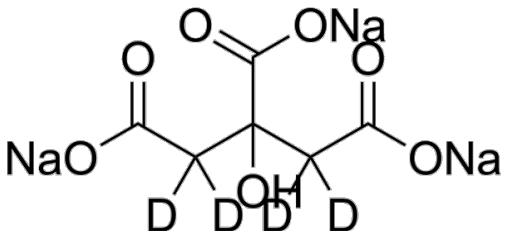
Product Data Sheet

Product Name: Citric acid-d 4 trisodium
Cat. No.: GC79842

Chemical Properties

Cas. No.	2704-20-3		
Formula	$C_6HD_4Na_3O_7$	M.Wt	262.09
Solubility		Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			



Background

Citric acid-d 4 trisodium (Sodium citrate-d 4) is the deuterium labeled Citric acid trisodium. Citric acid trisodium (Sodium citrate) is a natural preservative and food tartness enhancer. Citric acid trisodium induces apoptosis and cell cycle arrest at G2/M phase and S phase in HaCaT cells. Citric acid trisodium cause oxidative damage of the liver by means of the decrease of antioxidative enzyme activities. Citric acid trisodium is also an acidulant, emulsifier, sequestrant and buffering agent widely used across many industries [1] [2] [3] [4].

Caution: Product has not been fully validated for medical applications. For research use only.
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In Vitro, Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs.

References:

[1]. Ciriminna R, et al. Citric acid: emerging applications of key biotechnology industrial product. Chem Cent J. 2017 Mar 8;11:22.
[2]. Chen X, et al. Study on injury effect of food additive citric acid on liver tissue in mice. Cytotechnology. 2014 Mar;66(2):275-82.
[3]. Chen X, Lv Q, Liu Y, Deng W. Effects of the food additive, citric acid, on kidney cells of mice. Biotech Histochem. 2015 Jan;90(1):38-44.
[4]. Ying TH, et al. Citric acid induces cell-cycle arrest and apoptosis of human immortalized keratinocyte cell line (HaCaT) via caspase- and mitochondrial-dependent signaling pathways. Anticancer Res. 2013 Oct;33(10):4411-20.

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