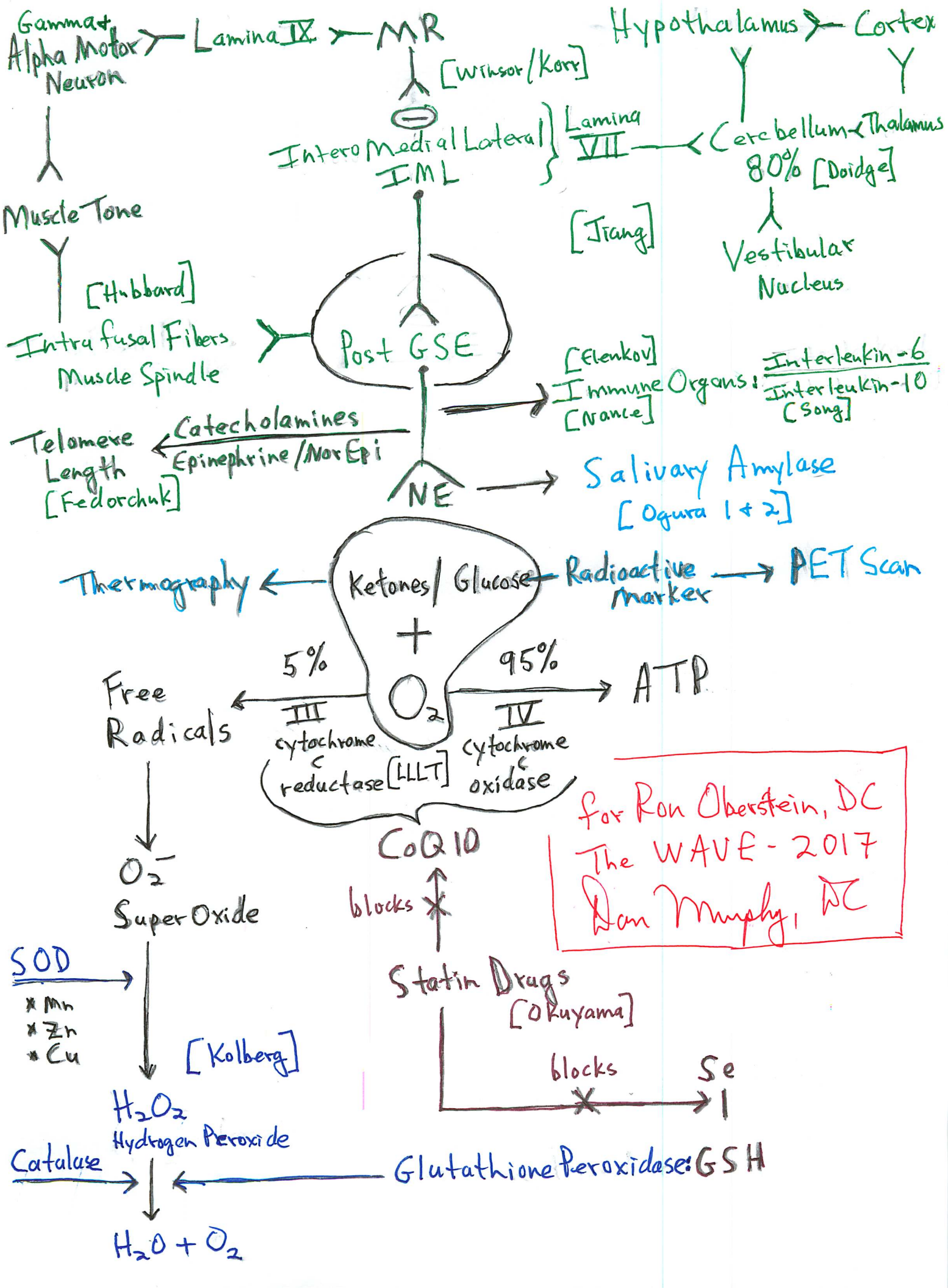
ChiroHealthUSA

Pain Webinar
September 2017

Dan Murphy, DC


Inflammation
Fibrosis


Gamma Alpha Motor Neuron
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[Jiang]
Vestibular Nucleus
Muscle Tone
[Hubbard]
Intra fusal Fibers Muscle Spindle
Post GSE
[Elenkov]
Immune Organs: Interleukin-6 / Interleukin-10 [Song]
[Nance]
Telomere Length [Fedorchuk]
Catecholamines
Epinephrine/NorEpi
NE
Salivary Amylase [Ogura 1 + 2]
Thermography
Ketones/Glucose
Radioactive Marker
PET Scan
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95%
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III cytochrome c reductase [LLT]
IV cytochrome c oxidase
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Super Oxide
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* Zn
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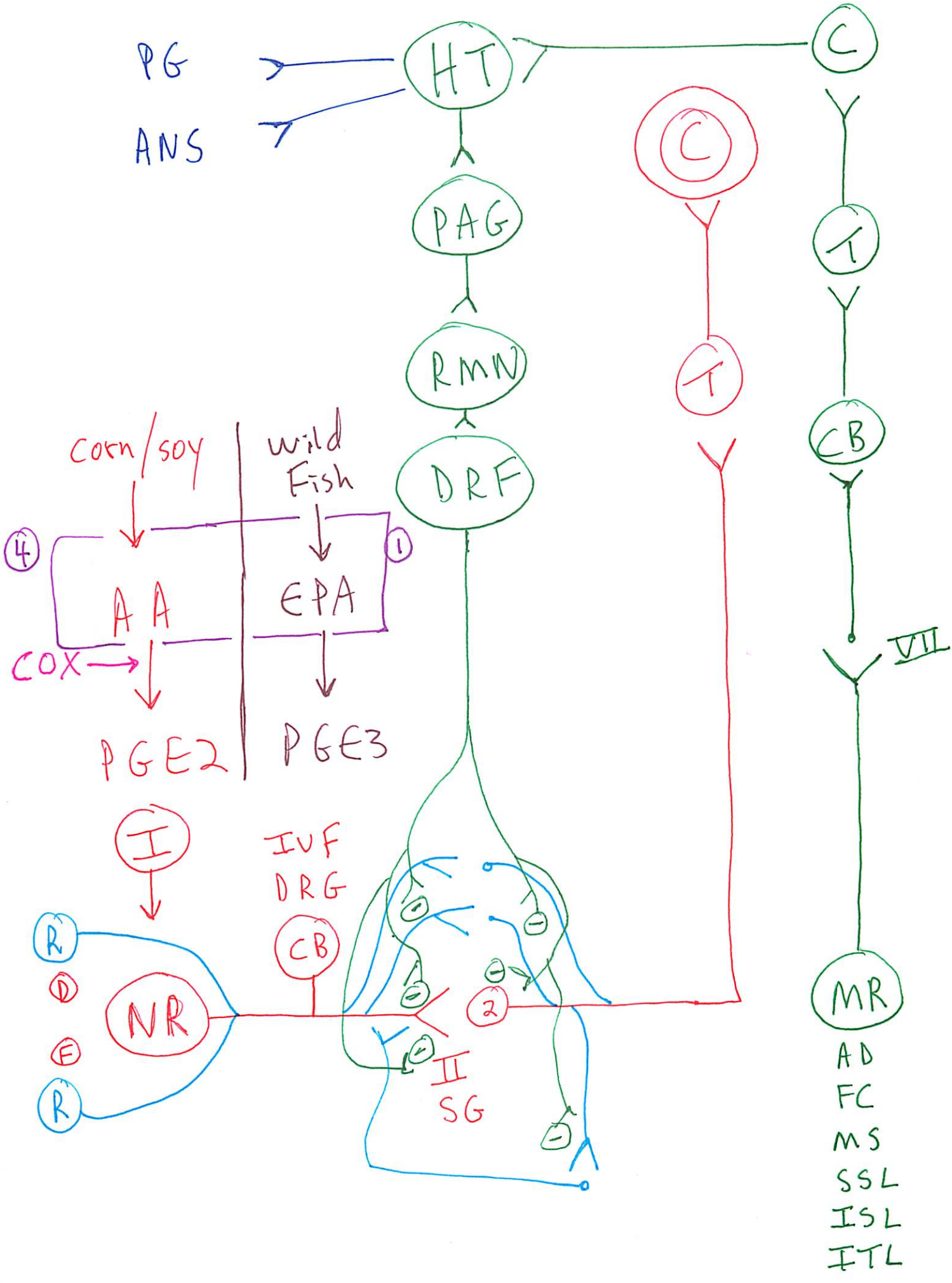
for Ron Oberstein, DC
The WAVE - 2017
Don Murphy, DC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On Cervical Zygapophysial Joint Pain After Whiplash

Spine
December 1, 2011; Volume 36, Number 25S, pp S194–S199

Nikolai Bogduk, MD, PhD

FROM ABSTRACT
Objective
To summarize the evidence that implicates the cervical zygapophysial joints as the leading source of chronic neck pain after whiplash.

Summary of Background Data
Reputedly a patho-anatomic basis for neck pain after whiplash has been elusive. However, studies conducted in a variety of disparate disciplines indicate that this is not necessarily the case.

There is convergent validity from (1) whiplash postmortem studies, (2) whiplash biomechanics studies, and (3) whiplash clinical studies indicating that the primary source of chronic whiplash pain is injuries to the cervical zygapophysial joints

Results
1) Postmortem studies show that a spectrum of injuries can befall the zygapophysial joints in motor vehicle accidents.

2) Biomechanics studies of normal volunteers and of cadavers reveal the mechanisms by which the zygapophysial joints can sustain injury during whiplash.

3) Whiplash studies in cadavers and laboratory animals have produced zygapophysial joints injuries.

4) Clinical studies have shown that zygapophysial joint pain is very common among patients with chronic neck pain after whiplash, and that this pain can be successfully eliminated by radiofrequency neurotomy.

Conclusion
The fact that multiple lines of evidence, using independent techniques, consistently implicate the cervical zygapophysial joints as a site of injury and source of pain, strongly implicates injury to these joints as a common basis for chronic neck pain after whiplash.

DR. BOGDUK ALSO NOTES:

1) "Convergent validity arises when multiple, independent approaches point to the same conclusion," which allows for greater confidence in the conclusion.

2) In whiplash, four convergent lines of evidence "implicate the cervical zygapophysial joints as the leading source of pain in patients with chronic whiplash-associated disorder."

3) To prepare this paper, Dr. Bogduk used only original data on the topic from his personal library and by searching PubMed with the terms whiplash, neck pain, zygapophysial joint, and injury:

The **postmortem studies** identified a variety of nonlethal injuries:

A) Nerve-root lesions
B) Rim-lesions to the intervertebral discs (the disc is traumatically separated from the cartilaginous end-plate of the vertebral body)
C) Intraarticular hemorrhages
D) Fractures of the facet articular cartilage
E) Fractures of the facet subchondral bone
F) Fractures of the entire facet articular processes

Virtually none of these lesions were seen on postmortem radiography. **[Important]**

"Medical imaging in vivo may fail to identify lesions that are definitely present at postmortem. Consequently, in the context of whiplash injury, normal radiographs, or even normal magnetic resonance imaging, do not mean that the patient has no lesion."

The **human biomechanics studies** identified these injurious mechanisms:

A) The cervical spine undergoes a "highly abnormal" "S" shaped deformation with extension of the lower cervical spine and flexion of the upper cervical spine.

B) During the extension of the lower cervical spine, the anterior margins of the vertebral bodies are widely separated, resulting in an avulsion of the annulus fibrosus from the vertebral endplate (a rim lesion).

C) Also during the extension of the lower cervical spine the inferior facet articular process chisels into the superior facet articular surface of the vertebra below, allowing for the spectrum of lesions to the facet joints.

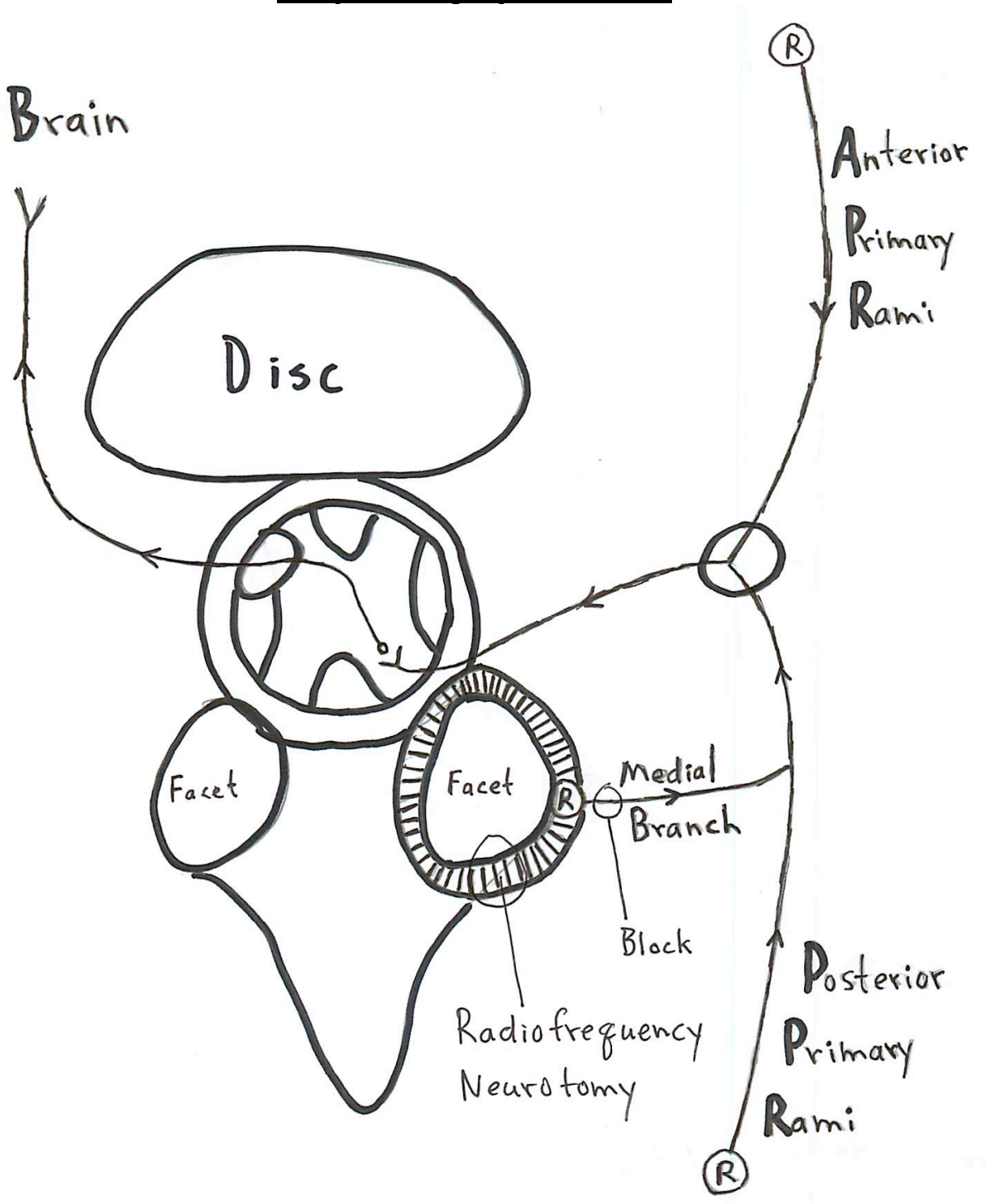
- The intraarticular meniscoids could be contused or ruptured

- Impaction fractures of the articular processes could occur

- Cadavers studies during whiplash show that the facet joints undergo compression that exceeds physiological limits and the capsules undergo strains beyond normal limits.

- Strains in the annulus fibrosus can exceed normal limits.

Whiplash Injury Facet Pain



Whiplash injures the *facet* joint.
The *facet* joint has nociceptors "R" which are connected to the brain through the medial branches of the posterior primary rami.
If diagnostic anesthetic blocking of the medial branch of the posterior primary rami eliminates pain, it indicates the *facet* is the source of the pain.
Radiofrequency neurotomy of the *facet* joint capsules coagulates the neurofiliment proteins, giving 70% of the patients longer relief of their whiplash pain.

Spine Pain

European Journal of Radiology
May 2015; Vol. 84; pp. 746–756

R. Izzo, T. Popolizio, P .D'Aprile, M. Muto

KEY POINTS FROM THIS ARTICLE:

1) "Spinal pain, and especially low back pain (LBP), represent the second leading cause for a medical consultation in a primary care setting and is a leading cause of disability worldwide."

2) Low back pain (LBP) is the 2nd leading cause of disability worldwide. About 1% of the world population is disabled with low back pain.

3) The resolution of chronic LBP can occur in less than 5–10% of cases.

4) Acute nonspecific back pain is *NOT* a benign, transient and self-limiting condition. Its rate of one-year recurrence is 20–44%, and a lifetime recurrence of up to 72%.

5) The most frequent cause of LBP is "internal disc disruption (IDD) and is referred to as discogenic pain." **[Key Point]** "Internal disc disruption refers to annular fissures, disc collapse and mechanical failure, with no significant modification of external disc shape, with or without endplates changes."

6) Discogenic pain is "considered as the most frequent cause of chronic low back pain." Discogenic pain secondary to internal disc disruption is the main cause of chronic LBP and disability.

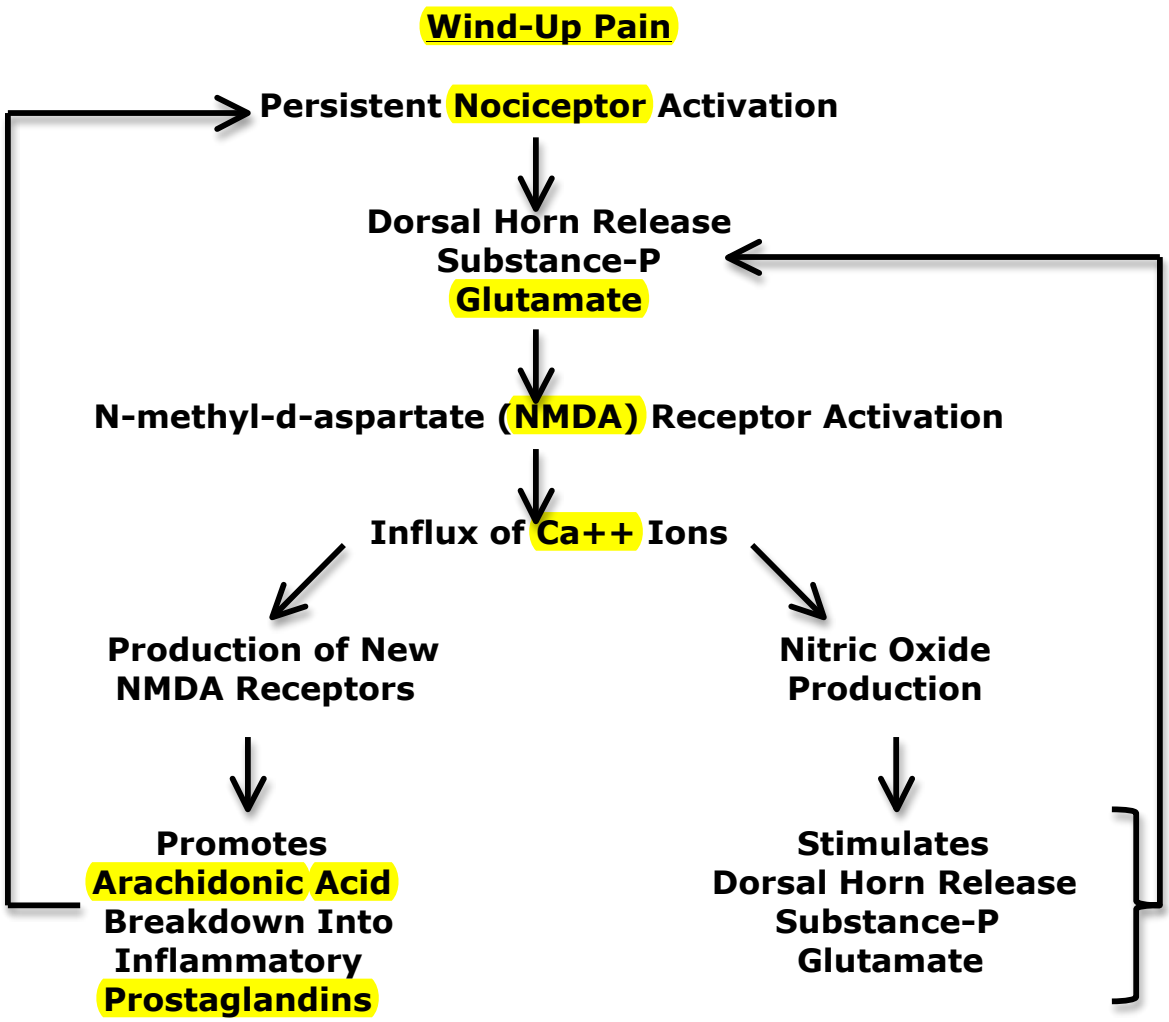
7) Both discogenic and radicular pain have either a mechanical and/or inflammatory genesis.

8) Advanced spinal imaging is "not sufficient for a definitive diagnosis because similar findings could be present in either asymptomatic and symptomatic subjects."

9) The posterior disc annulus and cartilaginous end plates are innervated by the sinuvertebral nerves. The sinuvertebral nerve is formed by the union of the grey ramus communicans [post ganglionic sympathetic efferents] and a small branch from the anterior primary ramus of the spinal nerve. **[Sympathetic Innervation]**

10) "Sympathetic trunks and ganglia directly innervate the anterior longitudinal ligament (ALL), the anterior periostium and vertebral body, the paravertebral muscles and fascia as well as the anterolateral disc." **[Sympathetic Innervation]**

- Inflammatory prostaglandins activate the peripheral nociceptors.

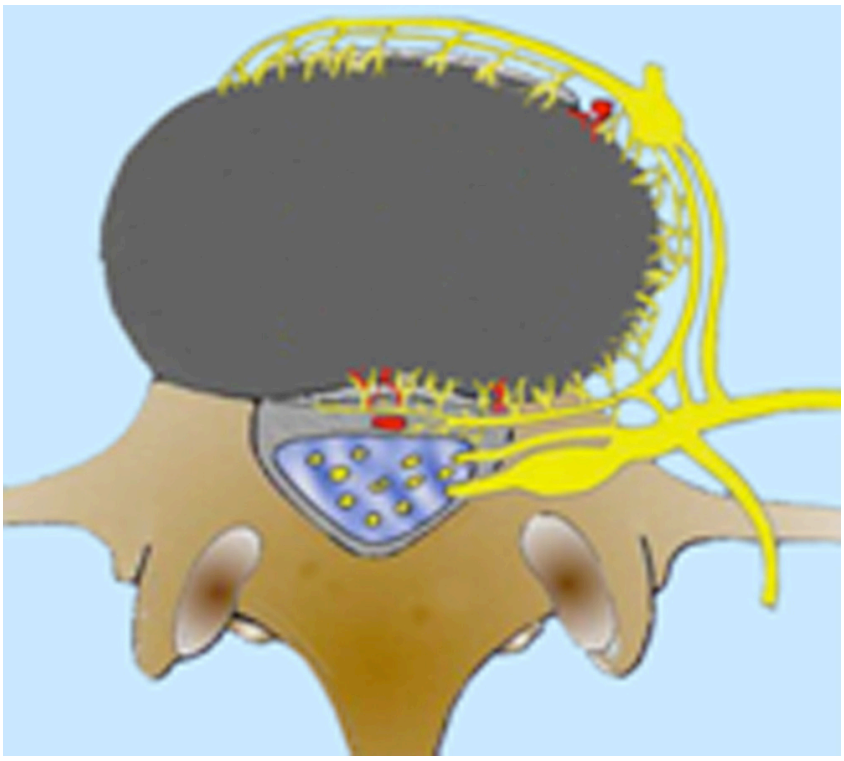


18) Chronic LBP patients have
- Impaired motion
- Spinal repositioning errors
- Postural imbalance
- Early muscle fatigue
- The absence of muscle relaxation at full lumbar flexion

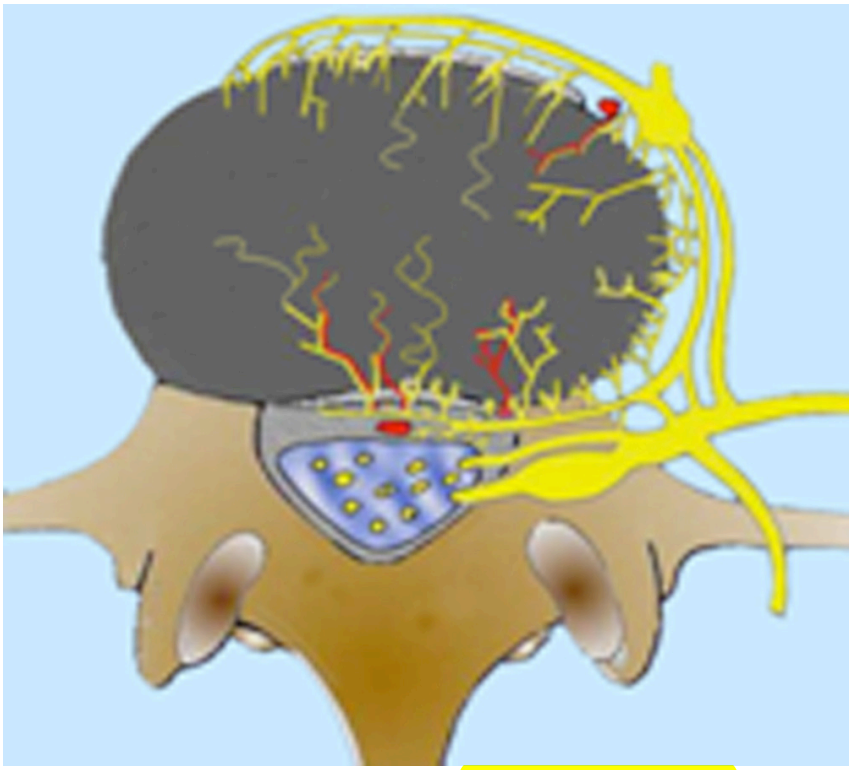
19) Patients with psychological distress can develop an abnormal spinal motion control that can elicit progressive chronic pain in absence of spinal injury.

20) Stability is required for correct function of the spine. "Degenerative instability is a common cause of axial and radicular pain and disability." Degenerative instability is often misdiagnosed, and is a frequent indication for surgery.

21) "Mechanical receptors located within discs, ligaments and joint capsules convey to CNS proprioceptive inputs on the spatial position, the loading status and the movement of each motion segment." Damage to any spinal structures and receptors generate altered inputs for the CNS that promote an inappropriate muscle



Normal Disc With Peripheral Innervation



Degenerated Disc With Neoneuralization

General Principles of Chronic Pain Management

Dan Murphy, DC

1) Gate Theory: Chiropractic management of asymmetries of posture/motion; improving mechanoreceptive afferentation while existing, living, and functioning in a gravity environment.

2) Balance the omega-6/omega-3 ratio (1.5-4/1); inflammation alters the threshold of the nociceptive afferent system. The billions Americans spend on pain drugs is primarily to counter the effects of too much omega-6 in the diet (or too low omega-3s).

3) Reduce pro-inflammatory cytokines:
   A) omega-6/omega-3 balance
   B) resveratrol (100 mg/day); curcumin (200 mg/day)

4) Vitamin D: 50-70 ng/ml. Perhaps as many as 60% of those with chronic pain have low levels of vitamin D.

5) Reduce / avoid / eliminate dietary excitotoxins (glutamate, aspartate [half of aspartame]). Excitotoxins are known to create chronic pain sensitization, and their elimination can 100% "cure" chronic pain syndromes.

6) Malic Acid: the sodium pump keeps the nociceptors further away from excitation threshold. The sodium pump runs on ATP energy. A proven disruption of lower ATP and chronic pain is low levels of malic acid in the Krebs Cycle. Supplementation with malic acid (2400 mg/day) and Mg++ (600 mg/day) has been shown to greatly help.

7) Manage insulin resistance:
   A) Insulin resistance lowers ATP (#6)
   B) Insulin upregulates the delta-5-desaturase enzyme (D5D), accelerating the conversion of linoleic acid (soy, corn, etc.) into the arachidonic acid cascade towards pro-inflammatory eicosanoids.

8) Any strategy that increases ATP is helpful:
   A) Chiropractic Adjusting
   B) Exercise
   C) Low Level Laser Therapy
   D) Breathing Exercises
   E) Anti-oxidant supplements
   F) Mitochondrial nutritional support (acetyl-l-carnitine, alpha-lipoic acid)
   G) Detoxification: undenatured whey protein, N-acetyl cysteine (NAC), Infrared sauna
   H) Stop Smoking

   ETC.

PubMed

Display Settings: Abstract

Can Fam Physician. 1985 Mar;31:535-40.

Spinal manipulation in the treatment of low-back pain.

Kirkaldy-Willis WH, Cassidy JD.

Abstract

Spinal manipulation, one of the oldest forms of therapy for back pain, has mostly been practiced outside of the medical profession. Over the past decade, there has been an escalation of clinical and basic science research on manipulative therapy, which has shown that there is a scientific basis for the treatment of back pain by manipulation. Most family practitioners have neither the time nor inclination to master the art of manipulation and will wish to refer their patients to a skilled practitioner of this therapy. Results of spinal manipulation in 283 patients with low back pain are presented. The physician who makes use of this resource will provide relief for many patients.

PMID: 21274223 [PubMed] PMCID: PMC2327983 **Free PMC Article**

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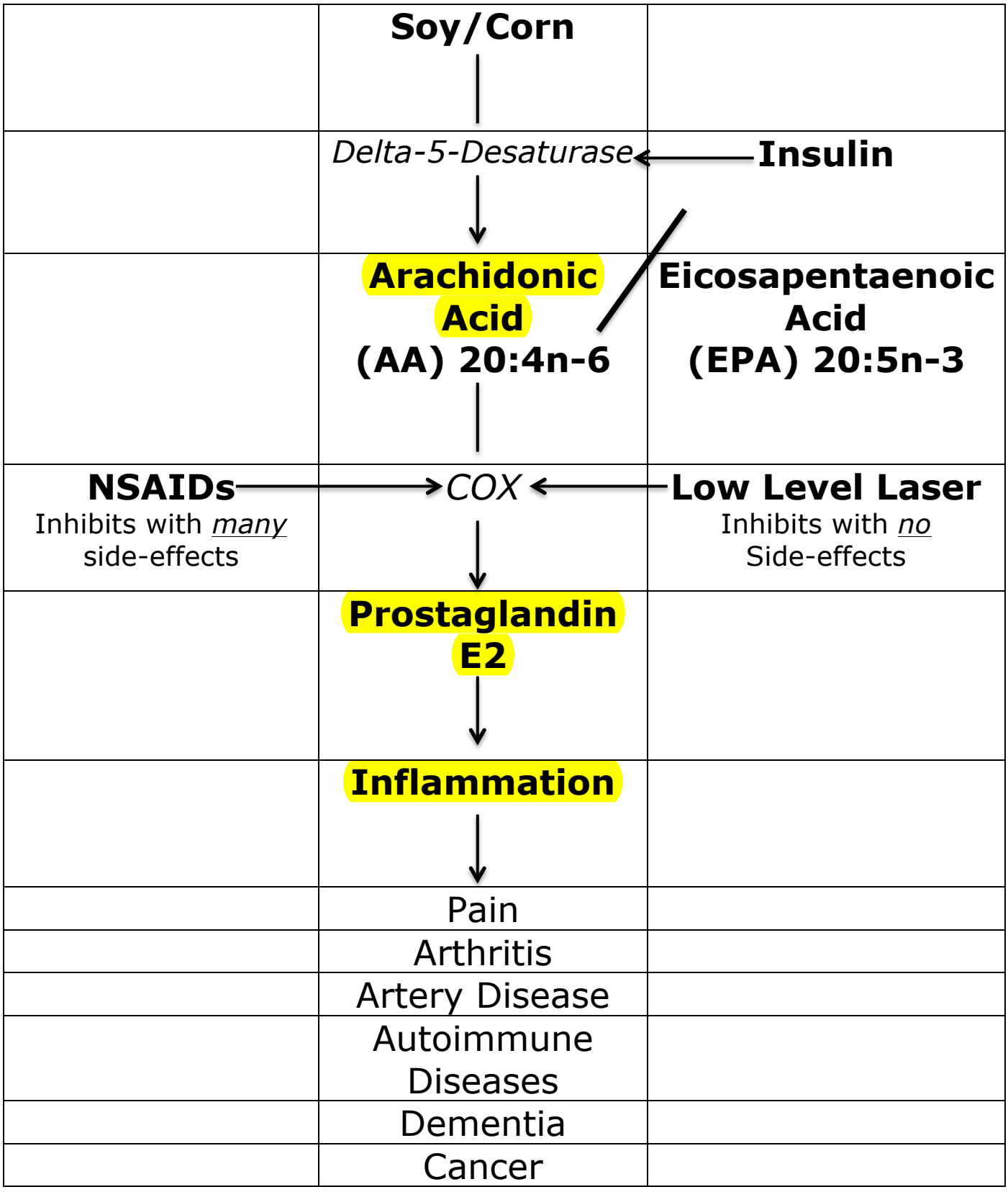
The Theory of Everything
Michael Pollan
In Defense of Food, 2008



ıan, MD

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EAT FAT, GET THIN

Why the Fat We Eat Is the Key to Sustained Weight Loss and Vibrant Health

Mark Hyman, MD

2016

LITTLE, BROWN AND COMPANY
New York Boston London

Eat Fat, Get Thin

Why the Fat We Eat Is the Key to Sustained Weight Loss and Vibrant Health

Mark Hyman, MD
Little, Brown and Company, 2016

"Soybean oil consumption has increased 1,000-fold since 1900." p. 80

"Thanks to farm subsidies and the power of Monsanto's global soybean monopoly, Americans now consume about 18 billion pounds of soybean oil a year. About 20 percent of our calories come from soybean oil." "Check your grocery store labels; soybean oil is in almost everything." p. 117

"At the turn of the century, vegetable oils were almost unknown in the food supply." p. 117

"What's surprising to most people is that meat and chicken are big sources of omega-6 fats." "Industrial farming practices have led farmers to switch their feed from grass to corn and cereal grains, and now those omega-6 vegetable fats comprise a significant portion of 'animal' fat. You are what you eat. Or more accurately: You are what your food eats." p. 117

"Our dietary omega-6 fats have skyrocketed, while omega-3 fats have declined. Now we eat about ten times as much omega-6 as omega-3 oils, and some people eat up to twenty-five times as much." p. 118

"Balance is critical. The omega-6 fats fuel inflammatory pathways in the body, while omega-3 fats are anti-inflammatory." p. 118

“Dr. Hibbeln found that the increase of linoleic acid [plant based omega-6, found in corn, soy, sunflower, safflower, cotton, peanut, canola, etc.], mostly from soybean oil, in the diet from 1960 to 1999 in five countries studies predicted a 100-fold increase in the risk of homicide deaths.” p. 124

“Not only do these vegetable oils result in more heart attacks, obesity, and cancer, but they may make people murderers!” p. 124

“Considering omega-3 fats make up much of your brain tissue, this makes sense because high intake of omega-6 fats interferes with the benefits of omega-3 fats.”

Increases in world LA (linoleic acid) consumption over the past century “may have contributed to increased societal burdens of aggression, depression, and cardiovascular mortality.” p. 124

“It's quite likely that most of the diseases of modern civilization, major depression, heart disease, and obesity are linked to the radical and dramatic shift in the composition of the fats in the food supply.” p. 124

“Increasing tissue concentrations of omega-3 fats on a population level may result in a substantial decrease in health care costs by reducing the illnesses that account for the largest burden of disease worldwide.” p. 124

PubMed

Format: Abstract

Lipids. 2004 Dec;39(12):1207-13.

Increasing homicide rates and linoleic acid consumption among five Western countries, 1961-2000.

Hibbeln JR[1], Nieminen LR, **Lands** WE.

Author information

[1]Laboratory of Membrane Biochemistry and Biophysics, National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health, Bethesda, Maryland 20892, USA. jhibbeln@mail.nih.gov

Abstract

Clinical intervention trials and animal studies indicate that increasing dietary intakes of long chain n-3 FA or reducing linoleic acid intake may reduce aggressive and violent behaviors. Here we examine if economic measures of greater n-6 consumption across time and countries correlate with greater risk of homicide. Linoleic acid available for human consumption was calculated from World Health Organization disappearance data for 12 major seed oils in the food supply for the years 1961 to 2000 in Argentina, Australia, Canada, the United Kingdom, and the United States (US). Homicide mortality rates, adjusted for age, were obtained from the central judicial authority of each country. Apparent linoleic acid intake from seed oil sources ranged from 0.29 en% (percentage of daily food energy) (Australia 1962) to 8.3 en% (US 1990s). Greater apparent consumption of linoleic acid correlated with higher rates of homicide mortality over a 20-fold range (0.51-10.2/100,000) across countries and time in an exponential growth regression model ($r = 0.94$, $F = 567$, $P < 0.00001$). Within each country, correlations between greater linoleic acid disappearance and homicide mortality over time were significant in linear regression models. Randomized controlled trials are needed to determine if reducing high intakes of linoleic acid by seed oils with alternative compositions can reduce the risk of violent behaviors. These dietary interventions merit exploration as relatively cost-effective measures for reducing the pandemic of violence in Western societies, just as dietary interventions are reducing cardiovascular mortality. Low linoleate diets may prevent behavioral maladies that correctional institutions, social service programs, and mental health providers intend to treat.

PMID: 15736917

[PubMed - indexed for MEDLINE]

Out of 238 million American adults, 100 million live in chronic pain. And yet the press has paid more attention to the abuses of pain medications than the astoundingly widespread condition they are intended to treat. Ethically, the failure to manage pain better is tantamount to torture. When chronic pain is inadequately treated, it undermines the body and mind. Indeed, the risk of suicide for people in chronic pain is twice that of other people. Far more than just a symptom, writes author Judy Foreman, chronic pain can be a disease in its own right—the biggest health problem facing America today.

In *A Nation in Pain,* Foreman offers a sweeping, deeply researched account of the chronic pain crisis, from neurobiology to public policy, and presents practical solutions that are within our grasp today. Drawing on both her personal experience with chronic pain and her background as an award-winning health journalist, she guides us through recent scientific discoveries, including genetic susceptibility to pain; gender disparities in pain conditions and treatments, perhaps linked to estrogen; the problem of undertreated pain in children; the emerging role of the immune system in pain; advances in traditional treatments such as surgery and drugs; and fair-minded assessments of the effectiveness of alternative remedies, including marijuana, acupuncture, massage, and chiropractic care. For many people, the real magic bullet, Foreman writes, is exercise. Though many patients fear it will increase their discomfort, studies show it consistently produces improvement, often dramatic.

A Nation in Pain

Healing Our Biggest Health Problem

Judy Foreman

2014

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International Association for the Study of Pain
IASP
Working together for pain relief

Monday
9/19/11

Pain management: Education is key

USAT

By Kevin Pho 9/19/11

A fellow physician recently shared a frustrating clinic visit with me, in which a patient had left by saying, "You doctors need to wake up and realize that patients (who are) in pain are in a no-win situation."

The patient was absolutely right. This summer, the Institute of Medicine released a report, "Relieving Pain in America," which found that 116 million Americans suffer from chronic pain, costing the U.S. up to $635 billion in treatment and lost productivity. Chronic pain even increases the risk of depression and suicide.

But when it comes to treating pain, doctors also face no-win situations. While chronic pain is effectively treated with opioids — a class of medications that includes morphine and OxyContin, as well as heroin — close monitoring of the patient is essential because the drugs can be addictive.

Sales exploding

In a column in *The New England Journal of Medicine* last fall, physician Susan Okie noted the explosion in the sales of pain medication, as well as a marked increase in emergency room visits for pain drug overdoses. In fact, according to the Centers for Disease Control and Prevention, deaths from unintentional drug overdoses in the USA, primarily driven by opioids, are the second-leading cause of accidental death.

Medical Hypotheses (2007) 69, 1169–1178

medical hypotheses

http://intl.elsevierhealth.com/journals/mehy

The biochemical origin of pain: The origin of all pain is inflammation and the inflammatory response. Part 2 of 3 — Inflammatory profile of pain syndromes

Sota Omoigui *

Division of Inflammation and Pain Research, L.A Pain Clinic, 4019 W. Rosecrans Avenue, Los Angeles, CA 90250, United States

Received 27 June 2007; accepted 27 June 2007

Summary Every pain syndrome has an inflammatory profile consisting of the inflammatory mediators that are present in the pain syndrome. The inflammatory profile may have variations from one person to another and may have variations in the same person at different times. The key to treatment of Pain Syndromes is an understanding of their inflammatory profile. Pain syndromes may be treated medically or surgically. The goal should be inhibition or suppression of production of the inflammatory mediators and inhibition, suppression or modulation of neuronal afferent and efferent (motor) transmission. A successful outcome is one that results in less inflammation and thus less pain. We hereby briefly describe the inflammatory profile for several pain syndromes including arthritis, back pain, neck pain, fibromyalgia, interstitial cystitis, migraine, neuropathic pain, complex regional pain syndrome/reflex sympathetic dystrophy (CRPS/RSD), bursitis, shoulder pain and vulvodynia. These profiles are derived from basic science and clinical research performed in the past by numerous investigators and serve as a foundation to be built upon by other researchers and will be updated in the future by new technologies such as magnetic resonance spectroscopy. Our unifying theory or law of pain states: the origin of all pain is inflammation and the inflammatory response. The biochemical mediators of inflammation include cytokines, neuropeptides, growth factors and neurotransmitters. Irrespective of the type of pain whether it is acute or chronic pain, peripheral or central pain, nociceptive or neuropathic pain, the underlying origin is inflammation and the inflammatory response. Activation of pain receptors, transmission and modulation of pain signals, neuro plasticity and central sensitization are all one continuum of inflammation and the inflammatory response. Irrespective of the characteristic of the pain, whether it is sharp, dull, aching, burning, stabbing, numbing or tingling, all pain arise from inflammation and the inflammatory response. We are proposing a re-classification and treatment of pain syndromes based upon their inflammatory profile. Published by Elsevier Ltd.

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E-mail address: medicinechief@aol.com

0306-9877/$ - see front matter Published by Elsevier Ltd.
doi:10.1016/j.mehy.2007.06.033

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The Nobel Prize in Physiology or Medicine 1982

Sune K. Bergström, Bengt I. Samuelsson, John R. Vane

Sune K. Bergström

Bengt I. Samuelsson

John R. Vane

The Nobel Prize in Physiology or Medicine 1982 was awarded jointly to Sune K. Bergström, Bengt I. Samuelsson and John R. Vane *"for their discoveries concerning prostaglandins and related biologically active substances"*.

TO CITE THIS PAGE:
MLA style: "The Nobel Prize in Physiology or Medicine 1982". Nobelprize.org. 13 Jan 2012 http://www.nobelprize.org/nobel_prizes/medicine/laureates/1982/

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American Academy of Pain Management
Weiner's Pain Management
A Practical Guide for Clinicians
Seventh Edition, 2006, pp.584-585
Edited by Mark Boswell and B. Eliot Cole

"Changes in the modern diet are largely responsible for the increasing incidence of essential fatty acid (EFA) imbalances and deficiencies."

"The ratio of omega-6 to omega-3 fats has changed dramatically due to the widespread use of vegetable oils (mostly n-6 fats) in cooking and to the processing of oils to alter omega-3 fats to improve shelf life and eliminate their stronger taste (just think of the distinctive tastes of cod liver or flax oil)."

"Historical estimates place the ratio of omega-6 to omega-3 oils at nearly 1:1 for prehistoric humans."

By the turn of the century (1900), the ratio had increased to about 4:1.
The current American ratio is about 25:1.

"The sharp rise is due to increased vegetable oil consumption:
from 2 lb. per year in 1909 to 25 lb. per year in 1985!"

"Many of the chronic inflammatory conditions that accompany EFA imbalance are currently treated with symptom-specific pharmaceutical drugs such as steroids, prednisone, aspirin, and other nonsteroidal anti-inflammatory drugs (NSAIDs), sulfasalazine, and colchicine."

"The problem with such drug therapies is that they prevent the formation of 'good' anti-inflammatory eicosanoids, or they shift the production of one type of eicosanoid to another."

"For effective, long-term management, eicosanoid production should be modified through dietary changes (balancing dietary intake of specific fats) and controlling insulin levels in the circulation."

"Maintaining a proper balance between the various families of dietary fats may be one of the most important preventative measures a person can take to reduce the likelihood of developing one of the chronic diseases of modern civilization, such as diabetes, heart disease, obesity, irritable bowel syndrome, and autoimmune disease."

"And for patients who may already have one of these diseases, EFA testing and therapy has been demonstrated to reduce both morbidity and mortality associated with these diseases."

In Defense of Food
Michael Pollan
2008

Omega-3s

"We're eating a lot more seeds and a lot fewer leaves (as do the animals we depend on)." "Leaves provide a host of critical nutrients a body can't get from a diet of refined seeds. There are antioxidants and phytochemicals; there is the fiber; and then there are the essential omega-3 fatty acids found in leaves, which some researchers believe will turn out to be the most crucial missing nutrient of all." pp. 124-125

"Too much omega-6 may be just as much a problem as too little omega-3" p. 126

"For years plant breeders have been unwittingly selecting for plants that produce fewer omega-3s, because such crops don't spoil as quickly." p. 127

"When food makers partially hydrogenate oils to render them more stable, it is the omega-3s that are eliminated." p. 127

"Researchers are convinced that these historically low levels of omega-3 (or, conversely, historically high levels of omega-6) bear responsibility for many of the chronic diseases associated with the Western diet." pp. 127-128

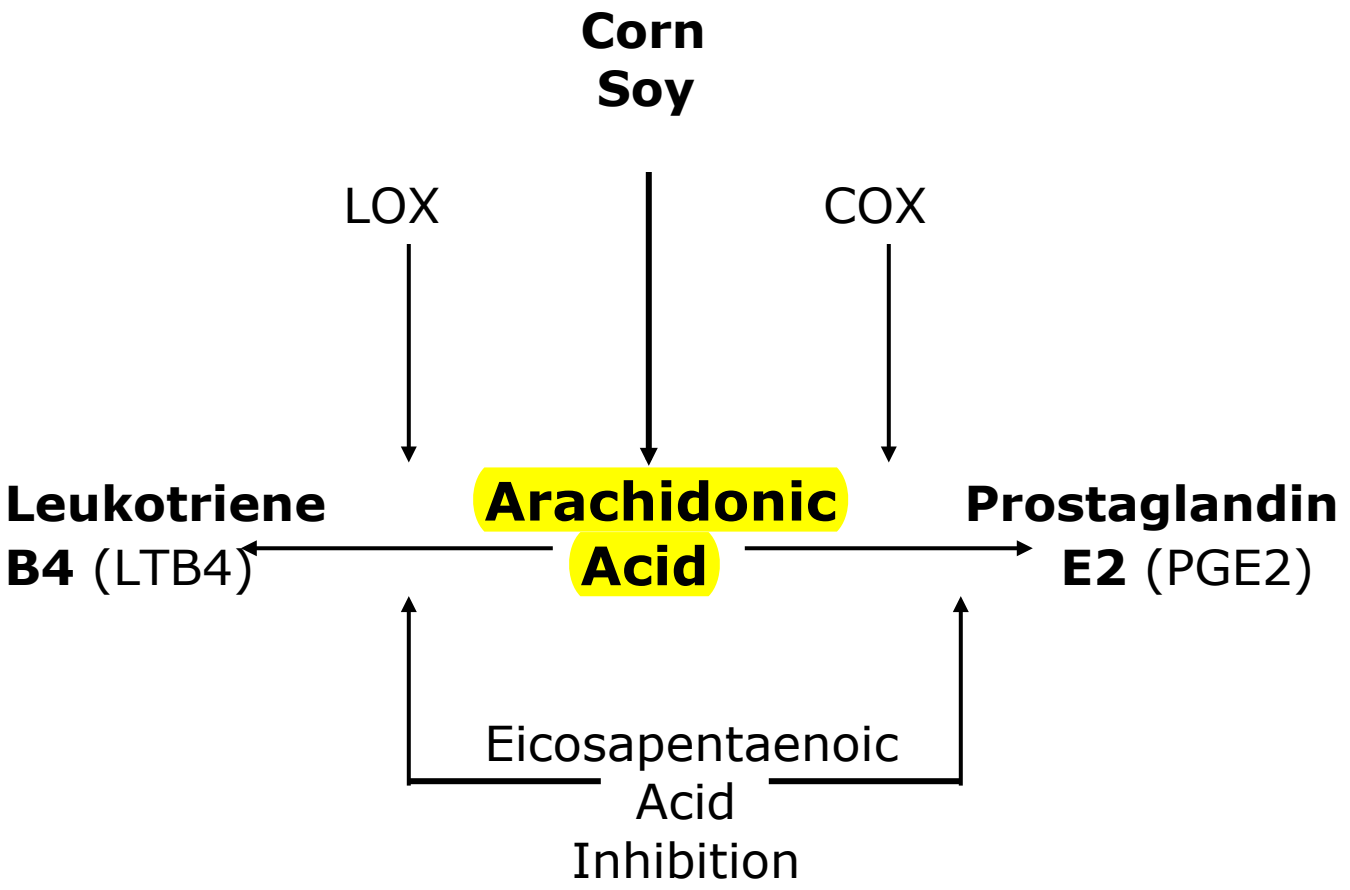
"Could it be that the problem with the Western diet is a gross deficiency in this [omega-3s] essential nutrient? A growing number of researchers have concluded that it is." p. 130

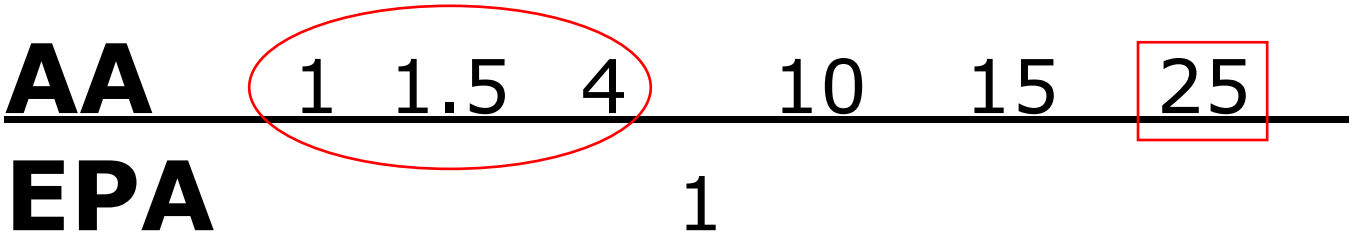
"The billions we spend on anti-inflammatory drugs such as aspirin, ibuprofen, and acetaminophen is money spent to undo the effects of too much omega-6 in the diet." p. 131

"Of all of the changes to our food system that go under the heading "The Western Diet," the shift from a food chain with green plants as its base to one based on seeds may be the most far reaching of all. Nutritional scientists focus on different nutrients—whether the problem with the modern diet is too many refined carbohydrates, not enough good fats, too many bad fats, or a deficiency of any number of micronutrients or too many total calories. But at the root of all these biochemical changes is a single ecological change. For the shift from leaves to seeds affects much more than the levels of omega-3 and omega-6 in the body. It also helps account for the flood of refined carbohydrates in the modern diet and the drought of so many micronutrients and the surfeit of total calories. From *leaves* to *seeds*: It's almost, if not quite, a Theory of Everything." p. 132

"The rule about eating more leaves and fewer seeds applies not only to us but also to the animals in our food chain." p. 168

The Theory of Everything





Chronic Spinal Pain: A Randomized Clinical Trial Comparing Medication, Acupuncture, and Spinal Manipulation

Spine July 15, 2003; 28(14):1490-1502

Lynton G. F. Giles, DC, PhD; Reinhold Muller, PhD

FROM THE ABSTRACT:

Study Design.
A randomized controlled clinical trial was conducted.

Objective.
To compare medication, needle acupuncture, and spinal manipulation for managing chronic (>13 weeks duration) spinal pain because the value of medicinal and popular forms of alternative care for chronic spinal pain syndromes is uncertain.

Summary of Background Data.
Between February 1999 and October 2001, 115 patients without contraindication for the three treatment regimens were enrolled at the public hospital's multidisciplinary spinal pain unit.

Methods.
One of three separate intervention protocols was used: medication, needle acupuncture, or chiropractic spinal manipulation.
[THE MANIPULATION WAS DONE BY CHIROPRACTORS]

Patients were assessed before treatment by a sports medical physician for exclusion criteria and by a research assistant using the Oswestry Back Pain Disability Index (Oswestry), the Neck Disability Index (NDI), the Short-Form-36 Health Survey questionnaire (SF-36), visual analog scales (VAS) of pain intensity and ranges of movement.

These instruments were administered again at 2, 5, and 9 weeks after the beginning of treatment.

Results.
The highest proportion of early (asymptomatic status) recovery was found for manipulation (27.3%), followed by acupuncture (9.4%) and medication (5%). **[WOW!]**

Manipulation achieved the best overall results, with improvements of 50% on the Oswestry scale, 38% on the NDI, 47% on the SF-36, and 50% on the VAS for back pain, 38% for lumbar standing flexion, 20% for lumbar sitting flexion, 25% for cervical sitting flexion, and 18% for cervical sitting extension.
[WOW, THIS SHOWS SUBJECTIVE IMPROVEMENT, FUNCTIONAL IMPROVEMENT, OBJECTIVE IMPROVEMENT IN RANGE OF MOTION, AND SIGNIFICANT IMPROVEMENT IN GENERAL HEALTH STATUS]

However, on the VAS for neck pain, acupuncture showed a better result than manipulation (50% vs 42%).

Conclusions.
The consistency of the results provides evidence that in patients with chronic spinal pain, manipulation, if not contraindicated, results in greater short-term improvement than acupuncture or medication.

However, the data do not strongly support the use of only manipulation, only acupuncture, or only nonsteroidal antiinflammatory drugs for the treatment of chronic spinal pain.

KEY POINTS FROM DAN MURPHY

1) It is impossible to reach specific diagnosis for the pathologic cause for 85% of those with an episode of spinal pain.

2) Patients with low back pain do exhibit abnormal spinal motion.

3) There is insufficient evidence for the use of NSAIDs to manage chronic low back pain.

4) The new COX-2 nonsteroidal antiinflammatory (NSAIDs) have problems and significant contraindications.

5) Gastrointestinal toxicity induced by NSAIDs is one of the most common serious adverse drug events in the industrialized world.

6) In this study, in the medication group, more patients experienced adverse events (6.1%) than recovered from their spinal complaints (5%).

7) Even though the chiropractic treatment group was the most chronic (8.3 years), 27.3% recovered with 18 spinal adjustments over a period of 9 weeks, or less. **[VERY IMPRESSIVE]**
This means that better than every fourth patient became asymptomatic with 9 weeks or less of chiropractic manipulation, even though they had been chronic for more than 8 years. **[WOW!]**

8) The chiropractic treatment group showed significantly greater improvement in subjective complaints, functional abilities, objective range of spinal motion, and in general health status than acupuncture and medication.

9) In this study, patient involvement in litigation did not influence the outcome measures.

10) In the treatment of chronic spinal pain, chiropractic manipulation is superior to acupuncture and medication.

Chronic Spinal Pain: A Randomized Clinical Trial Comparing Medication, Acupuncture, and Spinal Manipulation

Spine, July 15, 2003; 28(14): 1490-1502

Treatment	**Drugs (Celebrex or Vioxx)**	**Acupuncture**	**Chiropractic Adjustments**
Years Of Chronic Spinal Pain	**4.5 or 6.4**	**4.5 or 6.4**	**8.3**
% Asymptomatic within 9 weeks	**5%**	**9.4%**	**27.3%**
% That suffered an adverse event	**6.1%**	**0%**	**0%**
% Improvement In General Health Status	**18%**	**15%**	**47%**

Pain

Omega-3 Fatty acids (fish oil) as an anti-inflammatory: an alternative to nonsteroidal anti-inflammatory drugs for discogenic pain

Surgical Neurology
65 (April 2006) 326– 331

This paper won first prize in the poster competition at the American Association of Neurological Surgeons Annual Meeting, New Orleans, LA, April 2005

Joseph Charles Maroon, MD, Jeffrey W. Bost, PAC
These authors are from the Department of Neurological Surgery, University of Pittsburgh Medical Center

KEY POINTS FROM DAN MURPHY

1) The use of NSAIDs is associated with extreme complications, including gastric ulcers, bleeding, myocardial infarction, stroke, and even death.

2) In this study, after 75 days on fish oil, 59% of patients who were taking NSAIDs for chronic spinal pain and who had degenerative spine disease, were able to discontinue their prescription NSAIDs, and 88% stated they were satisfied with their improvement and that they would continue to take the fish oil.

3) In this study, fish oil supplementation was not associated with any significant side effects.

4) "Omega-3 EFA fish oil supplements appear to be a safer alternative to NSAIDs for treatment of nonsurgical neck or back pain."

5) "More than 70 million NSAID prescriptions are written each year, and 30 **b**illion over-the-counter NSAID tablets are sold annually."
[Notice, 30 BILLION over-the-counter NSAID tablets are sold annually]

6) "5% to 10% of the adult US population and approximately 14% of the elderly routinely use NSAIDs for pain control."

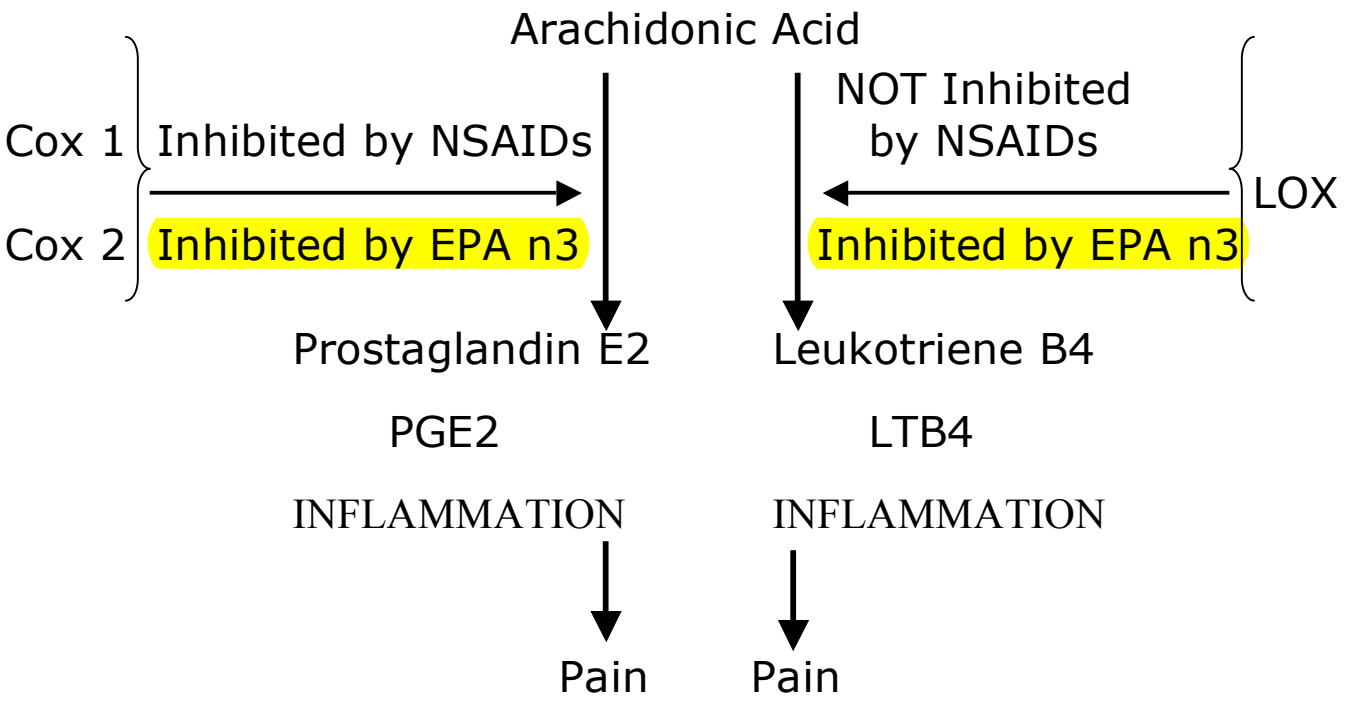
7) Selling NSAIDs is a 9 billion dollar per year US industry.

8) Prescription NSAIDs for rheumatoid and osteoarthritis alone conservatively cause 16,500 deaths per year.

9) "NSAIDs are the most common cause of drug-related morbidity and mortality reported to the FDA and other regulatory agencies around the world."

Fish oil: what the prescriber needs to know

Arthritis Research & Therapy
Volume 8, Issue 1, 2006, pp. 402
Leslie G Cleland, Michael J James and Susanna M Proudman
KEY POINTS FROM DAN MURPHY



1) There is a general belief among doctors that patients with arthritis need nonsteroidal anti-inflammatory drugs (NSAIDs). This is because the pain of arthritis is primarily caused by PGE2, which is derived from the omega-6 fatty acid arachidonic acid through the activity of the enzyme COX. NSAIDs inhibit the COX enzyme.

2) However, NSAIDs increase the risk for cardiovascular events.

3) Fish oils contain a natural inhibitor of COX, reduce reliance on NSAIDs, and reduce cardiovascular risk.

4) Omega-6s (n6) and omega-3s (n3) are dietary essential fatty acids which cannot be synthesized endogenously.

5) Diets in industrialized Western countries are generally abundant in n6 PUFAs and poor in n3 PUFAs.

6) "Because Western diets are typically low in LC n3 PUFAs, substantial increases in tissue LC n3 can be achieved by taking a fish oil supplement."

7) It is unlikely that one can consume the amount of fish required to achieve anti-inflammatory doses (minimum of 2.7 g/day) of LC n3 PUFAs.

8) "The conversion of C18 n3 PUFAs [such as flax oil] to C20 and C22 n3 PUFAs [fish oil] occurs relatively inefficiently in humans, and so vegetable sources of dietary n3 PUFAs alone fail to achieve the tissue levels seen with fish oil."

9) "EPA [fish oil omega-3] is both an inhibitor of AA metabolism and an alternate substrate for COX."

10) "EPA [fish oil omega-3] also inhibits the metabolism of arachidonic acid into leukotriene B4 by LOX enzymes, which NSAIDs do not do. Consequently, EPA fish oil is superior to NSAIDs in creating an anti-inflammatory effect."

11) NSAIDs increase the synthesis of tumor necrosis factor (TNF) alpha which causes both inflammation and cartilage degradation. In contrast, EPA fish oil inhibits TNF alpha.

12) "The anti-inflammatory dose of fish oil requires delivery of 2.7 g or more of LC n3 PUFAs daily." **[Very Important]**

13) A daily intake of less than 2.7 g EPA plus docosahexaenoic acid (DHA) is "insufficient for an anti-inflammatory effect." **[Very Important]**

14) Symptomatic improvement from fish oil supplementation can take 2–3 months, and "it is important that potential users understand that this delay exists."

15) Patients should also reduce ingestion of n6 PUFA by substituting olive oil for vegetable oils.

16) "At anti-inflammatory doses, cod liver oils, which are rich in the fat-soluble vitamins A and D, contain more vitamin A than recommended intakes." Vitamin A has been associated with reduced bone density and increased risk for hip fracture.

17) Vitamin A toxicity is not a problem with anti-inflammatory doses of fish body oils because they contain very little vitamin A. **[Important]**

18) "Fish oil (obtained from the body of the fish) is preferable to cod liver oil, which can deliver undesirable amounts of vitamin A at anti-inflammatory doses."

19) "The odour of fish oil can be minimized by keeping fish oil refrigerated once open."

20) "Because most anti-inflammatory **drugs** can have adverse effects on the fetus, they are generally withdrawn during pregnancy and lactation."

21) LC n3 PUFAs are strongly represented among neural lipids, and neural development is particularly active in utero and during infancy.

22) "There is a dramatic fall in maternal plasma DHA in the immediate postpartum period."

23) "There is no evidence of harm at supplementation levels of at least 2.7 g/day of LC n-3 PUFAs" during pregnancy.
24) "Within the Western context, fish oil supplements have not been associated with an increased bleeding tendency, even in patients taking aspirin or warfarin for antithrombotic effect."

25) "Methylmercury is an industrial contaminant that accumulates in long-lived fish (e.g. swordfish, marlin, sea perch, shark)."

26) "Methylmercury is a neurotoxin that impairs neural development, especially in the foetus and infants."

27) Fish consumption is associated with increased blood and urine mercury.

28) "Properly processed fish oils contain very little mercury."

29) "Chlorinated biphenyls (PCBs) are byproducts of industrial synthesis of organic chemicals. They are structurally related to dioxins and are potentially toxic."

30) PCBs are poorly biodegradable and they accumulate in the land and marine food chains.

31) Polybrominated biphenyl (PBB) fire retardants are similar to PCBs.

32) "Halogenated biphenyls can be removed from fish oils by molecular distillation and should be present at low levels in good quality products."

33) There is a "striking reduction in cardiac mortality and, in particular, sudden cardiac death seen with fish oil and diets rich in n3 PUFAs."

34) Increased LC n3 PUFA intake reduce annualized death rates better than statin drugs. "That fish oil is not used more widely to manage cardiovascular risk appears to reflect more the influence of pharmaceutical product marketing on the practice of 'evidence-based medicine' than the merits of fish oil relative to those of commonly used proprietary agents." **[Very Important]**

35) "In a medical environment in which messages molded by pharmaceutical interests stress the 'need' for NSAIDs, prescribers should consider the NSAID-sparing effects, the lack of serious side effects and the positive health benefits of fish oil."

36) "Although modest increases in intake of n3 LC PUFAs can reduce cardiovascular risk, relatively large doses (more than 2.7 g/day EPA plus DHA) are required for anti-inflammatory effects."

A meta-analysis of the analgesic effects of omega-3 polyunsaturated fatty acid supplementation for inflammatory joint pain

Pain
May 2007, 129(1-2), pp. 210-223

Robert J. Goldberg and Joel Katz

Abbreviations:
ALA Alpha-linolenic acid
18 carbon long omega-3 plant fatty acid
EPA Eicosapentaenoic acid
20 carbon long omega-3 fish fatty acid
DHA Docosahexaenoic acid
22 carbon long omega-3 fish fatty acid
LA Linoleic acid
18 carbon long omega-6 plant fatty acid
AA Arachidonic acid
20 carbon long omega-6 animal fatty acid

KEY POINTS FROM DAN MURPHY

1) "Between 40% and 60% of Americans use complementary and alternative medicine to manage medical conditions, prevent disease, and promote health and well-being."

2) 33% of those who use complementary medicine cite pain as the primary reason.

3) "Supplementation with n-3 PUFAs for 3–4 months reduces patient reported joint pain intensity, minutes of morning stiffness, number of painful and/or tender joints, and NSAID consumption."

4) Omega-3 PUFAs are an adjunctive treatment for joint pain associated with rheumatoid arthritis, inflammatory bowel disease, and dysmenorrhea.

5) Nonsteroidal anti-inflammatory drugs are associated with gastrointestinal bleeding and myocardial infarction.

6) "The typical North American diet is very low in EPA and DHA and conversion is limited from dietary alpha-linolenic acid, found in vegetable oils, to EPA and DHA."

7) Fish oil is a rich source of long-chain n-3 PUFAs EPA and DHA.

8) "In humans, supplementation with fish oil, or EPA/DHA capsules, increases the incorporation of n-3 PUFAs into phospholipids, conferring anti-inflammatory effects."

9) The therapeutic effects of n-3 PUFAs usually manifest after approximately 3 months, and "taking n-3 PUFA supplementation for 2 months or less would not benefit significantly." **[Important]**

10) Studies that provided high-dose (more than 2.7 g/day of EPA and DHA) n-3 PUFAs showed greater improvements in morning stiffness and number of painful and/or tender joints compared to low-dose n-3 PUFAs.

11) "The results of the present meta-analysis support the hypothesis that n-3 PUFA supplementation improves pain outcomes after three months, particularly with respect to patient assessed pain, duration of morning stiffness, number of painful and/or tender joints, and [reduced] NSAID consumption."

12) A minimum of three months of supplementation with a dose of 2.7 g/day of EPA and DHA is required to achieve an anti-inflammatory and a therapeutic effect." **[Important]**

13) "Significant improvements were noted in patient assessed pain and morning stiffness among studies providing high-dose but not low-dose n-3 PUFA supplementation." **[Important]**

14) "Reducing the intake of n-6 fatty acids (e.g., linoleic acid), which are metabolized to arachidonic acid and inflammatory eicosanoids, would be expected to increase the effectiveness of n-3 PUFA supplements."

15) EPA/DHA supplements may also be useful for other types of chronic inflammatory pain, such as osteoarthritis or chronic back pain.

16) Alpha-linolenic acid [flax seed oil, etc.] is poorly converted to EPA and DHA.

17) This meta-analysis indicates that n-3 PUFA supplementation in patients with rheumatoid arthritis or joint pain secondary to inflammatory bowel disease and dysmenorrhea, reduces patient assessed joint pain intensity, morning stiffness, number of painful and/or tender joints, and reduces NSAID consumption.

Omega-3 Fatty Acids for Neuropathic Pain: Case Series
The Clinical Journal of Pain

February 2010, Vol. 26, No, 2, pp 168-172

Ko GD, Nowacki NB, Arseneau L, Eitel M, Hum A
The authors are from the University of Toronto, Canada

KEY POINTS FROM DAN MURPHY

1) The benefits of omega-3 fatty acid supplementation are well documented in the literature for the prevention and management of a wide variety of health conditions including:
Inflammatory joint pain
Chronic spinal pain
Autoimmune disease
Cardiovascular disease
Depression
Fibromyalgia syndrome

2) The probable mechanism for the benefit of omega-3 supplementation in the treatment of inflammatory pain is through the suppression of the pro-inflammatory eicosanoids (Prostaglandin E2 [PGE2], Leukotriene B4 [LTB4]).

3) This is the first study to assess the use of omega-3 supplements in the treatment of neuropathic pain. Neuropathic pain can exist in the absence of pro-inflammatory eicosanoids. Rather, neuropathic pain is linked to pro-inflammatory cytokines (proteins made by immune system cells).

4) These authors present 5 case studies on chronic neuropathic pain patients with excellent results. The patients were treated with high oral doses of omega 3 fish oil (varying from 2400-7200 mg/day of EPA + DHA). Results were excellent both subjectively and objectively for all five, which included:
Cervical radiculopathy
Thoracic outlet syndrome
Fibromyalgia
Carpal tunnel syndrome
Burn injury

5) No serious adverse effects were reported from taking high oral doses of omega 3 fish oil (varying from 2400-7200 mg/day of EPA + DHA).

6) Patients taking Coumadin (or other blood thinners) should slowly begin to take omega-3s while monitoring clotting times.

7) Because of the blood thinning effects of omega-3s, patients should stop taking them 2 weeks prior to surgery, dental work, or invasive procedures such as a colonoscopy.

8) Patients taking omega-3s should have their blood analyzed for the arachidonic acid (omega-6) (AA) / eicosapentaenoic acid (omega-3) (EPA) ratio. **AA/EPA**

9) "An optimal [**AA/EPA**] ratio for cardiovascular health is 1.5/1 to 3/1."

10) The lab analysis of [**AA/EPA**] is especially important if the patient is taking more than 7,500 mg of EPA + DHA per day.

11) "An AA/EPA ratio of 0.5/1 is associated with an increased risk for hemorrhagic stroke."

12) Fish oils should be purified.

13) "A recommended conservative dose is 2,700 mg of EPA + DHA. However, a more aggressive approach for more severe pain can be up to 7,500 mg of EPA + DHA. This will require serum laboratory tests to monitor AA/EPA ratio."

14) "Patients should clearly be instructed to take only omega-3 and not omega omega-3 to omega-6 to omega-9. The omega-6 fatty acids are pro-inflammatory and the use of such products will not help in relieving pain."

15) Although omega-6 fatty acids are essential, they are already in excess in the typical American diet.

16) Pain patients must also reduce their intake of arachidonic acid (omega-6) (AA), which is commonly found in red meat and fried foods.

17) The conversion of alpha linolenic acid (ALA) (plant omega-3) to the anti-inflammatory EPA omega-3 is enhanced with adequate levels of vitamin B6, magnesium, and zinc.

18) The conversion of alpha linolenic acid (ALA) (plant omega-3) to the anti-inflammatory EPA omega-3 is impaired by trans fats and caffeine.

19) "To conclude, the use of omega-3 fatty acids supplements for the treatment of neuropathic pain shows promise, on the basis of these case studies."

Dietary omega-3 fatty acids and risk of type-2 diabetes: Lack of antioxidants?

American Journal of Clinical Nutrition
August 2011; Vol. 94; No. 2; pp. 618-619

Bjarne Osterud

This author cites evidence that increased intake of dietary omega-3 fatty acids may increase the risk of type-2 diabetes.

Yet, he also notes that type-2 diabetes "is strongly associated with pro-inflammatory products," and therefore omega-3 fatty acids should prevent type-2 diabetes because they suppress the production of these pro-inflammatory products, noting that it "seems difficult to understand why long-chained omega-3 fatty acids are associated with [increased] risk of type-2 diabetes."

"Intake of omega-3 fatty acids may not always be beneficial because incorporation of these polyunsaturated fatty acids (PUFAs) in the cell membranes makes the cells more susceptible to oxidation if there is a lack of antioxidants where the omega-3 fatty acids are present in the membranes."

"Contrary to an anti-inflammatory effect, PUFAs may cause oxidative stress whereby the production of pro-inflammatory products is enhanced."

"Everywhere where PUFAs are present in live material, there is always an excess of antioxidants, which are removed when the omega-3 fatty acids are refined or when they are isolated. This means that these fatty acids when taken as dietary supplements may cause severe oxidation in their local environment in which support of antioxidants may be quite low. Omega-3 fatty acids may thereby cause oxidative stress and subsequently an increase in pro-inflammatory products known to promote type-2 diabetes."

The lack of type-2 diabetes in native Greenlanders despite their high intakes of omega-3 fatty acids in their diet through seal and whale blubber may be credited to the fact that "their diet is also rich in natural antioxidants associated with omega-3 fatty acids contained in blubber."

Thus, antioxidants may be required for the anti-inflammatory benefit of omega-3 fatty acids in humans.

COMMENTS FROM DAN MURPHY

Over the years a number of other authors have made similar claims as this author: increasing the intake of omega-3 fatty acids increases the requirement for antioxidants. As examples:

Natural Strategies For Cancer Patients
Russell Blaylock, MD
Twin Streams Books, 2003

"The universal problem with polyunsaturated oils, even the good ones, is that they oxidize very easily. When an oil oxidizes, it becomes rancid. Rancid oils can produce harmful substances (lipid peroxides) and free radicals." p. 134

Healthy Fats For Life
Preventing and Treating Common Health Problems with Essential Fatty Acids
Lorna Vanderhaeghe and Karlene Kasrst
Wiley, 2004

"Research has consistently shown that increased intake of essential fatty acids increases the need for antioxidants." p. 184

When taking essential fatty acids, "increasing your antioxidant consumption to prevent free radical damage is very important." p. 185

•••••

I have always advocated that one should take a combination of antioxidants when consuming fish oil. The antioxidant formula I use is a network of exogenous antioxidants that help the cell produce the endogenous antioxidant glutathione, from the book Nutrition and Immune Function, edited by Phillip Calder, CABI Publishing, 2002. It includes a ratio of vitamin E, vitamin C, B2, B6, and B12, plus some necessary minerals. The formula is available from **Nutri-West (800-443-3333)**, and is called **Complete Omega-3 Co-Factors**. I advocate taking 1 **Co-Factor** per gram of fish oil consumed.

Essential Fatty Acid Balancing Blood Work
For The Life Chiropractic College West Rugby Teams

Eicosanoids are hormones that are derived from 20-carbon long fats.

The eicosanoid hormones derived from the 20-carbon long omega-**6** fatty acid ***arachidonic acid*** are inflammatory.

The eicosanoid hormones derived from the 20-carbon long omega-**3** fatty acid ***eicosapentaenoic acid*** are **anti**-inflammatory.

Inflammatory	***Anti*-Inflammatory**
Omega-**6** Fatty Acids	Omega-**3** Fatty Acids
Corn, Soy, Sunflower, Safflower, Cottonseed, Peanut, Canola	Flax
Grained-out Meat / Eggs	Wild Fish and Omega-3 Eggs
Arachidonic Acid (*AA*) 20:4n-6	Eicosapentaenoic Acid (*EPA*) 20:5n-3

In their 1996 book *Protein Power*, physicians Michael Eades, MD, and Mary Eades, MD, note:

"Eicosanoids, a gang of at least 100 powerful hormone-like substances that control virtually all physiological actions in your body."

Eicosanoids "are the most powerful agents known to [hu]mans, yet they are totally controlled by the diet."

"The most important thing about eicosanoids is to keep them in balance."

"...the maintenance of the dynamic balance between the various eicosanoids [is] the *definition* of optimal health."

In his 2008 book *In Defense of Food*, Michael Pollen, PhD, notes that the balance of eicosanoids is so critical for human heath, wellness, longevity, and peak performance that he refers to it as ***The Theory of Everything***.

In his 2008 book *Toxic Fat*, biochemist Barry Sears. PhD, notes:

"The underlying cause of chronic disease comes from increased production of a natural fatty acid called arachidonic acid (AA), which can be incredibly toxic at high concentrations. This is the toxic fat that is key to not only understanding our obesity epidemic but also providing the linkage between obesity and chronic disease."

"Classic inflammation hurts; silent inflammation slowly kills."

"All forms of inflammation (including silent inflammation) are ultimately controlled by a group of hormones known as eicosanoids."

"It is the balance of eicosanoids in your body that is the ultimate key to wellness."

"All eicosanoids are ultimately derived from dietary fat, in particular the polyunsaturated essential fatty acids that must be supplied by the diet."

"Virtually all chronic diseases can be viewed as a consequence of a continuing imbalance of good and bad eicosanoids."

"The AA/EPA ratio will tell you the extent of silent inflammation in the body."

••••••••••

Understanding The Numbers

The AA/EPA ratio needs to be between 1.5—4.0/1

- A ratio above 4/1 is a problem
- A ratio above 10/1 is a serious problem, unacceptable
- A ratio above 15/1 is a critical problem

Total EPA should be >4.0% of membrane fatty acids.

Total AA should be < 9.0% of membrane fatty acids.

It is unlikely that one can stay in the optimal ratio zone by taking less than 3,000 mg of EPA+DHA per day. In his 2014 book *Wheat Belly Total Health*, cardiologist William Davis, MD, notes:

"I advocate an intake of 3,000 to 3,600 mg per day (the dose of combined omega-3 fatty acids, EPA and DHA, not fish oil)."

It takes 4 months of supplementation to change the ratios. I advise:

- 3,000 mg/day of EPA+DHA for everyone as a maintenance dose.
- 5,000 mg/day of EPA+DHA if the ratio is above 10/1.
- 7,500 mg/day of EPA+DHA if the ratio is above 15/1.
- 10,000 mg/day of EPA+DHA if the ratio is above 20/1.

Cytokines

Cytokines are small proteins that function as singling molecules for intercellular communication.

Virtually all nucleated cells can produce cytokines, but they are primarily produced by endothelial cells (vascular system cells) and macrophages (innate immune cells).

Cytokines can be either inflammatory or anti-inflammatory. The primary inflammatory cytokines are Interleukin-1 (IL-1), Interleukin-6 (IL-6), and Tumor Necrosis Factor-alpha (TNF-a).

Interleukin-1 plays a central role in the regulation of immune and inflammatory responses to infections or trauma.

Interleukin-6 is secreted by T cells and macrophages to stimulate immune response during infection or after trauma, leading to inflammation.

Tumor necrosis factor (cachexin or cachectin) is involved in systemic inflammation as a portion of the acute phase response. It is produced by macrophages. Dysregulation of TNF production is implicated in a variety of human diseases, including Alzheimer's, cancer, major depression, and inflammatory bowel disease.

Younger Next Year®

Live Strong, Fit, and Sexy—Until You're 80 and Beyond

HARRY'S RULES

1
Exercise six days a week
for the rest of your life.

2
Do serious aerobic exercise four days a week
for the rest of your life.

3
Do serious strength training, with weights,
two days a week for the rest of your life.

4
Spend less than you make.

5
Quit eating crap!

6
Care.

7
Connect and commit.

by Chris Crowley &
Henry S. Lodge, M.D.

2007

WORKMAN PUBLISHING • NEW YORK

Cytokines

"Proteins that control inflammation are called cytokines, and they regulate every aspect of your biology."

Balance

Interleukin-6	**Interleukin-10**
Inflammatory	Anti-Inflammatory
Decay	Regenerative
Degenerative	Restorative
	Growth
	"is the master chemical for repair and growth."
	"is the key because growth is the magic you are after"
	It's effect is to build a "stronger, healthier, younger body."

Triggers

Inflammatory Diet (O-6/O-3)	Regular Vigorous Exercise
Ageing	
Chronic Stress	
Being Sedentary	
A burst of IL-6 is ***not*** bad because	It triggers the release of IL-10
It is the slow steady low-grade secretion, trickle, of IL-6 that is bad because	It does ***not*** trigger the release of IL-10
	Low Level Laser Therapy
	Chiropractic Adjustment

Omega-3 fatty acids and synovitis in osteoarthritic knees

Nature Reviews Rheumatology
April 2012 [epub]

Leslie G. Cleland and Michael J. James

KEY POINTS FROM THIS ARTICLE:

1) "Recently reported associations between synovitis, cartilage damage and plasma levels of omega-3 and omega-6 fatty acids in patients with osteoarthritis suggest that fish oil supplements might be beneficial additions to the therapeutic regime in this disease."

2) Osteoarthritis (OA) is characterized by degeneration and loss of articular cartilage, and accompanying synovial inflammation (synovitis).

3) Synovitis can cause swelling, tenderness and restricted movement in OA patients.

4) In OA, inflammatory cytokines (IL-1B, TNF, IL-6) amplify the pathophysiological processes that result in joint damage.

5) In OA, low-grade inflammation influences the long-term outcomes in patients.

6) Treatments that safely reduce the inflammation underlying cartilage degeneration in OA are important.

7) Plasma levels of long chain omega-6 (n-6) and omega-3 (n-3) fatty acids correlate with MRI evidence of synovitis in the knees of patients with OA.

8) The inflammatory effects of omega-6 eicosanoids derived from arachidonic acid are greater than the anti-inflammatory effects derived from the omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).

9) The "availability of arachidonic acid for production of inflammatory eicosanoids could be a predisposing factor for synovitis in early OA." **[Key Point]**

10) The n-6 (AA) and n-3 (EPA) fatty acid ratios could be more important than the absolute amounts of these fatty acids.

11) "EPA and DHA reduced expression of degradative enzymes and inflammatory cytokines."

12) Long-term fish oil treatment at anti-inflammatory doses inhibits prostaglandin E2 synthesis.

13) Established benefits for omega-3-rich supplements in patients with rheumatoid arthritis include:

- Reduced symptom severity
- Increased remission
- Improvement in markers of cardiovascular risk
- Decreased use of NSAIDs

14) "The reduced use of NSAIDs is important as these drugs—whilst providing a prompt analgesic effect—have not been shown to improve long-term outcomes in RA, and their use can distract clinicians from prescribing more effective long-term disease-suppressing agents."

15) "Moreover, NSAIDs are associated with an increased risk of potentially life-threatening gastrointestinal bleeding and serious thrombotic cardiovascular events, including myocardial infarction and stroke." **[Important]**

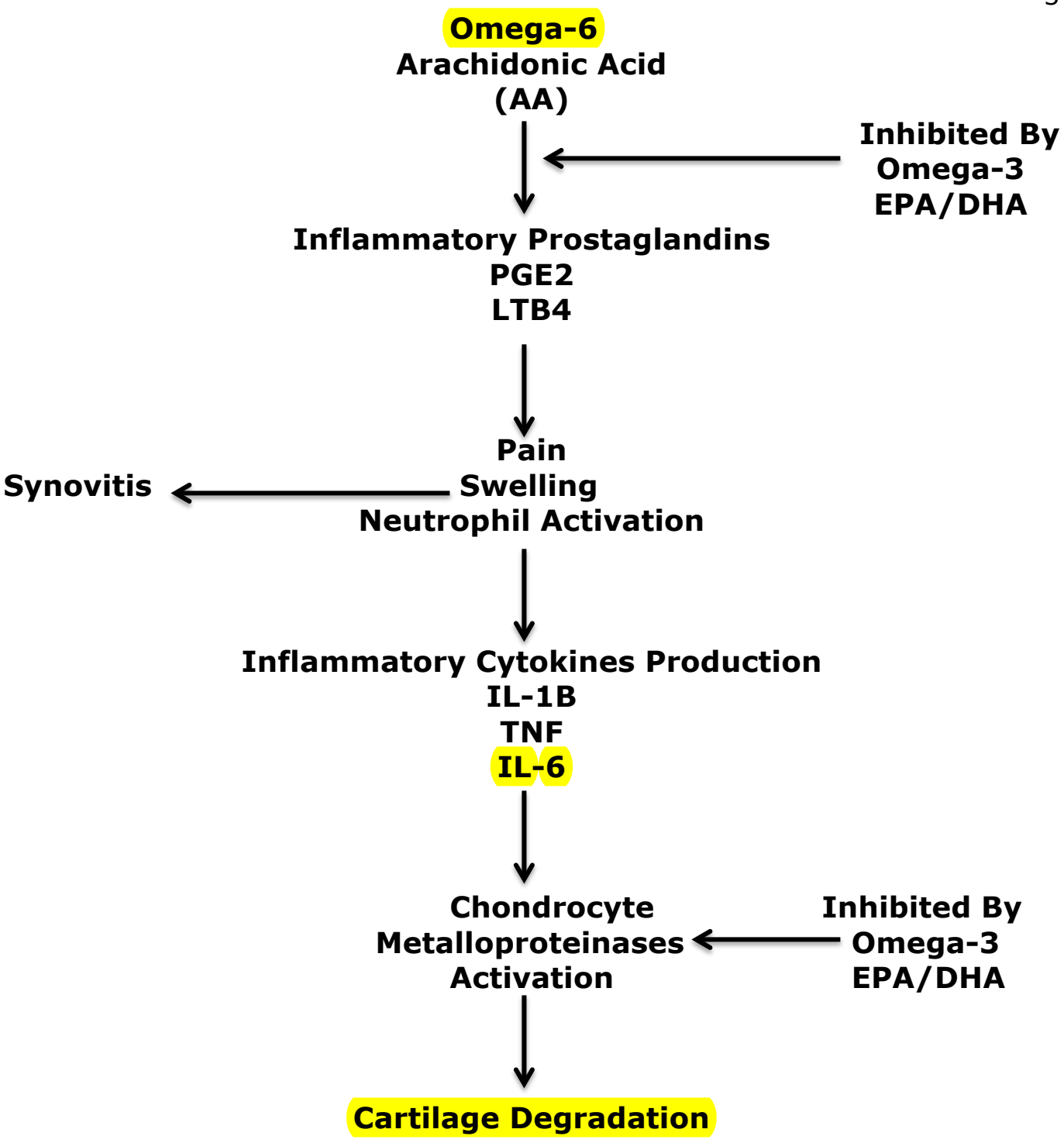
16) "The NSAID-sparing effect and the direct collateral cardiovascular benefits are important potential advantages of fish oil use for long-term analgesia in a disease such as osteoarthritis."

17) Treatment with omega-3 fatty acids "has the potential to play a key part in the management of patients with osteoarthritis."

18) The omega-3 fats in dietary fish oil, EPA and DHA, inhibit the omega-6 fatty acid arachidonic acid cascade into the inflammatory prostaglandins and leukotrienes such as PGE2 and LTB4.

19) "Competitive inhibition of arachidonic acid metabolism by EPA and DHA could reduce inflammation, pain and synovitis."

20) EPA and DHA suppress chondrocyte metalloproteinases production, and dietary fish oil has a protective effect on cartilage and subchondral bone in OA.


Omega-6
Arachidonic Acid
(AA)
Inhibited By
Omega-3
EPA/DHA
Inflammatory Prostaglandins
PGE2
LTB4
Pain
Swelling
Neutrophil Activation
Synovitis
Inflammatory Cytokines Production
IL-1B
TNF
IL-6
Chondrocyte
Metalloproteinases
Activation
Inhibited By
Omega-3
EPA/DHA
Cartilage Degradation

Natural anti-inflammatory agents for pain relief

Surgical Neurological International
December 2010

Joseph C. Maroon, Jeffrey W. Bost, and Adara Maroon
Department of Neurosurgery, University of Pittsburgh Medical Center, Pittsburgh, PA, and Vanderbilt University, Nashville, TN

FROM ABSTRACT:

The use of both over-the-counter and prescription nonsteroidal medications is frequently recommended in a typical neurosurgical practice. But persistent long-term use safety concerns must be considered when prescribing these medications for chronic and degenerative pain conditions.

Although nonsteroidal medications can be effective, herbs and dietary supplements may offer a safer, and often an effective, alternative treatment for pain relief, especially for long-term use.

SIDE EFFECTS OF STEROID DRUGS:

Increased Infection	Dermatitis	Fluid retention	Hyperglycemia
Mood Changes	Hypertension	Stomach Ulcers	Osteoporosis
Cataracts	Increased Appetite	Weight Gain	Depression
Impaired Wound Healing	Adrenal Suppression	Fat Deposits Upper Back-Stomach	Fat Deposits Face-Chest

THESE AUTHORS ALSO NOTE:

"In most cases, the genesis of pain is inflammatory, regardless of the etiology."

This inflammation has 2 primary causes:

1) Inflammatory hormones (PGE2, LTB4, etc.)
[derived from the omega-6 fatty acid arachidonic acid]

2) Inflammatory cytokines (interleukin ((IL))-1a, IL-1b, IL-6 and tumor necrosis factor ((TNF-a)).
[proteins that are derived form the immune system cells]

"The use of non-steroidal anti-inflammatory drug (NSAID) medication is still the mainstay of most classically taught clinicians for joint and spine related inflammatory pain, despite their commonly known side effects."

The pro-inflammatory cytokines stimulate the production of the pro-inflammatory hormone prostaglandin E2 (PGE2).

NSAIDs' ability to interfere with the production of prostaglandin E2 (PGE2) is the major mechanism for the anti-inflammatory success of these drugs.

INFLAMMATORY PATHWAYS

"Prostaglandins act as short-lived localized hormones that can be released by any cell of the body during tissue, chemical, or traumatic injury, and can induce fever, inflammation, and pain, once they are present in the intercellular space."

Thromboxane hormones increase the inflammatory response.

"A major component of the inflammatory pathway is called the arachidonic acid pathway because arachidonic acid is immediately released from traumatized cellular membranes."

Cell membrane trauma releases arachidonic acid. Arachidonic acid is then transformed into the pro-inflammatory hormones prostaglandins and thromboxanes through the enzymatic action of cyclooxygenase.

"Nonselective NSAIDs' major side effects include significant gastrointestinal upset, gastritis, ulceration, hemorrhage, and even death. By blocking COX-1, which also normally acts to protect the gastrointestinal mucosa, nonselective NSAIDs and aspirin can cause significant gastric tissue damage."

"NSAIDs can delay muscle regeneration and may reduce ligament, tendon, and cartilage healing."

NSAIDs also have adverse effects on kidney function. "The National Kidney Foundation asserts that approximately 10% of kidney failures per year are directly correlated to substantial overuse of NSAIDs."

Selective COX-2 inhibiting NSAIDs were thought to reduce inflammatory pain without enhancing GI bleeding. Celebrex was FDA approved in 1998, followed by the approval of Vioxx and Bextra in 1999. These drugs "quickly became the mainstay for the treatment of chronic pain conditions related to inflammation."

On September 30, 2004, Vioxx was withdrawn because it "doubled the risk of serious thromboembolic events, including myocardial infarction."

Natural compounds for inflammation

"Because of the significant side effect profiles of steroidal and NSAID medications, there is a greater interest in natural compounds, such as dietary supplement and herbal remedies, which have been used for centuries to reduce pain and inflammation."

Nuclear Factor kappa-B (Nf-kB) controls the transcription of DNA for the perpetuation of the inflammatory immune response. "It acts as a switch to turn inflammation on and off in the body."

"Plant- and animal-derived nutraceutical preparations have been used for hundreds and even thousands of years to obtain effective pain relief. Herbal medications are becoming increasingly popular because of their relatively few side effects."

"The US governmental agencies, through the FDA and others, routinely inspect the manufacture of vitamins or supplements made in this country, as they do for any other food product."

"Products such as omega-3 essential fatty acids (EFAs) (O3) do have strong scientific support to be considered as an alternative and/or complementary agent to NSAIDs. Published studies have shown the effectiveness of O3 to successfully treat spine-related pain."

Omega-3 EFAs (fish oil)

The use of fish oil for the treatment of muscular, skeletal, and discogenic diseases, can be traced back to the late 18th century.

"Research has shown that the omega-3 polyunsaturated fatty acids are some of the most effective natural anti-inflammatory agents available." [7 references]

"With the discovery that vascular inflammation is the underlying cause of coronary artery disease, fish and fish oil supplements are now recommended by the American Heart Association for the prevention of this disease."

"Countries that have the highest fish consumption also have a lower incidence of neurodegenerative disease and depression."

"The biological basis for the effectiveness of fish oil in treating arthritis has been well documented with many positive clinical studies, when compared to traditional pharmaceutical anti-inflammatory agents."

"The active ingredients in fish oil, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), enhance the conversion of COX to prostaglandin E3. A natural anti-inflammatory agent, prostaglandin E3 competitively inhibits the effects of the arachidonic acid conversion to prostaglandin E2, a highly inflammatory substance."

"Prostaglandin E3 also inhibits the synthesis of TNF-a and IL1b, both of which are inflammatory cytokines."

"The EPA and DHA can inhibit the 5-LOX pathway, which converts arachidonic acid to inflammatory leukotrienes."

When EPA and DHA are incorporated into articular cartridge chondrocyte cell membranes, there is a dose-dependent decrease in the expression and activity of the enzymes that degrade cartilage.

Omega-3 EFA, found in fish oil, can directly reduce the degenerative enzymes and reduce the inflammation in synovial cartilage.

Belching may occur if fish oil supplements are not taken with meals.

"Persons on a regimen of anticoagulant medications should not take omega-3 EFAs because of the possibility of increasing the bleeding potential."

White willow bark

Bark from the white willow tree has analgesic and antipyretic properties.

Salicin from white willow bark is converted to salicylic acid by the liver and is considered to have fewer side effects than aspirin.

White willow bark should "not be used in children (to avoid the risk of Reye's syndrome), or in patients with peptic ulcer disease, poorly controlled diabetes, hepatic or renal disorders, or other conditions in which aspirin would be contraindicated. The usual dose of white willow bark is 240 mg/day."

Curcumin (turmeric)

"Curcumin is a naturally occurring yellow pigment derived from turmeric, a flowering plant of the ginger family. It has traditionally been used as a coloring and flavoring spice in food products. Curcumin is an anti-inflammatory agent, and has antioxidant, anti-inflammatory, and antineoplastic effects."

Curcumin is known to inhibit inflammation by suppressing NF-kB and COX enzymes, and "it may be considered a viable natural alternative to nonsteroidal agents for the treatment of inflammation."

"The usual dosage of standardized turmeric powder is 400–600 mg taken three times per day."

Green tea

Green tea has cardiovascular and cancer preventative characteristics due to its antioxidant properties; its use in the treatment of arthritic disease as an anti-inflammatory agent is more recent.

The constituents of green tea include polyphenolic compounds called catechins; epigallocatechin-3 galate is the most abundant catechin in green tea.

Epigallocatechin-3 galate inhibits NF-kB and the production of pro-inflammatory cytokines.

Green tea inhibits the aggrecanases that degrade cartilage.

Green tea has both anti-inflammatory and chondroprotective effects.

Increased green tea consumption in Asia may lead to significant cardiovascular, neuroprotective and cancer prevention properties.

The recommendation for green tea consumption is 3–4 cups a day, or green tea extract dosage of 300–400 mg a day.

Pycnogenol (maritime pine bark)

"Pycnogenol is derived from the bark of the maritime pine tree and has been used for more than 2000 years."

Pycnogenol is helpful for wound healing, treating scurvy, healing of ulcers, and reducing vascular inflammation.

Pycnogenol contains a potent blend of active polyphenols, which includes catechin, taxifolin, procyanidins, and phenolic acids. It is one of the most potent antioxidant compounds currently known.

Pycnogenol inhibits NF-kB production of pro-inflammatory cytokines.

"Studies have shown that pycnogenol is 50–100 times more potent than vitamin E in neutralizing free radicals and that it helps to recycle and prolong the activity of vitamins C and E."

"Studies have shown pycnogenol to be effective in reducing blood pressure and reducing the risk of venous thrombosis by its effect on vascular endothelium. The usual dosage is 100–200 mg daily."

Because pycnogenol enhances immune system function, it should not be taken by patients who are being treated with immunosuppressants or by those receiving corticosteroid drugs, both of which have the opposite effect on the immune system.

Boswellia serrata resin (Frankincense)

Boswellia possesses anti-inflammatory, anti-arthritic, and analgesic properties.

Boswellia can inhibit the leukotriene biosynthesis, thus affecting various inflammatory diseases that are perpetuated by leukotrienes.

Clinically, Boswellia is used in the treatment of degenerative and inflammatory joint disorders.

"A combination of Boswellia and curcumin showed superior efficacy and tolerability compared with nonsteroidal diclofenac for treating active osteoarthritis."

"Boswellia typically is given as an extract standardized to contain 30-40% boswellic acids (300-500 mg two or three times/day)."

Resveratrol

Resveratrol is a plant-based polyphenol molecule that is found in many different plant sources, but the skins of red wine grapes are believed to have the highest concentration.

In plants, resveratrol protects the plant from infection, excessive UV radiation and aids in general plant defense.

"Resveratrol has also been found to have significant anti-mutation, anti-inflammatory, antioxidant and DNA protective actions, when consumed by animals and humans."

"Most of the active research with resveratrol has been done in neuro and cardioprotection, but several studies are being reported on resveratrol's use for arthritic joint pain."

Resveratrol inhibits NFkB and the production of pro-inflammatory cytokines.

The typical dose for Resveratrol is 50 to 500 mg daily.

Uncaria tomentosa (cat's claw)

The bark of cat's claw is used to treat arthritis, bursitis, and intestinal disorders. The active ingredients appear to be polyphenols (flavonoids, proanthocyanidins, and tannins), alkaloids, and sterols.

Cat's claw inhibits NFkB and the production of pro-inflammatory cytokines.

"Cat's claw can be consumed as a tea (1000 mg root bark to 8 oz water), or as a dry, standardized extract in a capsule (20-60 mg daily)."

Capsaicin (chili pepper)

Capsicum accentuates chili's stinging pungency.

"Capsaicin produces highly selective regional anesthesia by causing degeneration of capsaicin-sensitive nociceptive nerve endings which can produce significant and long-lasting increases in nociceptive thresholds."

Capsaicin inhibits NF-kB, thus producing an anti-inflammatory effect.

CONCLUSIONS

Anti-inflammatory agents such as NSAIDs "can have undesirable side effects such as gastric ulceration and, infrequently, myocardial infarction and stroke."

"For centuries, natural anti-inflammatory compounds have been used to mediate the inflammatory process and often with fewer side effects [than NSAIDs]."

●●

PLA2 = Phospholipase A2 (the enzyme that cleaves AA from the cell membrane following trauma / injury)
AA = Arachidonic Acid (an omega-6 fatty acid commonly found in cell membranes)
LOX = Lipoxygenase (the enzymes that converts AA into pro-inflammatory LT hormones)
COX = Cycloxygenase (the pro-inflammatory enzymes that convert AA into the pro-inflammatory PG and TXA hormones)
LT = Leukotrienes
TXA = Thromboxanes
PG = Prostaglandins

Interleukin-1a = IL1a (a pro-inflammatory cytokine protein)
Interleukin-1b = IL1b (a pro-inflammatory cytokine protein)
Interleukin-6 = IL6 (a pro-inflammatory cytokine protein)

Tumor Necrosis Factor alpha = TNFa (a pro-inflammatory cytokine protein)

NF-kB= Nuclear Factor kappaB (pro-inflammatory protein that lives in the cell cytoplasm)
IkB = Inhibitory kappaB (a protein that when attached to NFkB inhibits its pro-inflammatory influence; consequently it's an anti-inflammatory protein)
IkBK = Inhibitory kappaB Kinase (an enzyme that cleaves NFkB away from its inhibitory Ikb protein, allowing NFkB to cross into the nucleus and activate the genes that produce pro-inflammatory cytokines)
CK = Cytokines (pro-inflammatory proteins made by immune system cells)

EPA = Eicosapentaenoic Acid (anti-inflammatory omega-3 fatty acid)

Is Vitamin D Deficiency associated with Non Specific Musculoskeletal Pain?

Global Journal of Health Science

Vol. 5, No. 1; 2013; pp. 107-111

Mahnaz Abbasi, Sima Hashemipour, Fatemeh Hajmanuchehri, Amir Mohammad Kazemifar
From the Metabolic Diseases Research Center, Qazvin University of Medical Science, Iran. This study evaluated 65 adult patients living in Iran.

The aim of this study is evaluation of the association of musculoskeletal pain with vitamin D deficiency and the response of the patients to vitamin D supplementation.

KEY POINTS FROM THIS STUDY:

1) Vitamin D deficiency is common worldwide.

2) Vitamin D deficiency is associated with non-specific musculoskeletal pain.

3) "Treatment with vitamin D can relieve the pain in a majority of the patients with vitamin D deficiency. Lack of response can be due to an insufficient increase in serum vitamin D concentration."

4) There are physiologic differences in the intestinal absorption of vitamin D.

5) "Calcium absorption from the GI is reduced in vitamin D deficiency."

6) "Mild vitamin D deficiency may produce a variety of musculoskeletal pains such as fibromyalgia-like pain, low back pain, and arthralgia." **[Key Point]**

7) Those with vitamin D deficiency were treated with 50,000 oral units of vitamin D3 per week **[averaging 7,143 IU/day]** for 12 weeks and 1,000 mg/day elemental calcium. Three months after end of the treatment they were reassessed for response of their pain to the treatment using the Visual Assessment Score (VAS).

8) 95.4% of the patients had vitamin D deficiency (serum 25 (OH) D concentration less than 50 nmol/l). [50 nmol/l = **20 ng/ml**]

9) In 85.5% of those with vitamin D deficiency, supplementation reduced their VAS scores more than 60%.

10) In 75.8% of those with vitamin D deficiency, supplementation completely eliminated their pain.

11) Supplementing with vitamin D significantly raised the serum concentrations of 25(OH)D, and especially in those who responded favorably to the intervention. **[This suggests that vitamin D supplementation in higher doses or for longer periods may improve musculoskeletal pain in the poorer responders].**
"In patients who respond to vitamin D supplementation, more notable rise in serum concentrations of 25(OH)D was detected."

12) These authors review literature showing:
- 2003: 93% of patients with musculoskeletal pain have vitamin D deficiency.
- 2003: 83% of patients with low back pain have vitamin D deficiency.
- 2010: 63% of patients with musculoskeletal pain have vitamin D deficiency.

13) Dr. Michael Hollick, MD, [the discoverer of the active form of vitamin D] proposes the following biological mechanism:

- Low vitamin D causes low GI absorption of calcium.
- Low calcium increases secretion of parathyroid hormone (PTH).
- Increased PTH increases bone osteoclast activity.
- Increased osteoclast activity causes lower bone matrix mineralization.
- Lower bone matrix mineralization absorbs water.
- Bone water absorption causes subperiosteal space edema.
- Subperiosteal space edema produces bone pain.

14) This study "confirmed that vitamin D supplementation can relieve the pain in majority of the patients" with musculoskeletal pain.

15) The rise in serum 25 (OH) D concentrations after vitamin D supplementation is variable between patients. Therefore, when musculoskeletal pain patients do not respond to vitamin D supplementation, their serum levels should be retested to assess their effectiveness of that level of supplementation for that individual.

16) It is suggested that the pain associated with vitamin D deficiency is usually sensed on the bone or muscle; bone tenderness is noted at the sternum, tibia, radius or ulna. Low back, thoracic, shoulder, ribs and pelvic pain is also common in those with vitamin D deficiency.

17) The findings of present the study show that vitamin D deficiency is widespread and associated with musculoskeletal pain which is relieve with vitamin D supplementation; "in pain nonresponders reassessment of serum 25(OH) D concentrations is recommended."

What We Have Learned About Vitamin D Dosing?

Integrative Medicine
Vol. 9, No. 1, Feb/Mar 2010

Joseph Pizzorno, ND, Editor in Chief

BACKGROUND FROM DAN MURPHY

The world standard uses nmol/l, while US standard uses mg/dl.

For vitamin D, to convert mg/dl to nmol/l, divide the mg/dl by 2.5.

For vitamin D, to convert nmol/l to mg/dl, just multiply by 2.5.

KEY POINTS FROM THIS ARTICLE:

1) "Over the past several years, the surprising prevalence of vitamin D deficiency has become broadly recognized."

2) Vitamin D deficiency is linked to:
Osteoporosis
Cardiovascular disease
Cancer
Autoimmune diseases
Multiple sclerosis
Pain
Loss of Cognitive function
Decreased strength
Increased rate of all-cause mortality

3) "Deficiency of vitamin D is now recognized as a pandemic, with more than half of the world's population at risk."

4) Approximately 50% of the healthy North American population and more than 80% of those with chronic diseases are vitamin D deficient.

5) 80% of healthy Caucasian infants are vitamin D deficient. [And the rate of vitamin D deficiency tends to be greater in African American and Hispanic children].

6) Those with vitamin D deficiency experience 39% higher annual healthcare costs than those with normal levels of vitamin D.

7) Suggested levels of vitamin D as measured by 25(OH)D3 is:

Caucasians	125 – 175 nmol/l	=	50 - 70 mg/dl
Hispanics	100 – 150 nmol/l	=	40 - 60 mg/dl
African Americans	80 – 120 nmol/l	=	32- 48 mg/dl

8) The minimum blood levels of vitamin D [25(OH)D3] is 80 nmol/l (32 mg/dl).

9) Prolonged intake of 10,000 IU of supplemental vitamin D3 "is likely to pose no risk of adverse effects in almost all individuals."

10) The maximum safe levels for vitamin 25(OH)D3 in the blood is 275 nmol/l (100 mg/dl).

11) Sarcoidosis patients (and other granulomatous diseases) should not supplement with vitamin D because it increases granuloma production increasing the risk of hypercalcemia.

12) A loading dose of supplemental vitamin D3 of 10,000 IU/day for 3 months and maintenance dose of 5,000 IU/day "is not enough for most people in northern climes."

13) The loading dose of supplemental vitamin D3 should be about 20,000 IU/day for 3 – 6 months with a maintenance dose of 5,000 IU/day. Those taking this amount of supplemental vitamin D3 should periodically have their serum 25(OH)D3 levels measured.

COMMENTS FROM DAN MURPHY

The lab we use to test blood vitamin D3 [25(OH)D3] uses a finger prick analysis:
ZRT Laboratory
8605 SW Creekside Pl
Beaverton, OR 97008
866-600-1636
www.zrtlab.com
Vitamin D Testing Finger prick

The vitamin D3 my family takes is ***Complete Hi D3***, from Nutri-West (5,000 IU):
800-443-3333
The primary researcher on this product was Don Bellgrau, PhD. Dr. Bellgrau is a tenured Professor of Immunology and Medicine at the University of Colorado, Denver, where he is a Program Leader in Immunology and Immunotherapy at the Cancer Center on vitamin D3 supplementation. Dr. Bellgrau has conducted experiments with nutrients/vitamin D and immune cells. He has published in over 100 peer-reviewed articles, including the Journal of Neurooncology, Nature, Clinical Immunology, Cancer Research, Cancer Immunology and Immunotherapy, and Cell Transplantation.

PubMed

Display Settings: Abstract

Support Care Cancer. 2009 Nov;17(11):1409-15. doi: 10.1007/s00520-009-0603-9. Epub 2009 Feb 22.

Cyclooxygenase-2 and vascular endothelial growth factor expression in 5-fluorouracil-induced oral mucositis in hamsters: evaluation of two low-intensity laser protocols.

Lopes NN, Plapler H, Chavantes MC, Lalla RV, Yoshimura EM, Alves MT.

Department of Experimental Surgery, Federal University of São Paulo, São Paulo, Brazil, CEP 04023-062. nnflopes@terra.com.br

Abstract

GOAL OF WORK: The aim of this study was to investigate the mechanisms whereby low-intensity laser therapy may affect the severity of oral mucositis.

MATERIALS AND METHODS: A hamster cheek pouch model of oral mucositis was used with all animals receiving intraperitoneal 5-fluorouracil followed by surface irritation. Animals were randomly allocated into three groups and treated with a 35 mW laser, 100 mW laser, or no laser. Clinical severity of mucositis was assessed at four time-points by a blinded examiner. Buccal pouch tissue was harvested from a subgroup of animals in each group at four time-points. This tissue was used for immunohistochemistry for cyclooxygenase-2 (COX-2), vascular endothelial growth factor (VEGF), and factor VIII (marker of microvessel density) and the resulting staining was quantified.

MAIN RESULTS: Peak severity of mucositis was reduced in the 35 mW laser group as compared to the 100 mW laser and control groups. This reduced peak clinical severity of mucositis in the 35 mW laser group was accompanied by a significantly lower level of COX-2 staining. The 100 mW laser did not have an effect on the severity of clinical mucositis, but was associated with a decrease in VEGF levels at the later time-points, as compared to the other groups. There was no clear relationship of VEGF levels or microvessel density to clinical mucositis severity.

CONCLUSION: The tissue response to laser therapy appears to vary by dose. Low-intensity laser therapy appears to reduce the severity of mucositis, at least in part, by reducing COX-2 levels and associated inhibition of the inflammatory response.

PMID: 19234862 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms, Substances

LinkOut - more resources

Where It Hurts

U.S. ADULTS REPORTED FEELING PAIN IN THESE SPOTS OVER A THREE-MONTH SPAN:

16.1%
Severe Headache or Migraine

One of the most common ailments of the nervous system.

15.1%
Neck Pain

Often mistaken initially for shoulder pain.

9.0%
Shoulder Pain

7.6%
Finger Pain

Can be caused by injuries, repeated motion and osteoarthritis.

One in four people is estimated to develop symptoms by age 85.

7.1%
Hip Pain

19.5%
Knee Pain

One in two Americans may develop knee pain from osteoarthritis.

28.1%
Lower-Back Pain

Sources: Centers for Disease Control and Prevention; World Health Organization

Getty Images (5); Alamy (back); Corbis (hip)

Wall Street Journal
October 7, 2013

PubMed

Display Settings: Abstract

Full text links

Mary Ann Liebert,

Photomed Laser Surg. 2014 Dec;32(12):669-77. doi: 10.1089/pho.2014.3821.

The fluence effects of low-level laser therapy on inflammation, fibroblast-like synoviocytes, and synovial apoptosis in rats with adjuvant-induced arthritis.

Hsieh YL[1], Cheng YJ, Huang FC, Yang CC.

Author information

Abstract

Abstract Objective: The aim of this study was to evaluate the effect of low-level laser therapy (LLLT) operating at low and high fluences on joint inflammation, fibroblast-like synoviocytes (FLS), and synovial apoptosis in rats with adjuvant-induced arthritis.

BACKGROUND DATA: Rheumatoid arthritis (RA) is characterized by pronounced inflammation and FLS proliferation within affected joints. Certain data indicate that LLLT is effective in patients with inflammation caused by RA; however, the fluence effects of LLLT on synovium are unclear.

METHODS: Monoarthritis was induced in adult male Sprague-Dawley rats (250-300 g) via intraarticular injection of complete Freund's adjuvant (CFA) into the tibiotarsal joint. Animals were irradiated 72 h after CFA administration with a 780 nm GaAlAs laser at 4.5 J/cm(2) (30 mW, 30 sec/spot) and 72 J/cm(2) (80 mW, 180 sec/spot) daily for 10 days. After LLLT, the animals were euthanized and their arthritic ankles were collected for histopathological analysis, immunoassays of tumor necrosis factor (TNF)-α, matrix metallopeptidase (MMP)3 and 5B5, and terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL) assays.

RESULTS: LLLT at a fluence of 4.5 J/cm(2) significantly reduced infiltration of inflammatory cells and expressions of TNF-α-, MMP3- and 5B5-like immunoreactivities, as well as resulting in more TUNEL-positive apoptotic cells in the synovium. No significant changes were observed in these biochemicals and inflammation in arthritic animals treated with 72 J/cm(2).

CONCLUSIONS: LLLT with low fluence is highly effective in reducing inflammation to sites of injury by decreasing the numbers of FLS, inflammatory cells, and mediators in the CFA-induced arthritic model. These data will be of value in designing clinical trials of LLLT for RA.

PMID: 25394331 [PubMed - in process] PMCID: PMC4267419 [Available on 2015/12/1]

Conclusions

Our results provide evidence of laser fluence-dependent reductions in TNF-a and MMP3, and of the ability of LLLT to inhibit the proliferation of inflammatory FLS.

This makes LLLT with LF a suitable treatment for synovitis that is associated with the early stages of inflammation in RA.

Our results also indicate a better understanding of the role of laser fluence in modulating these mediators that could be a basis for future therapeutic interventions.

We conclude that a single application of LLLT with a fluence of 4.5 J/cm2 is more efficient in modulating inflammatory mediators and inflammatory cells, and its effects can be observed by histological signs of attenuation of the inflammatory processes.

In addition, there are significant improvements in reduction of inflammation and FLS proliferation found in animals treated with 4.5 J/cm2, but not with 72 J/cm2.

The New Laser Therapy Handbook

A guide for research scientists, doctors, dentists, veterinarians and other interested parties within the medical field.

By

Jan Tunér and Lars Hode

615 pages + references

2126 references

Prima Books AB

2010

The New Laser Therapy Handbook

Jan Tuner and Lars Hode

Prima Books, 2010

"The effect of laser photon therapy [LPT] on inflammation is covered in many chapters of this book. The anti-inflammatory effect of LPT has been widely studied. LPT seems to have a similar effect to steroids and NSAIDs, but without the severe side effects of these very common pharmaceuticals." p. 248

Wheat Belly Total Health
The Ultimate Grain-Free Health and Weight-Loss Life Plan

William Davis, MD

2014

"Most modern people are boiling pots of inflammation:

hot, steaming, churning cauldrons of disordered, chaotic inflammatory responses, much of them due to food choices that conform poorly to human dietary needs."

Wheat Belly Total Health
The Ultimate Grain-Free Health and Weight-Loss Life Plan

William Davis, MD

2014

"Fish oil is the only reliable and sufficiently potent source of EPA and DHA. Krill oil, while interesting for its astaxanthin content (a carotenoid similar to beta-carotene), provides only a trivial amount of EPA and DHA. Krill is often marketed as having a more highly absorbed phospholipid from of omega-3s, which is true, but it contains so little that you'd have to consume an entire bottle every day to yield sufficient quantity of EPA and DHA. I advocate an intake or 3,000 to 3,600 mg per day (the dose of combined omega-3 fatty acids. EPA and DHA, not fish oil)." "This quantity yields a level of omega-3 fatty acid in the bloodstream of 10% or more, meaning that 10% of all fatty acids in red blood cells are compromised of EPA and DHA, the level that provides maximal protection from cardiovascular disease and a variety of anti-inflammatory benefits."

The Initial Effects of a Cervical Spine Manipulative Physiotherapy Treatment on the Pain and Dysfunction of Lateral Epicondylalgia

Pain; November 1996; Vol. 68; No. 1; pp. 69-74.

Bill Vicenzino, David Collins, Anthony Wright; University of Queensland

"A significant treatment effect beyond placebo or control was demonstrated."

"This study has demonstrated a clear hypoalgesic effect of a manipulative therapy technique in the period immediately following its application in a group of patients with lateral epicondylalgia."

"The beneficial effects of treatment [cervical manipulation] may continue after its application."

The author's theoretical model to explain their results involved manipulative therapy activation of the hypoalgesic effects of the endogenous supraspinal pain inhibitory systems.

"The [manipulative] treatment technique used in this study provided a non-noxious sensory input at the cervical spine which resulted in a reduction of elbow pain that outlasted the duration of its application." This is thought to activate the descending pain inhibitory system [DPIS] as a major component of their pain-relieving effects."

The DPIS is activated by stimulation of the periaqueductal grey (PAG).

"These findings indicate that manipulative therapy may constitute an adequate physical stimulus for activating DPIS."

A common finding in other studies "was the predominance of hypomobility at the lower cervical motion segments. It is feasible that part or all of the impairment in this study were "projected from the hypomobile cervical spine motion segment(s), and that the improvements gained following application of the [manipulative] technique resulted from treating the source of the pain."

"Manipulative therapy [may] recruit the DPIS, through which it exerts a portion or all of its pain relieving effects. That is, manipulative therapy applied to the cervical spine produces a sensory input which could be sufficient to activate DPIS."

"In a group of patients with LE, a manipulative therapy treatment technique applied to the lower cervical spine produced hypoalgesia at the elbow as manifest by increased pressure pain threshold, increased grip strength, improved neurodynamics and reduced pain over a 24 h period. This finding substantiates clinical observations that manipulative therapy is capable of producing improvements in pain and function immediately following application."

The Role of the Descending Inhibitory Pain Mechanism in Musculoskeletal Pain Following High-Velocity, Low Amplitude Thrust Manipulation
A Review of the Literature

Journal of Back and Musculoskeletal Rehabilitation
2014; Vol. 27; No. 4; pp. 377–382

Christos Savva, Giannis Giakas, Michalis Efstathiou

"The purpose of this review is to explore the role of the DIPM (descending inhibitory pain mechanism) in musculoskeletal pain following HVLAM (high-velocity, low amplitude thrust manipulation) as well as to identify the pain-relieving importance of this technique within clinical practice."

KEY POINTS FROM THIS ARTICLE:

1) "Musculoskeletal pain is one of the most common complaints for which patients attend hospitals."

2) "Musculoskeletal disorders often lead to chronic disability and increases the expenses of public health."

3) "Patients who suffer from musculoskeletal pain often report difficulties and limitations to perform their daily activities and job tasks and often present impairments in their physical performance, loss of function and reduced quality of life."

4) "Based on the kinetic chain principles that the upper and lower limb along with the spine is a kinetic chain of linked segments working together to perform daily movement and activity, the development of painful musculoskeletal disorders in these regions provoke muscle imbalances and alters the patient's normal movement patterns."

5) "HVLAM is an alternative treatment method and it is used as an analgesic modality for the rehabilitation of musculoskeletal dysfunctions including low back pain, neck pain, chronic ankle sprain, cervicogenic headache and dizziness etc."

6) HVLAM "has been used by physiotherapists, osteopaths and chiropractors for more than 2000 years and it is recommended by the majority of international clinical guidelines due to its immediate analgesic effect on musculoskeletal pain."

7) "The anti-nociceptive effect of high-velocity, low amplitude thrust manipulation (HVLAM) has been recognized by numerous systematic reviews."

8) "Many studies have investigated the mechanism of hypoalgesia induced by the application of manipulation in humans and animals suggesting that, the excitation of the descending inhibitory pain mechanism (DIPM) might play the most important role for musculoskeletal pain relief."

9) An increasing number of studies have suggested that HVLAM may excite the descending inhibitory pain mechanism (DIPM), which "might play the most important role for musculoskeletal pain relief."

10) "Findings from current literature support that HVLAM has a profound influence on nociceptive stimulus via the possible activation of the DIPM."

11) HVLAM "activates the periaqueductal gray region area of the midbrain, stimulates the noradrenergic descending system and at the level of the spinal cord, the nociceptive afferent barrage is reduced and mechanical hypoalgesia is induced."

12) "The clinical importance of the activation of the DIPM should not be ignored since the resulted analgesic effect of this technique can provide a window of opportunity to restore impaired physical performance and disability."

13) "Noxious stimuli generated by pathology of the musculoskeletal system are initially transferred to the dorsal horn of the spinal cord and then to the pain center located in the cerebral cortex of the brain." **[1]**

14) "The PAG [periaqueductal gray] has been found to be an important component of the central nervous system with regard to post-manipulation hypoalgesia." The DIPM projected from the periaqueductal gray region (PAG) of the midbrain to the dorsal horn of the spinal cord, has a profound role in regulating pain related signals at the spinal cord level. **[2]**

15) Activation of the DIPM inhibits the nociceptive afferent barrage at the level of the spinal cord and produces immediate analgesic effect on musculoskeletal pain.

16) From the PAG to the spinal cord, two different descending systems exist:

- The noradrenergic control system utilizes noradrenaline [norepinephrine] to inhibit pain. When this system is activated, it causes a *temporary excitation* of the sympathetic nervous system.

- The serotonergic control system which uses the serotonin to increase the thermal nociceptive threshold. When this system is activated, it produces sympathetic *inhibition*.

17) HVLAM provokes an immediate activation of the PAG **[2]**, excites the noradrenergic [norepinephrine] descending system and produces pain inhibition along with a *period* of sympathetic excitation. "The mechanical hypoalgesic effect occurs within minutes of manipulation and is associated with an increase in pressure pain threshold." "HVLAM does not modulate sensitivity to thermal pain suggesting that the serotonergic control system may not be stimulated."

18) "Both mobilization and manipulation techniques induce hypoalgesia via the activation of DIPM." Both techniques produce hypoalgesia through the exact same mechanism.

19) The analgesic effect of mobilization and manipulation occurs within minutes as a consequence of sympathetic excitation, but the "evaluation of these techniques in the next few hours could reveal thermal hypoalgesia and sympathetic inhibition." **[Important]**

20) "HVLAM used as an analgesic modality due to the possible activation of the DIPM can provide a window of opportunity to manage patients' symptoms and retrain the impaired motor function."

21) "When using HVLAM to produce pain inhibition, the patient is able to perform pain-free movements which were restricted due to pain."

22) To inhibit pain, HVLAM can be applied on the injured joint or on a joint proximal to the affected joint. **[Adjusting the precise painful joint is not necessary because adjacent joints are also capable of initiating the DIPM].**

23) HVLAM, through its immediate analgesic effect on musculoskeletal pain, can enable physiotherapists, osteopaths and chiropractors to "improve the limited joint mobility and restore the muscle imbalance around the area of symptoms."

24) "Activation of the DIPM might play the most important role with regard to post-manipulation hypoalgesia." **[Key Point]**

25) "HVLAM combined with conventional treatment such as strengthening, stretching and functional exercises can therefore contribute to improve range of motion, increase joint function and integrity and treat the altered proprioceptive input and movement patterns in order to restore impaired physical performance and disability."

COMMENTS FROM DAN MURPHY [See Graphic Below]

The key message from this study is that the pain reduction noted immediately after being adjusted *is not* primarily attributed to biochemistry/neurology changes at the adjusted joint, but rather are primarily attributed to activating the supra-segmental descending pain inhibitory control system, which involves the periaqueductal gray matter of the mesencephalon. This is very important because it is established that the periaqueductal gray is itself influenced by the hypothalamus. This adds to the evidence for explaining how spinal adjusting can influence a number of aspects of systemic physiology, as the hypothalamus controls whole body homeostasis, the pituitary gland, and the autonomic nervous system.

Whiplash, Real or Not Real? A Review and New Concept

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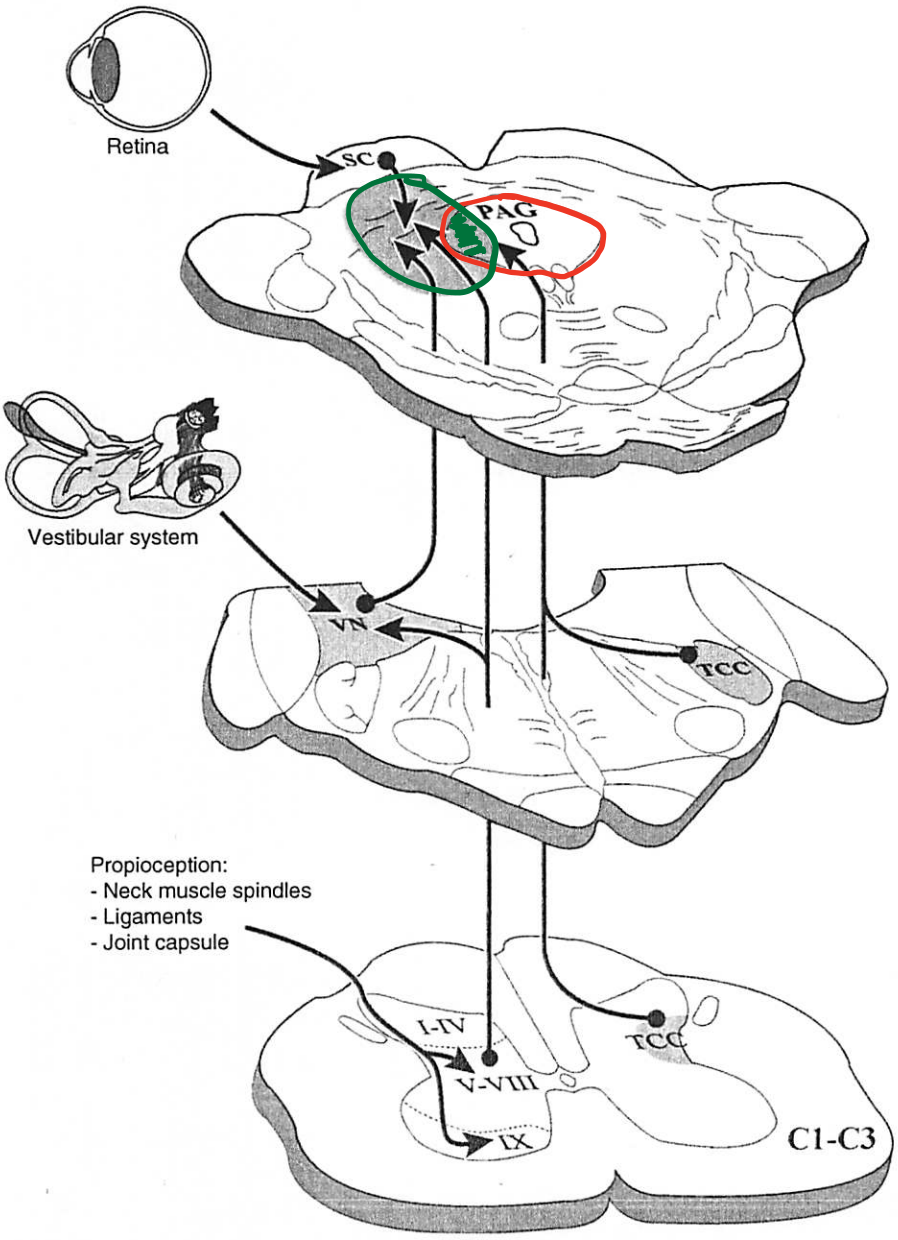

Fig. 46.5 Ascending afferents from the upper cervical segments (C1–C3) to the periaqueductal gray and its adjoining regions. *TCC* trigeminocervical complex, *VN* vestibular nuclei, *PAG* periaqueductal gray, *SC* superior colliculus

The involvement of the PAG and adjoining areas in the pathophysiology of WAD can explain the alterations in pain perception, but also depressive-like symptoms (Northoff et al. 2011). The relation of the trigeminocervical complex and the PAG can explain the headaches and temporomandibular pain symptoms in many WAD


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