Uptake and metabolism of fructose by rat neocortical cells in vivo and by isolated nerve terminals in vitro

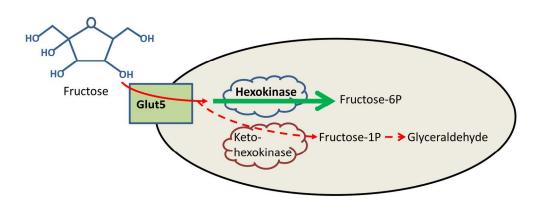
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Genes	Forward primer	Reverse primer
HPRT1	ggtccattcctatgactgtagatttt	caatcaagacgttctttccagtt
β ACTIN	ccaaccgtgaaaagatgacc	accagaggcatacagggaca
GAPDH	ggtgaaggtcggtgtgaac	ccttgactgtgccgttgaa
HK1	gaactgtcaccaaagtgtaccg	cgaagggtctcctctgagc
КНК	gtgtggatgtgtctcaagtgg	gacacatctggcaggttcg
GLUT5	tggagtggttcctcagctct	agtcagacccaggaggattg
GLUT3	tgacgatttctctgttactgaagg	agccacaataaaccagggaat
GLUT1	accctgcatctcattggtct	ggccacgatactcagataggac



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We asked how the brain handles fructose, which may react spontaneously with proteins to form 'advanced glycation end products' and trigger inflammation. Neocortical cells took up and metabolized extracellular fructose oxidatively in vivo, and isolated nerve terminals did so in vitro. The low expression of fructose transporter Glut5 limited uptake of extracellular fructose. Hexokinase was a main pathway for fructose metabolism, but ketohexokinase (which leads to glyceraldehyde formation) was expressed too. Neocortical cells also took up and metabolized glyceraldehyde oxidatively.

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