REVIEW ARTICLE

Role of Resolvins and Protectins in the Treatment of the Periodontal Diseases

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ABSTRACT

Resolvins and protectins are resolution of inflammation, which have historically been viewed as a passive process, occurring as a result of the withdrawal of pro-inflammatory signals, including lipid mediators such as leukotrienes and prostaglandins.

Keywords: Inflammation, Resolution, Resolvins, Protections

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INTRODUCTION

The term "resolvins" or "resolution-phase interaction products" was coined by Professor Charles Serhan and colleagues because these compounds were first encountered in resolving inflammatory exudates. These novel mediators were endogenous agonists controlling inflammation through stimulation of resolution. These novel endogenous anti-inflammatory and pro-resolving mediators also provide the biotemplates to produce stable analogs and mimetics. Together with the lipoxins, they constitute a pharmacologic genus of pro-resolving and anti-inflammatory molecules[1] [Figures 1 and 2].

Resolvins and their Therapeutic Value (in Periodontitis)

The basic structure of this potent bioactive product generated from eicosapentaenoic acid (EPA) proved to be 5, 12,18R-trihydroxyeicosapentaenoic acid. RvE1 possesses an interesting and novel distinct structure consisting of a conjugated diene plus a conjugated diene chromophore present within the same molecule. Resolvins offer an entirely novel biological approach to treating significant inflammatory diseases, with a decreased potential for immunosuppression. Resolvins are potential candidates for drugs to treat a broad range of acute and chronic diseases caused by a failure to resolve the inflammatory response and restore immune homeostasis. Such diseases include autoimmune diseases (Crohn's disease, psoriasis, and rheumatoid arthritis), allergic diseases (asthma), and chronic inflammatory diseases. Resolvins offer an entirely novel biological approach to treat significant inflammatory diseases, with a decreased potential for immunosuppression.[2]

Topical application of 4 mg resolvin E1 per tooth every other day for 6 weeks in cases of periodontitis was shown to reduce PMN infiltration and stop inflammation-induced tissue and bone loss. These mediate resolution and counterregulate excessive acute inflammation. These are biosynthesized from precursors such as omega-3 polyunsaturated fatty acids, EPA, and docosahexaenoic acid (DHA) through sequential steps involving lipoxygenases and cyclooxygenases. The role of these endogenous chemical mediators is similar to that of lipoxins, i.e., inhibition of neutrophil recruitment, etc. These stereoselective players counterregulate excessive acute inflammation and stimulate molecular and cellular events that define resolution.[3]

PROTECTINS (NEUROPROTECTINS)

In studies of resolvin formation in brain tissue in response to aspirin treatment, it was shown that new docosatrienes termed initially "neuroprotectins" were produced. Like the leukotrienes, there are three double bonds in conjugation, hence the term "triene," though there are six double bonds in total. As it is now recognized that the formation and actions of these docosanoids are not restricted to neuronal tissue, it has been suggested that the simpler term "protectins" is preferable. PD1 is present in murine inflammatory exudates and lung, in peripheral human blood and exhaled breath condensates and in a wide range of cell types. The lipoxygenase product 17S-hydroperoxy-DHA is converted first to a 16-epoxide and then to the

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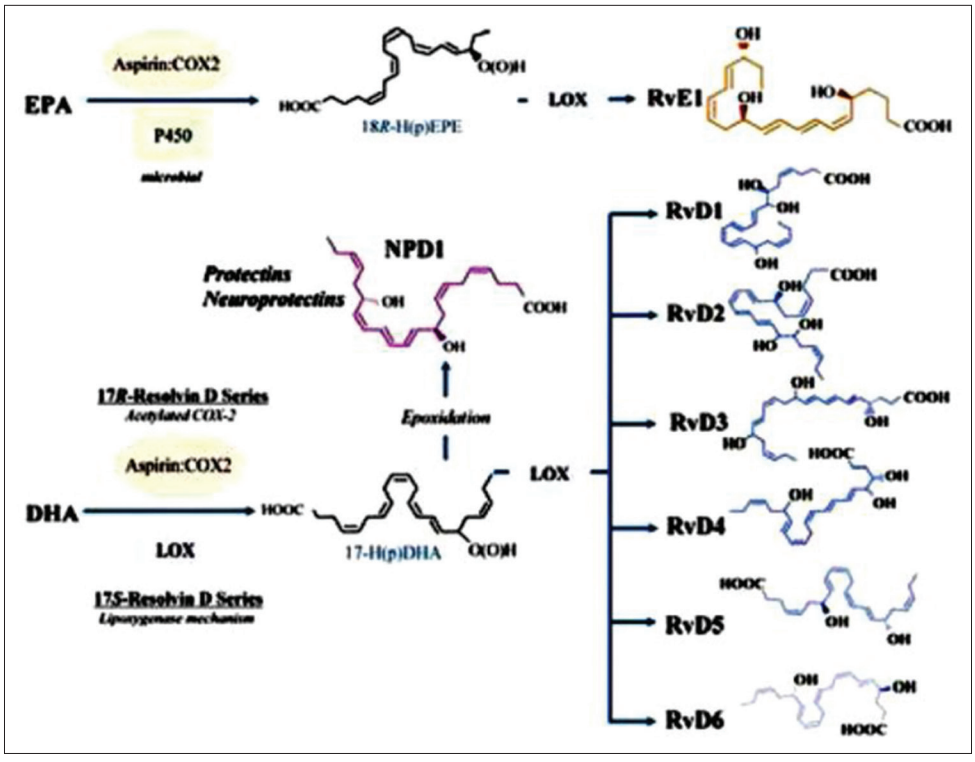

Figure 1: Families of lipid autacoids

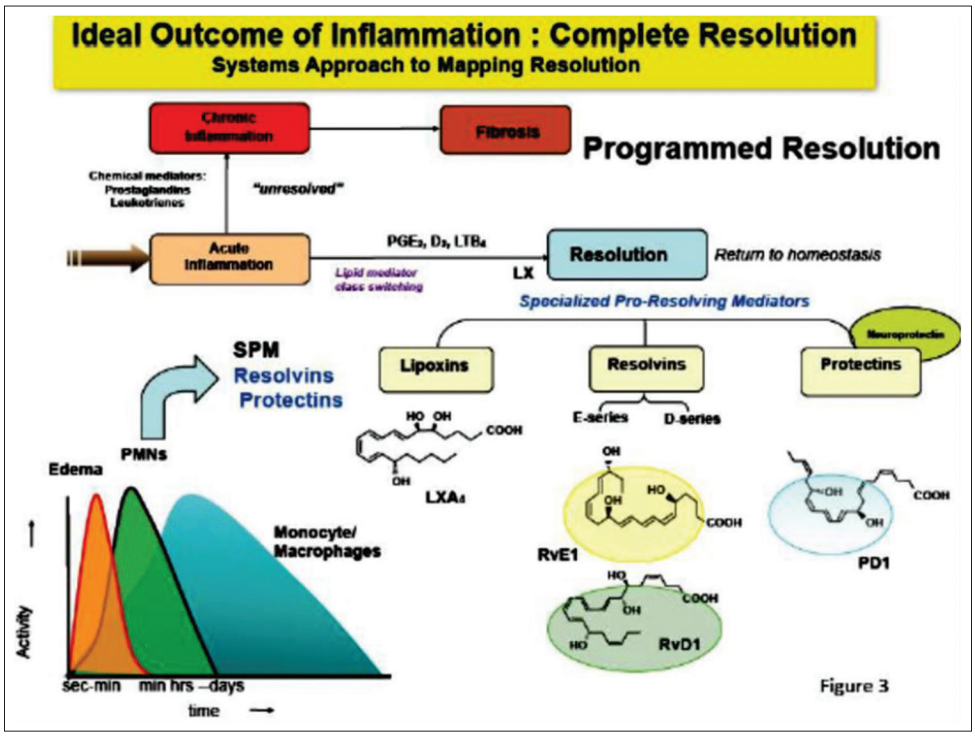

Figure 2: Specialized pro-resolving mediators are generated through inflammatory resolution

10,17-dihydroxydocosatriene (10R,17S-dihydroxy–docosa-4Z, 7Z, 11E, 13E, 15Z, 19Z-hexaenoic acid) denoted as 10R, 17S-DT or PD1 (or NPD1). Synthesis of NPD1 is induced as a response to oxidative stress and/or activation of neurotrophils, and again, it appears that this highly stereospecific structure is essential for biological activity. Further, oxygenation can occur and 17S-hydroperoxy-DHA can be oxidized at the terminal carbon atom by cytochrome P450 enzymes or it can react with 5-lipoxygenase to form two regioisomeric dihydroxyp[3] [Figure 3].

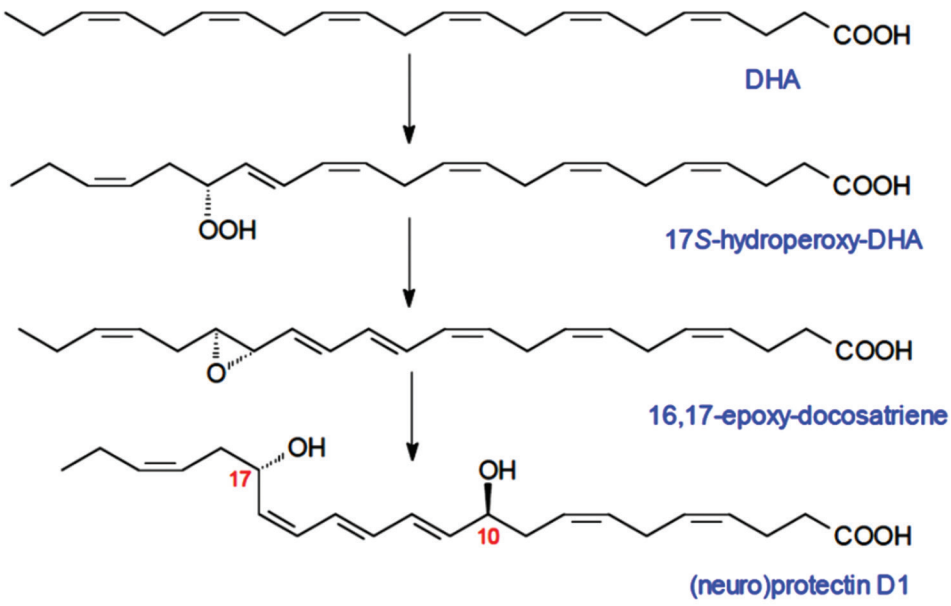

Figure 3: Biosynthetic pathway to protectins

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