Journal of Autacoids

Open access Commentary

Role of Catabolism of Platelet Activating Factor as a Potent Inflammatory Mediator

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DESCRIPTION

Platelet Activating Factor (PAF) includes a recent clinical trial of the drug darapladib, which targets plasma platelet activating factor acetylhydrolase. We recently demonstrated that a previously unknown acyl analogue of PAF, co-produced during PAF biosynthesis, acts as a sacrificial substrate for platelet activating factor acetylhydrolase and possibly as an endogenous PAF receptor antagonist/partial agonist function. Provided experimental evidence that it suppresses PAF signaling by If this is the case, platelet activating factor acetylhydrolase should catalyze the selective hydrolysis of alkyl pafs, but not acyl pafs. The interactions between acyl-PAF, alkyl-PAF, platelet activating factor acetylhydrolase and PAF-receptor are complex, but the consequences of this interaction have not been evaluated. This review describes this interaction based on recent findings. It is highly likely that the relative abundance of acyl and alkyl pafs and their interaction with PAF-R in the presence of the hydrolase platelet activating factor acetylhydrolase have regulatory effects on PAF signaling during inflammation.

Platelet-activating factor is a potent inflammatory mediator that exerts its actions through a single PAF receptor (PAF-R). Both PAF species are degraded by plasma-type platelet activating factor acetylhydrolase. Alkyl-PAF causes sudden death in Swiss albino mice, and this effect can also be suppressed by simultaneous bolus administration of acyl-Platelet-activating factor. The protective effect of acyl-pafs against alkyl-Platelet-activating factor induced death decreased successively with stepwise increases in platelet activating factor acetylhydrolase levels. Acyl-Platelet-activating factor alone is mildly proinflammatory, but in pathophysiological settings, abundant acyl-PAF suppresses the effects of alkyl-PAF.

Recent studies have confirmed the existence of 1-acyl-2-acetyl-sn-glycero-3-phosphocholine. An acyl analogue of platelet-activating factor, exhibited in unstimulated tissues and its formation with platelet-activating factor upon stimulation of a variety of cells. We show herein that this acyl analogue of Platelet-activating factor can be catabolized by purified bovine liver lysophospholipases I and II to form 2-acetyl-sn-glycero-3-phosphocholine in near stoichiometric proportions. This suggests that some lysophospholipases may contribute to the intracellular inactivation of her Platelet-activating factor through deacetylation. However, similar experiments with rat kidney cytosol, rat and human platelet cytosol clearly defined lysophospholipase activity without Platelet-activating factor acetylhydrolase activity and specific Platelet-activating factor acetylhydrolases without lysophospholipase activity. Thus, lysophospholipases are clearly involved in the metabolism of acyl analogues of PAF and may also contribute to the abolition of Platelet-activating factor's bioactivity through deacetylation in some tissues such as the liver.

We used platelet-activating factor and its synthetic analogues to investigate the substrate specificity of Platelet-activating factor degrading enzymes from different sources. The results were: Tissue-derived acetyl hydrolases, such as the rat kidney soluble enzyme, catalyze 1S-methyl-1-O-hexadecyl-2-acetyl-sn-glycero-3-phosphocholine (1S-Me-PAF) slightly faster than PAF. Deacetylate. On the other hand, plasma-acetylhydrolase hydrolyzed His-Platelet-activating factor more efficiently than 1S-Me-PAF. Homogenates of polymorphonuclear leukocytes, monocytes and lymphocytes from rats showed considerable acetylhydrolase activity and their substrate specificity was similar to that of plasma enzymes. Pleural effusions from experimental pleurisy induced by carrageenan in rats contained acetylhydrolase activity with properties similar to plasma enzymes.

ACKNOWLEDGEMENT

None.

CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

Received:	31-May-2023	**Manuscript No:**	JAC-23-17066
Editor assigned:	02-June-2023	**PreQC No:**	JAC-23-17066 (PQ)
Reviewed:	16-June-2023	**QC No:**	JAC-23-17066
Revised:	21-June-2023	**Manuscript No:**	JAC-23-17066 (R)
Published:	28-June-2023	**DOI:**	10.35841/jac.4.2.13

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Citation Popov A (2023) Role of Catabolism of Platelet Activating Factor as a Potent Inflammatory Mediator. Autacoids J. 4:13.