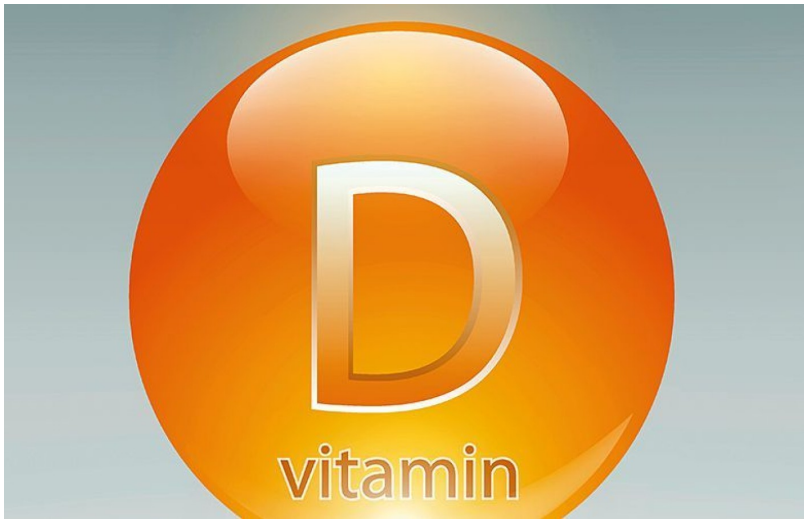
Dynamic Chiropractic



VITAMINS / SUPPLEMENTS

Vitamin D Fails to Help Knee OA? The Proper Perspective

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Author's Note: The authors in this study delivered just 50,000 IU of vitamin D per month, rather than the more customary 50,000 IU per week dosing. This means subjects received only 1,667 IU per day, rather than 7,000 IU daily, as I mistakenly wrote in the print version of this article. Such a small dose makes the study and conclusion even less relevant, and should be taken with a grain of salt.

The March 8, 2016 issue of *JAMA* includes a study about vitamin D supplementation for osteoarthritis of the knee.[1] This is a really weird study. Here is the conclusion that should be ignored by any thinking physician: "These findings do not support the use of vitamin D supplementation for preventing tibial cartilage loss or improving WOMAC knee pain in patients with knee osteoarthritis."

I remain surprised by how smart people can perform foolish studies that are approved for publication by other smart people. There were 413 subjects in the study. The average age was 63 and the average BMI was 29.6. It's important to keep in mind that a BMI of 30 is considered obese, so this means the study was done on deconditioned old people who were "almost" obese. And somehow these researchers believed supplementing with vitamin D could overcome inflammatory joint changes associated with aging and obesity?

They used 50,000 IU per month, which amounts to a little over 1,667 IU per day – not much. Keep in mind that if you were to take 10,000 IU daily, this amounts to just ¼ of 1 mg or 250 mcg daily.

The average vitamin D level of subjects in the vitamin D and placebo groups was 17.6 ng/mL, which is considered deficient based on an accepted normal range of 32-100 ng/mL. Vitamin D researchers contend 40 ng/mL or more should be the goal, which means this population was obviously deficient.

Shockingly, in this "study" the sign of supplementation success in the vitamin D-supplemented group was reaching at least 24 ng/mL, which is still deficient by current laboratory standards. In other words, it was a "success" to be deficient and a "failure" to respond positively to a slightly higher deficient state, which apparently means vitamin D supplementation is not indicated in knee OA. I do not think I could have made up this nonsense if I'd tried.

OA and Chronic Inflammation: The Role of Dietary Omega-6

It seems to me that the authors of this study may not understand the nature of osteoarthritis, which is not a surprise. In part, the expression of OA is based on the accumulation of dietary omega-6 arachidonic acid in joint cartilage. As early as 1975, researchers identified that articular cartilage markedly accumulates dietary omega-6 arachidonic acid as we age.[2]

Important to understand is that healthy young joints are absolutely free of omega-6 fatty acids. The only way to accumulate arachidonic acid in tissues is by overeating omega-6 fatty acids. French fries, potato chips and corn chips are examples of calorie sources oozing with omega-6 fatty acids. This means we eat ourselves into an osteoarthritic state. Indeed, we now know that "lipid deposition in the joint is observed at the early stages of osteoarthritis (OA) before the occurrence of histological [or symptomatic] changes."[3]

Subsequently, normal loading can lead to a low-grade inflammation and joint-tissue degradation that, years later, is misperceived as a wear-and-tear condition. Rather, osteoarthritis more commonly manifests as a state of "wear lack of repair" due to chronic inflammation.

The mediators that promote joint degradation have been identified:[4] "Chondrocytes produce cytokines, chemokines, alarmins, prostanoids, and adipokines and express numerous cell surface receptors for cytokines and chemokines, as well as Toll-like receptors. These receptors activate intracellular signaling pathways involved in inflammatory and stress responses of chondrocytes in OA joints."

The above quote should give us pause and cause us to rethink how we view joints. Most DCs have been taught chondrocytes produce cartilage, which is true; however, it is our chondrocytes that become pro-inflammatory and release inflammatory mediators that cause the cartilage degradation.

Other Contributing Factors

Bear in mind it is not just dietary omega-6 fatty acids that have been implicated in the development of osteoarthritis. Research also has implicated hyperglycemia as destructive to joints. Hyperglycemia and advanced glycation end products stimulate inflammatory and degradative processes in chondrocytes.[5-6]

In short, the inflammatory chemistry of the metabolic syndrome, diabetes and heart disease is the same chemistry associated with the expression of osteoarthritis. In 2005, researchers used the following title for their paper:[7] "Is Progressive Osteoarthritis an Atheromatous Vascular Disease?" We now know the answer is probably yes.[2-6]

This explanation about the inflammatory chemistry of osteoarthritis is not meant to imply joint trauma does not evolve into osteoarthritis. This can and does happen, of course, and this is a separate issue. The greater issue is that osteoarthritis commonly develops in joints that have not been traumatized, which is known to be promoted by a low-grade, chronic inflammatory state caused by a pro-inflammatory diet that contains an excess of sugar, flour and refined omega-6 oils (which I've labeled "dietary crack").

How We Can Help Patients

So, the notion a monotherapy like vitamin D can substantially influence OA is highly suspect. Even more unbelievable is expecting that increasing serum vitamin D to a clinically ineffectual level will lead to measurable improvements ... and then claiming vitamin D has no benefit. The *JAMA* study was doomed to fail from the beginning.

There may never be definitive information to guide the treatment of OA, but we must still act. The best evidence suggests we should "deflame" patients so they no longer manifest the metabolic syndrome. This involves the avoidance of dietary crack and supplementing with glucosamine / chondroitin, vitamin D, ginger / turmeric, magnesium, and omega-3 fatty acids.

References

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