**Opinion Article** 

## Short Note on Lipid Autacoid Medicine

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## ABOUT THE STUDY

Autacoid medicine is based on formulations that contain autacoids or autacoid derivatives, where autacoids are 'local tissue hormones' or modifying factors that direct the activity of adjacent cells and/or tissues and are metabolized by the same tissues. Autacoids include neurotransmitters like NO and ATP, as well as a variety of endocannabinoids including 2-Arachidonoylglycerol (2-AG). The Greek words autos, which means "self," and akos, which means "medicine," combine to form the term autacoid. As a result, autacoids are "self-healing" molecules.

Autacoids have been identified as mediators of a range of immunological activities since 1981, and the concentration of autacoids in tissues during inflammation is sufficient to attract a large number of immune cells to the site of inflammation, thus controlling both cell-mediated and humoral immunity. The majority of these autacoids have an important role in chronic pain. It has long been recognized that these "self-medications" can be used as a substitute for a variety of medicines.

Autacoid medicine is based on the body's natural defense and healing processes. Autacoid treatment has a variety of benefits over NCE-based therapies, the most notable of which being the lack of harmful metabolites associated with these molecules due to their endogenous nature. As a result, long-term safety concerns have been ruled out.

Lipid autacoids are the most researched of all the autacoids. In the case of hyperactive inflammation, these autacoids and their derivatives help to restore a healthy balance and aid in the resolution of the inflammation and subsequent wound healing. One of the most important elements of the lipid autacoid's function in neuropathic pain is this property. From small molecular weight signaling lipids to high molecular weight glycerophospholipids, as well as structural isomers, the lipid autacoid family has a diverse spectrum of compounds. As a result, identifying and profiling these chemicals is exceedingly difficult.

In 1988, Alessandro Bruni of the University of Padova's Department of Pharmacology was one of the first to discover that lipid autacoids were produced in the plasma. One of the

most important members of the lipid autacoid family is palmitoylethanolamide. Rita Levi-Montalcini, an Italian Nobel Laureate, discovered it in the 1990s. She was the first to point out that autacoids had repair function.

In the 1990s, a tiny Italian business called Life Group Spa submitted the first patents on lipid autacoids. Levi-research Montalcini's on PEA and its derivatives in inflammatory models may be found in these patents. PEA is the most studied lipid autacoid in both preclinical and clinical research, with over 650 PubMed indexed publications. PEA's anti-inflammatory properties have been shown to be effective in the treatment of a wide range of diseases, including Alzheimer's disease, arthritis, asthma, depression, glaucoma, multiple sclerosis, reperfusion damage, sepsis, and sciatic and neuropathic pain. PEA has retinoprotective effects, according to its neuroprotective profile.

PEA was initially approved for the treatment of respiratory viral infections in Spain in the 1980s (brand name Palmidrol). PEA was then developed into novel neutraceutical formulations, and it became more widely available. It's worth noting that, despite the fact that PEA is now classed as a supplement in Europe, its therapeutic benefits have been well-documented in a number of placebo-controlled, randomized clinical trials. In addition, there is enough clinical evidence to suggest that taking PEA as a supplement is safe.

PEA has also been shown to have analgesic and antiinflammatory properties. PEA dosages of up to 100 mg/kg body weight have been given to individuals with no obvious side effects. Currently, there is a lot of clinical information about PEA. PEA treatment appears to be safe and helpful in a variety of neuropathic pain disorders, including diabetic neuropathy and chronic idiopathic axonal neuropathy. A number of placebocontrolled clinical trials show that PEA is helpful and safe in nerve entrapment neuropathy disorders such carpal tunnel syndrome and sciatic pain. The NNT (Number required to treat) in a randomized controlled study including approximately 600 sciatic pain patients was fewer than 2. As a result, pain doctors should think carefully about using PEA in people.

There are a variety of PEA formulations on the market, but only those containing micro PEA have been proven to be safe and

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effective. Only three of these formulations, optimized PEA (PEA-opt), micronized PEA (PEA-m), and ultra-micronized PEA, are found as nutraceutical supplements (PEA-um).

We've been using PEA at our clinic for the past eight years, and we've helped a lot of people with axonal neuropathies including Chronic Idiopathic Axonal Neuropathy (CIAP), diabetic neuropathy, and sciatica. In neuropathic pain, a daily dose of 1200 mg for at least two months can be safely suggested, according to our research. PEA appears to reset the system during the intermittent time. Our findings also show that partial responders may be converted into complete responders by increasing the dose.

As a result, lipid autacoids provide a ray of hope for pain treatment, especially because their side effects are minor and they are metabolized via endogenous metabolic pathways; their main and secondary metabolites are well-known. Serious research and development in this sector might lead to pain medications that are both safer and more effective. Now that a number of patents on autacoids have expired, new opportunities for generic autacoids formulations to reach the market have opened up.