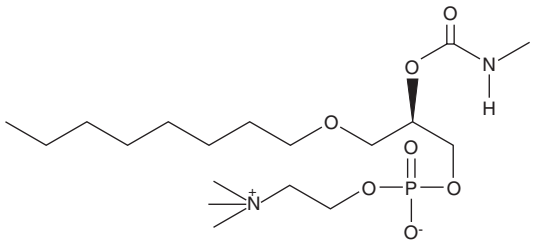
PRODUCT INFORMATION

Methylcarbamyl PAF C-8

Catalog No. 9000332

Formal Name:	1-O-octyl-2-O-(N-methylcarbamoyl)-*sn*-glyceryl-3-phosphorylcholine
MF:	$C_{18}H_{39}N_2O_7P$
FW:	426.5
Purity:	≥98%
Supplied as:	A solution in ethanol
Storage:	-20°C
Stability:	≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Methylcarbamyl PAF C-8 is supplied as a solution in ethanol. To change the solvent, simply evaporate the ethanol under a gentle stream of nitrogen and immediately add the solvent of choice. Solvents such as ethanol, DMSO, and dimethyl formamide purged with an inert gas can be used. The solubility of methylcarbamyl PAF C-8 in these solvents is approximately 25 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. If an organic solvent-free solution of methylcarbamyl PAF C-8 is needed, it can be prepared by evaporating the ethanol and directly dissolving the neat oil in aqueous buffers. The solubility of methylcarbamyl PAF C-8 in PBS, pH 7.2, is approximately 25 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Methylcarbamyl PAF C-8 is the C-8 analog of methylcarbamyl PAF C-16. Methylcarbamyl PAF C-16 is a stable analog of PAF C-16 with a half-life greater than 100 minutes in platelet poor plasma due to its resistance to degradation by PAF-AH.[1,2] It is nearly equipotent with PAF C-16 in its ability to induce platelet aggregation both in isolated platelets and in platelet-rich plasma.[1] In NRK-49 cells overexpressing the PAF receptor, both PAF C-16 and methylcarbamyl PAF C-16 cause the induction of c-Myc, c-fos, and the activation of mitogen-activated protein kinase.[2] Methylcarbamyl PAF C-16 induces G_1 phase cell cycle arrest, suggesting a potential role for PAF in the inhibition of oncogenic transformation.[2]

References

1. Hadváry, P., Cassal, J.-M., Hirth, G., *et al.* Structural requirements for the activation of blood platelets by analogues of platelet-activating factor (PAF-acether). *Platelet-Activating Factor INSERM Symposium* **23**, 57-64 (1983).
2. Kume, K. and Shimizu, T. Platelet-activating factor (PAF) induces growth stimulation, inhibition, and suppression of oncogenic transformation in NRK cells overexpressing the PAF receptor. *J. Biol. Chem.* **272**, 22898-22904 (1997).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM