human designed to last about 35 years



Human longevity is due to advancements in civilization. Whilst we can prevent ourselves from dying due to exposure, starvation, predators and medical conditions, we become burdened with an ageing spinal column, which, having done its duty perfectly well in getting us to adulthood, now finds itself past its sell-by date.

Human sell-by date

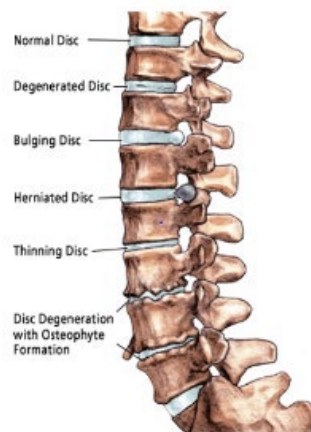


2026 update: The science of measuring “biological age” — as distinct from the birthday-candle version — has moved fast in the last few years. A 2026 study in Nature Biotechnology used six independent “proteomic ageing clocks” (models trained on patterns of blood proteins) to track how quickly people were biologically ageing during a 12-week drug trial for lung fibrosis. All six clocks agreed that the trial drug measurably slowed the pace of biological ageing, apparently by damping down cellular senescence and growth-factor signalling — the same pathways targeted by caloric restriction and drugs like rapamycin. None of this rewrites the mechanical reality of a worn disc or a

stiff facet joint, but it's a reminder that the broader “ageing” process our joints are riding on top of may be more measurable, and more malleable, than a fixed 35-year design life suggests.

Zhavoronkov, A. et al. *Integration of proteomic aging clocks in a phase 2a clinical trial supports simultaneous geroprotective assessment.* *Nature Biotechnology* (2026). doi:10.1038/s41587-026-03286-y

It is highly inconvenient then that our spines start to make their presence felt once we are in the prime of our productive lives and careers; but this is a normal part of the ageing process. Normally, spinal pain peaks in the middle decades and starts to settle after 50-60 years of age. It is interesting that our pain improves as we age, given that the degeneration tends to worsen. Degeneration is the process of spinal deterioration: joint spaces narrow as cartilage thins, discs soften, lose their hydration and the powerful disc annular ligaments may crack, fissure and bulge out as the structures age. Osteophyte formation occurs and the spinal column stiffens up. On occasion it may even become rigid with bony ankylosis across vertebral segments.



Episodic spinal pain is the frequent symptom in this process and a wide variety of health care practitioners are consulted with an equally diverse array of remedies for spinal ailments. Often the treatment may culminate in an x-ray report with scary terms such as degeneration, spondylolisthesis, osteoarthritis and osteophytes.

Spinal degeneration is part of being human



Very few remedies are proven scientifically to work, and most spinal pain tends to improve in line with natural history-spontaneous improvement in 6-8 weeks, irrespective of type of treatment. The diverse group of non-interventional spinal pain practitioners, don't particularly have more success over the natural history of benign spinal pain, but they do provide reassurance, comfort and help manage symptoms while the pain subsides naturally.

Non-interventional Treatments

Medication- NSAIDS, Paracetamol, Opiates, Tricyclics, Muscle relaxants

Physiotherapy

Chiropractic

Acupuncture

Spinologists

Massage

Aromatherapy

Exercise

Eur Spine J (2006) 15 (Suppl. 2): S136–S168
DOI 10.1007/s00586-006-1070-3

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Chapter 2 **European guidelines** **for prevention in low back pain** **November 2004**



Looking at all the available evidence, the European working group for prevention and management of lower back pain (LBP) reviewed all publications on the subject.

They found: Limited evidence for preventative treatments in LBP

Some evidence for exercise and appropriate biopsychosocial education

Individuals reported various anecdotal strategies, but this lacked scientific evidence

Other findings from the working group were:

- Traditional back schools giving biomechanical information, advice and instruction are not recommended for prevention LBP
- Lumbar supports / Shoe insoles/ specific chairs or mattress are not recommended
- No evidence for manipulative treatment for prevention LBP
- Leg-length adjustments no evidence
- Physical ergonomics for prevention in LBP no evidence

Regarding exercise: the European working group found

- Physical exercise is recommended to prevent work absenteeism due to back pain
- Insufficient evidence to recommend for or against any specific kind of exercise, or the frequency/intensity of training

Where my view of the management of spinal pain tends to diverge from mainstream, is the notion that somehow, we can improve a normally degenerating system of complex joints by moving them and strengthening the muscles supporting them. There is a perception that somehow, the spine needs to remain supple, needs to be supported by the ever popular "core-muscles" and the reason the patient has developed spinal pain is because they are out of balance, their core-muscle support is deficient, and they don't have the proper posture. This has no scientific basis, and some publications are critical of this thinking.

Core Muscles Assumptions

Certain muscles are more important for stabilisation

Weak abdominal muscles lead to back pain

Strengthening abdominal or trunk muscles can reduce back pain

Unique group of “core” muscle working independently of other trunk muscles

Back pain can be improved by normalising the timing of core muscles

Relationship between stability and back pain



I see a select group of patients who unwillingly find themselves at the surgeon when all other modalities have failed, and despite rigorous rehabilitation, remain symptomatic and painful. Unfortunately, surgery is almost never an option in non-specific degenerative spinal pain and my advice is often surprising to patients.

In orthopaedics, an osteoarthritic joint (painful joint) is not managed with prescription of movement of the joint or muscle strengthening. Instead, it is rested, splinted, fused or replaced (arthroplasty) as is commonplace with knee or hip joints. A joint fusion permanently stiffens the joint and was the best option before the advent of joint replacements. A stiff knee is particularly cumbersome getting in and out of cars, but is very functional, enduring and painless. Arthritic finger joints are painful while still mobile, but as the fingers become increasingly stiff, the pain settles. A joint, like a wheel-bearing, cannot un-degenerate itself with movement and stretching and muscle training. These modalities cannot prevent the onset of arthritis either.



I see many top fitness-instructors, cross-fit champions, bodybuilders, yoga, Pilates instructors as well as elite athletes, where muscle strength and conditioning are in peak condition, but they suffer from spinal pain and have advanced degeneration and spinal joint osteoarthritis.

Years of "core strengthening"



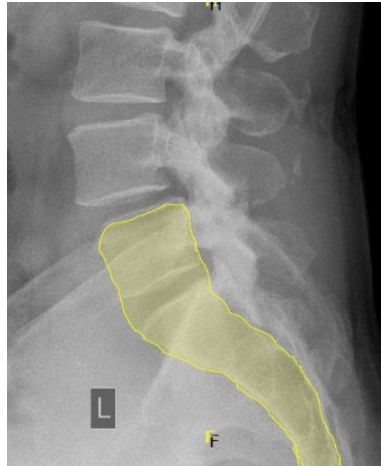
There are no programs for osteoarthritic knees having quads and hamstrings strengthened to make them less painful, yet against all known orthopaedic principles for degenerate joints, we manage spinal joints with muscle strengthening and movement. Luckily, spinal pain tends to improve irrespective of treatments, and as per the natural history of the condition, mostly resolves in 6-8 weeks.

Where my advice applies, is the patient who fails to improve despite the normal advice and treatment. Axial spinal pain improves after our middle decades, mostly as we become more sedentary, equating to less strain on ageing joints. Additionally, our spines stiffen up from joint osteophytes, ossification of ligaments, intervertebral discs becoming hardened and less viscoelastic. Functional loss is minimal as mostly bending occurs at the hips anyhow.

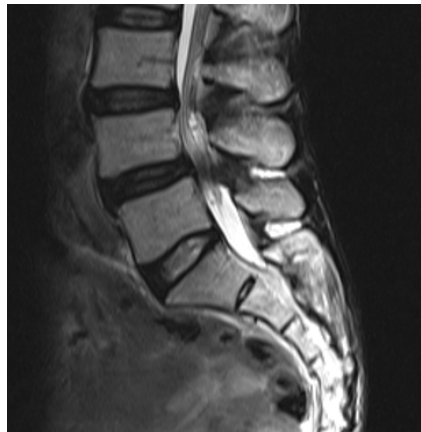
A good example of this is ankylosing spondylitis (an inflammatory disease) which is painful during the joint destruction period, but once the joints stiffen and auto-fuse (ankylosis), the spine becomes rigid but painless. Spinal fusions are based on removing motion between spinal segments to relieve pain. So, as we become less flexible, pain mostly subsides.



Do spinal disks need movement and loading to be healthy? On face value, one assumes yes, but like a car tyre, intervertebral discs degenerate (wear out) almost proportionally to the amount of loading they endure. Whilst they are designed well enough to last the prerequisite 35 years we evolved for, they become problematic when we age further. High-impact sportsmen such as rugby players, have marked degenerative changes in their spines, in-spite of massive "core muscles" and strength training.



A great example of how movement and loading inevitably cause disc degeneration, is the patient with a transitional L5 vertebra-where the L5 is partially sacralised and not fully separated from the sacrum, the L5/S1 disc does not experience the same loading as when the L5 segment is mobile. The L5/S1 disc shows no degeneration on MRI, while the longer stiffer segment including L5/S1, now expose the L4/5 segment to greater mechanical forces with subsequent accelerated degeneration. This is often the reason for L4/5 level having a slip (spondylolisthesis) over a pristine L5/S1 disc.



My somewhat controversial advice is to follow nature's lead and encourage arthritic spinal joints to stiffen up and immobilize. To this effect, I don't advise any trunk exercises, stretching or loading activities. Even isometric exercises load and unload the spinal column which is contrary to the desired goal- of allowing arthritic joints to stiffen and become less inflamed. The spine does not actually become "stiff" if one is not doing stretching exercises, any different than your knees not becoming stiff and rigid from not doing stretching exercises; normal daily activity is sufficient.

Far from avoiding exercise, I am a firm advocate of exercise to manage spinal pain, however it should be exercise that does not load or move the vertebral column unnecessarily. To this purpose, cardiovascular exercise is the best option and has more effects than simply strengthening and conditioning muscle. Exercise produces endorphins which are major factors in mood elevation, pain modulation and well-being, but exercise also influences the immune system.

Endorphins and exercise

β-Endorphin Response to Exercise
An Update
Allen R. Goldberg and Alexander Z. Janicak
Exercise and Sport Sciences Department, University of North Carolina - Charlotte, Charlotte, NC

Exercise of sufficient intensity and duration increases circulating β-endorphin levels

A critical intensity of exercise **>60% maximum oxygen uptake** (VO₂max) needed to increase circulating p-endorphin levels

American College of Sports Medicine

Exercise at 60 percent of **VO₂ max** corresponds with a **heart rate** that is 75 percent of **maximum**

Exercise at 80 percent of **VO₂ max** corresponds with 88 percent of **maximum heart rate**

(MMR= 220- age)

Roughly 120 beats/min

Hypertension and diabetes are some of the important metabolic conditions responding positively to regular cardio. The spine is affected by similar processes as the cardiovascular system and is a marker of systemic health. In clinical obesity, adipose tissue has been shown to produce adipokines, which are a type of inflammatory cytokine implicated in disc degeneration, osteoarthritis and cardiovascular disease.



Cytokine & Growth Factor Reviews
Volume 44, December 2018, Pages 38–50



Pro-inflammatory cytokines: The link between obesity and osteoarthritis

Tiantian Wang ^{a, b}, Chengqi He ^{a, b}



The Spine Journal
Volume 18, Issue 11, November 2018, Pages 2140–2151



Review Article

The association between pro-inflammatory biomarkers and nonspecific low back pain: a systematic review ☆

R. van den Berg ^{a, b}, E.M. Jongbloed ^a, E.I.T. de Schepper ^a, S.M.A. Bierma-Zeinstra ^{a, b}, B.W. Koes ^a, P.A.J. Luijsterburg ^a

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2026 update: The same 2026 trial adds some interesting texture here. Gene-set analysis showed the drug suppressed a broad panel of circulating senescence markers — interleukins such as IL-6 and IL-1β, several matrix metalloproteinases (MMPs), and related chemokines — while boosting markers of senescence reversal. These are largely the same molecules already implicated in the adipokine-driven disc degeneration and osteoarthritis story above: cellular senescence, obesity-related inflammation and joint degeneration seem to share a common inflammatory vocabulary. The same paper also notes that the drug's molecular target, an enzyme called TNIK, has separately been shown to help prevent diet-induced obesity in mice, and that the growth-factor and nutrient-sensing pathways it suppresses are the same pathways dialled down by caloric restriction. None of this is

spine-specific evidence — it's a lung fibrosis trial — but it's a plausible mechanistic thread linking weight management to the broader ageing/inflammation picture above.

Same source as above; see also Pham, T.C.P. et al. TNIK is a conserved regulator of glucose and lipid metabolism in obesity. Science Advances 9, eadf7119 (2023).

On exercise, the same paper's introduction cites a 12-week supervised exercise programme in 26 men that produced a roughly ten-month reduction in “biological age” on one proteomic clock — a molecular line of evidence for regular cardio, distinct from (but pointing the same way as) the endorphin story above. A separate 40-month primate study found the diabetes drug metformin has a similar decelerating effect on ageing clocks across multiple tissues, which fits neatly with the hypertension/diabetes point already made above: the metabolic and the skeletal appear to age together.

Lee-Ødegård, S. et al. Reversal of proteomic aging with exercise—results from the UK biobank and a 12-week intervention study. NPJ Aging 12, 19 (2025); Yang, Y. et al. Metformin decelerates aging clock in male monkeys. Cell 187, 6358–6378 (2024).

One factor missing from this discussion so far is smoking. It is one of the best-replicated risk factors for disc degeneration in the literature — the classic 1991 twin study found 18% greater disc degeneration scores in smoking twins compared with their non-smoking identical siblings, with the effect spread across the whole lumbar spine rather than one level, pointing to a systemic rather than purely local mechanism. Smoking is also one of the more reliable ways to add years to your biological age on modern DNA-methylation clocks — a 2026 analysis found current smokers averaging roughly nine years of extra biological age compared with people who had never smoked, though former smokers showed a smaller gap that kept shrinking the longer they had quit. If weight management and exercise turn the ageing dial back, smoking is one of the most reliable ways to turn it forward.

Battié, M.C. et al. 1991 Volvo Award in Clinical Sciences: Smoking and lumbar intervertebral disc degeneration: an MRI study of identical twins. Spine 16, 1015–1021 (1991); Epigenetic Age Acceleration by Smoking: Evidence for Dose-Response and Recovery After Cessation. Am J Respir Crit Care Med 212 (Suppl) (2026), ATS conference abstract.

A weight loss program is probably the most understated form of treatment for backpain and is unfortunately very difficult with our westernized diet of cheap carbohydrates. My personal advice would be the 'South-Beach' diet, which seems to be more sustainable.

Exercise for life

Endorphins

Weight loss ·

Metabolic control

Immune system modulation

So, for the patient with mechanical spinal pain, regular non-impact cardio is the best advice I have. Spinning and swimming are two exercises safe for the painful spine, but the specific exercise is not particularly important as long there is no pain during or after the activity. More importantly, is getting your cardiac rate over 80% of your predicted rate ($220 - \text{age}$ is max predicted for age), this being the range where endorphin production lies and where the beneficial effects of exercise are found. Other than that, just let your back be.



No core, planking, sit-ups, no stretches or spinal loading. These are unnecessary for prevention of spinal pain and don't have any advantage for the management of acute pain. It is my opinion that these activities may even aggravate spinal pain by causing loading and movement of stiff degenerate segments that are best left alone.

Exercise recommendations

Non-impact cardio

Listen to your back

Exercise of your choice- sustainable (MHR >60% of Vmax)

No stretching

No core

(Note: These are my personal opinions based on years of management of spinal patients who have failed conventional treatment, and a thorough understanding of spinal biomechanics. There is some literature support for my concepts, but ultimately the evidence is mostly anecdotal, note that there is also no good evidence for any other)

The teal passages above are a September 2026 addition drawing on Zhavoronkov et al., Nature Biotechnology (2026) and the smoking/disc-degeneration and epigenetic-ageing literature cited alongside them — review and edit as you see fit.

Artificial Intelligence and new evidence for rate of biological ageing.

Proteomic aging clocks are predictive, machine-learning algorithms that estimate a person's **biological age**—how well their body and cellular systems are functioning—by measuring the concentrations and patterns of thousands of circulating proteins in a small blood sample.

Unlike **chronological age** (the number of years since birth), biological age reflects internal physiological wear and tear, susceptibility to disease, and overall health span.

Accelerated proteomic aging strongly correlates with higher future risks of major chronic conditions, including cardiovascular disease, neurodegenerative disorders, metabolic disease, and overall mortality.

Modifiable lifestyle factors—such as physical activity, smoking cessation, and diet—measurably influence proteomic clock scores- which ties in with my anecdotal advice above.

So, the key point is, your spinal health is a very good indicator of systemic health and if your body is healthy, so is your spine.

SUMMARY

DO- eat healthy, eat less (South Beach Diet). Keep your weight low (BMI around 25). I support glucagon-like peptide-1 (GLP-1) receptor agonists if available.

DO exercise- regular non-impact cardio (Swimming/Spinning/Cycling/Orbitrek). HR>120/min 30min daily

DONT smoke/vape/eCig- ever

DON'T do “core”, planking, sit-ups or weight training if you have a bad back or neck. If you particularly want to strengthen arms or legs, isolate those muscles on machines, not free weights.