

What Do Dysmenorrhea and Osteoarthritis Have in Common? by Dr. David Seaman

While we typically do not consider **dysmenorrhea** and **osteoarthritis** to be similar conditions, a quick look at the chemistry suggests otherwise. The pain associated with each condition is treated with nonsteroidal anti-inflammatory drugs (NSAIDs), which has allowed for the conclusion that each condition is mediated by excessive pro-inflammatory prostaglandin production.¹⁻² While the complexity of each disease can be describe for long hours by specialists and researchers, the clinical fact remains that the cornerstone of medical treatment for each condition is anti-inflammatory drugs. As NSAIDs are associated with unwanted side effects, patients may seek your advice regarding natural and safer alternatives to help with these conditions.

My suggestion? View dysmenorrhea and osteoarthritis as the same "chemical condition," i.e., pro-inflammatory prostaglandin excess, but in a different anatomical location.

Diet for Pro-Inflammatory Prostaglandin Excess

Dietary omega-6 fatty acids, specifically arachidonic acid, are converted into pro-inflammatory prostaglandins, such as PGE-2 and PGF-2, which cause the pain of osteoarthritis and dysmenorrhea. So, if we avoid excess omega-6 fatty acids, we avoid excess production of pro-inflammatory prostaglandins and the expression of these painful conditions.

We should avoid fast foods and packaged foods because they contain excess omega-6 fatty acids. Specifically, we should avoid corn, safflower, sunflower, cottonseed, and soybean oils. To make life easier, we should only use olive oil, butter and coconut oil.

From a food perspective, we should eat wild-caught fish, **omega-3**-fed chicken, grass-fed meat, wild game, and lean meats. Vegetables and fruit should be consumed in abundance – at least 1-2 pounds per day for the average person. If one is still hungry, a small handful of nuts should be consumed. Hemp, chia and flaxseeds can also be consumed, as they have excellent omega-6:omega-3 ratios. Condiment portions of other natural, whole foods can be consumed, such as grains, legumes and dairy.

Flour products should be avoided. If you absolutely must consume flour products, learn to bake with coconut flour.

When eating in the fashion described, the diet is characterized as being "anti-inflammatory."³ Several excellent articles discuss anti-inflammatory eating; free full-text versions are available via PubMed.gov.⁴⁻⁷ I suggest making these available for your patients, rather than trying to explain things during an office visit. A patient who remains unmotivated after reading these articles is unlikely to ever be motivated.

Smart Supplementation

NSAIDs inhibit the cyclooxygenase (COX) enzyme that converts dietary omega-6 and omega-3 fatty acids into either pro- or anti-inflammatory prostaglandins. We should view COX as an enzyme that we "feed." The dietary approach to feeding COX the proper balance of omega-6 and -3 fatty acids was described earlier. We should also feed COX the omega-3 fatty acids in fish-oil supplements. The typical recommendation is about 1-3 grams per day of eicosapentaenoic and docosahexaenoic acids.⁸

Various botanicals have the ability to inhibit the COX enzyme in a fashion similar to NSAIDs and with a better safety profile. Ginger, turmeric and boswellia have been the most popular over the years and can be taken long term with minimal to no side effects in most individuals.⁹⁻¹² The typical recommendation is 1-2 grams per day, which can be taken in an ongoing manner to modulate chronic inflammation.

To further augment anti-inflammatory activities, supplementation with vitamin D and magnesium is a reasonable consideration, as they each influence several inflammatory signaling pathways. Vitamin D should be supplemented to achieve adequate blood levels of 25(OH)D, which is typically about 4,000 IU or more per day. The typical supplemental recommendation for magnesium is 400 mg per day.

While experimental evidence is lacking, clinical experience suggests when the above interventions are applied to patients with osteoarthritis, the outcome with supplemental glucosamine and chondroitin is more rewarding compared to using them in a monotherapy fashion. Otherwise, we are simply providing a couple of grams of a joint-tissue supplement to an overly **inflamed body** and should not be surprised by mediocre results.

After spending years looking into the chemistry of most common conditions that afflict us, the obvious fact that has spoken to me is that the same inflammatory chemistry is at work. Consequently, dietary and supplement interventions should be very similar from person to person, no matter the presenting condition.

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