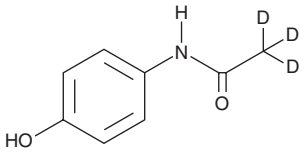
PRODUCT INFORMATION

Acetaminophen-d_3

Item No. 46351

CAS Registry No.:	60902-28-5
Formal Name:	N-(4-hydroxyphenyl)-acetamide-2,2,2-d_3
Synonyms:	4-Acetamidophenol-d_3, APAP-d_3, 4'-Hydroxyacetanilide-d_3, Paracetamol-d_3
MF:	$C_8H_6D_3NO_2$
FW:	154.2
Chemical Purity:	≥98% (Acetaminophen)
Deuterium Incorporation:	≥99% deuterated forms (d_1-d_3); ≤1% d_0
Supplied as:	A 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

Acetaminophen-d_3 is intended for use as an internal standard for the quantification of acetaminophen (Item No. 10024) by GC- or LC-MS The accuracy of the sample weight in this vial is between 5% over and 2% under the amount shown on the vial. If better precision is required, the deuterated standard should be quantitated against a more precisely weighed unlabeled standard by constructing a standard curve of peak intensity ratios (deuterated *versus* unlabeled).

Acetaminophen-d_3 is supplied as a solid. A stock solution may be made by dissolving the acetaminophen-d_3 in the solvent of choice, which should be purged with an inert gas. Acetaminophen-d_3 is soluble (≥10 mg/ml) in DMSO and sparingly soluble (1-10 mg/ml) in ethanol.

Description

Acetaminophen is an analgesic and antipyretic compound.[1,2] Unlike many NSAIDs, which inhibit both COX-1 and COX-2, early studies suggested that acetaminophen is a poor inhibitor of both isoforms.[3,4] However, it does inhibit COX-2 by 83% and COX-1 by 56% in human blood *ex vivo*, albeit at a high 1,000 mg dose, with IC_{50} values of 25.8 and 113.7 µM, respectively.[5] Acetaminophen is enzymatically and non-enzymatically converted to several reactive metabolites that contribute to adverse or indirect effects, including liver injury.[6-8] At toxic doses, the acetaminophen metabolite N-acetyl-4-benzoquinone imine (NAPQI; Item No. 16115) depletes glutathione reserves in the liver, leading to an accumulation of NAPQI and subsequent hepatocyte necrosis.[9] Acetaminophen decreases glutathione levels and reduces glutathione peroxidase activity in mice when administered at a dose of 250 mg/kg and induces ferroptotic cell death in primary mouse hepatocytes, an effect that can be blocked by the ferroptosis inhibitor ferrostatin-1 (Item No. 17729).[10,11] Acetaminophen has analgesic and antipyretic properties in animal models.[1,2]

References

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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

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4. Warner, T.D., Giuliano, F., Vojnovic, I., *et al.* Nonsteroid drug selectivities for cyclo-oxygenase-1 rather than cyclo-oxygenase-2 are associated with human gastrointestinal toxicity: A full *in vitro* analysis. *Proc. Natl. Acad. Sci. USA* **96(13),** 7563-7568 (1999).
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[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM