

PAF-acetylhydrolase and PAF-receptor are expressed in Adipocytes and Preadipocytes. TNF α and Leptin Effect on this Expression

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There is growing evidence suggesting that adipose tissue has an essential role in the inflammatory response and that obesity is a low grade inflammatory disease. Our research group has shown that adipocytes have the ability to synthesize PAF under stimulation. PAF is a well known proinflammatory mediator, which is synthesized in many cell types. Its effects on target cells are mediated by specific receptors (PAFR) which belong to 7TM family. The enzyme PAF-acetylhydrolase (PAF-AH) which hydrolyzes PAF to its inactive metabolite lyso-PAF controls PAF levels. We have also isolated and characterized PAF-AH from rat adipocytes.

According to our yet unpublished data, TNF α , the proinflammatory cytokine which is involved in obesity and NIDDM, stimulates PAF synthesis in rat preadipocytes and adipocytes. TNF α effect in adipocytes is 14-fold higher than in preadipocytes. We believe that these results contribute to the hypothesis that obesity is an inflammatory disease, as we showed that the differentiation of adipocytes, a process which is highly active during the development of obesity, results in the enhancement of the TNF α effect on the synthesis of a potent mediator, possibly in order to use it for exerting its inflammatory actions.

In the present study we show that PAF-AH and PAFR mRNA is expressed in adipocytes and in preadipocytes. We also investigate the effect of TNF α and leptin on PAF-AH and PAF-R expression in both cell types. Our interest for leptin-PAF interrelationship is originated from recent references which suggest that leptin, the main hormone of adipose tissue, is also involved in inflammation. Thus, rat preadipocyte and adipocyte cultures were incubated in the presence or absence of TNF α or leptin. Total RNA was extracted and reverse transcribed for first strand cDNA synthesis. PCR reaction was performed using specific primer pairs for PAF-AH and PAFR. Our results showed that TNF α and leptin induce PAF-AH and PAF-R mRNA expression in both cell types. These effects of leptin support its inflammatory role.

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