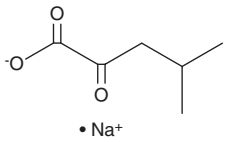
PRODUCT INFORMATION

α-Ketoisocaproic Acid (sodium salt)

Item No. 21052

CAS Registry No.: 4502-00-5
Formal Name: 4-methyl-2-oxo-pentanoic acid, monosodium salt
Synonyms: 2-Ketoisocaproate, α-Ketoisocaproate, 2-Ketoisocaproic Acid, KIC, 4-MOP, 2-Oxoisocaproic Acid, 4-methyl-2-Oxopentanoate, 4-methyl-2-Oxovalerate, 4-methyl-2-Oxovaleric Acid
MF: $C_6H_9O_3$ • Na
FW: 152.1
Purity: ≥95%
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

α-Ketoisocaproic acid (sodium salt) is supplied as a crystalline solid. A stock solution may be made by dissolving the α-ketoisocaproic acid (sodium salt) in the solvent of choice, which should be purged with an inert gas. α-Ketoisocaproic acid (sodium salt) is soluble in the organic solvent ethanol at a concentration of approximately 5 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. Organic solvent-free aqueous solutions of α-ketoisocaproic acid (sodium salt) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of α-ketoisocaproic acid (sodium salt) in PBS (pH 7.2) is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

α-Ketoisocaproic acid is a metabolite of L-leucine (Item No. 34342) catabolism.[1] It increases the production of reactive species and decreases the activity of mitochondrial complex I, also known as NADH dehydrogenase, and complex II-III in the rat hippocampus when administered at an intracerebroventricular dose of 4 µmol.[2] Urine levels of α-ketoisocaproic acid are reduced in *db/db* diabetic mice compared with heterozygous *db/m* non-diabetic mice.[3] α-Ketoisocaproic acid accumulates in the tissues and body fluids of patients with maple syrup urine disease, an inborn error of metabolism characterized by branched-chain α-keto acid dehydrogenase (BCKAD) deficiency and leads to progressive ketoacidosis, failure to thrive, neurological dysfunction, and, potentially, death.[2]

Reference

1. Schiff, M., Ogier de Baulny, H., and Dionisi-Vici, C. Branched-chain organic acidurias/acidaemias. *Inborn metabolic diseases: Diagnosis and treatment*. Saudubray, J.-M., Baumgartner, M.R., and Walter, J., editors, 6th edition, *Springer* (2016).
2. Farias, H.R., Gabriel, J.R., Cecconi, M.L., *et al.* The metabolic effect of α-ketoisocaproic acid: *In vivo* and *in vitro* studies. *Metab. Brain Dis.* **36(1)**, 185-192 (2021).
3. Kim, N.H., Hyeon, J.S., Kim, N.H., *et al.* Metabolic changes in urine and serum during progression of diabetic kidney disease in a mouse model. *Arch. Biochem. Biophys.* **646**, 90-97 (2018).

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