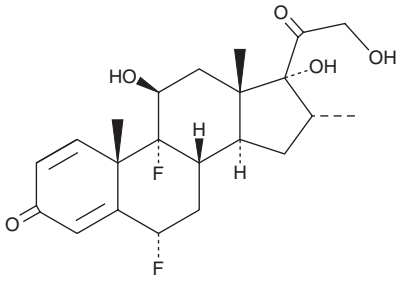
PRODUCT INFORMATION

Flumethasone

Item No. 24184

CAS Registry No.:	2135-17-3
Formal Name:	6α,9-difluoro-11β,17,21-trihydroxy-16α-methyl-pregna-1,4-diene-3,20-dione
Synonyms:	NSC 5402, NSC 54702, U-10974
MF:	$C_{22}H_{28}F_2O_5$
FW:	410.5
Purity:	≥98%
UV/Vis.:	λ_{max}: 239 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



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

Laboratory Procedures

Flumethasone is supplied as a crystalline solid. A stock solution may be made by dissolving the flumethasone in the solvent of choice, which should be purged with an inert gas. Flumethasone is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of flumethasone in these solvents is approximately 3, 30, and 25 mg/ml, respectively.

Flumethasone is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, flumethasone should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Flumethasone has a solubility of approximately 0.1 mg/ml in a 1:10 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Flumethasone is an agonist of glucocorticoid and mineralocorticoid receptors with EC_{50} values of 0.26 and 0.494 nM, respectively, in CV-1 cells expressing human receptors.[1] *In vitro*, it inhibits the growth of UM-UC-3, TCC-SUP, and 5637 urothelial carcinoma cell lines at a concentration of 100 nM.[2] Flumethasone (5 mg per animal) decreases tumor necrosis factor (TNF) production *ex vivo* in blood cells collected by bronchoalveolar lavage (BAL) from calves with experimentally-induced local lung inflammation.[3] It also inhibits phytohemagglutinin-induced delayed hypersensitivity in calf skin. *In vivo*, flumethasone (5 µM) impairs cell cycle re-entry of cardiac cells seven days post cryo-injury to the heart and impairs cardiac regeneration in zebrafish.[4]

References

1. Grossmann, C., Scholz, T., Rochel, M., *et al.* Transactivation *via* the human glucocorticoid and mineralocorticoid receptor by therapeutically used steroids in CV-1 cells: A comparison of their glucocorticoid and mineralocorticoid properties. *Eur. J. Endocrinol.* **151(3)**, 397-406 (2004).
2. Ishiguro, H., Kawahara, T., Zheng, Y., *et al.* Differential regulation of bladder cancer growth by various glucocorticoids: Corticosterone and prednisone inhibit cell invasion without promoting cell proliferation or reducing cisplatin cytotoxicity. *Cancer Chemother. Pharmacol.* **74(2)**, 249-255 (2014).
3. Bednarek, D., Szuster-Ciesielska, A., Zdzisińska, B., *et al.* The effect of steroidal and non-steroidal anti-inflammatory drugs on the cellular immunity of calves with experimentally-induced local lung inflammation. *Vet. Immunol. Immunopathol.* **71(1)**, 1-15 (1999).
4. de Preux Charles, A.-S., Bise, T., Baier, F., *et al.* Distinct effects of inflammation on preconditioning and regeneration of the adult zebrafish heart. *Open Biol.* **6(7)**, (2016).

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