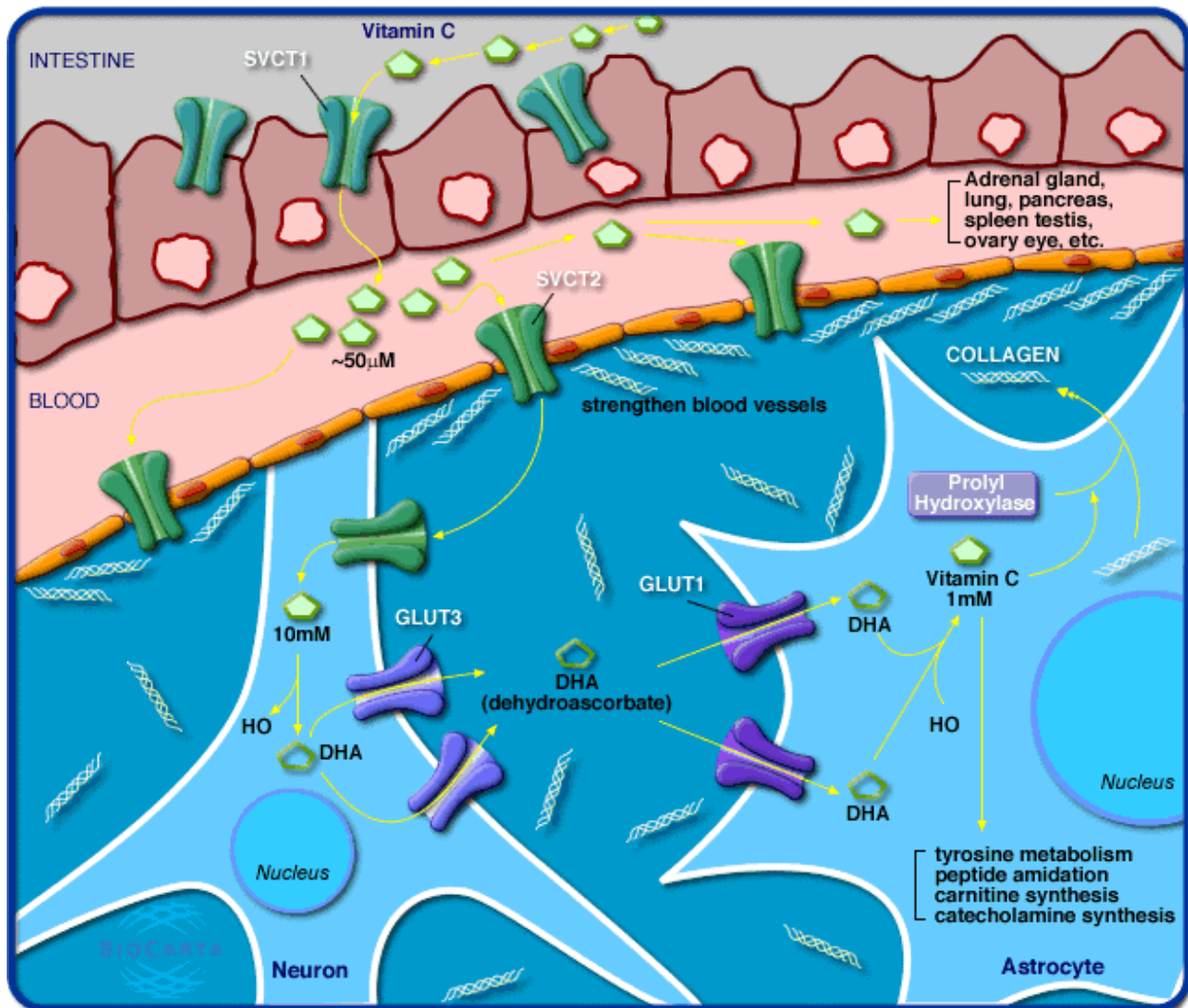


Vitamin C In The Brain



Vitamin C (ascorbic acid) was first identified by virtue of the essential role it plays in collagen modification, preventing the nutritional deficiency scurvy. Vitamin C acts as a cofactor for hydroxylase enzymes that post-translationally modify collagen to increase the strength and elasticity of tissues. Vitamin C reduces the metal ion prosthetic groups of many enzymes, maintaining activity of enzymes, also acts as an anti-oxidant. Although the prevention of scurvy through modification of collagen may be the most obvious role for vitamin C, it is not necessarily the only role of vitamin C. Svct1 and Svct2 are ascorbate transporters for vitamin C import into tissues and into cells. Both of these transporters specifically transport reduced L-ascorbic acid against a concentration gradient using the intracellular sodium gradient to drive ascorbate transport. Svct1 is expressed in epithelial cells in the intestine, upregulated in cellular models for intestinal epithelium and appears to be responsible for the import of dietary vitamin C from the intestinal lumen. The vitamin C imported from the intestine is present in

plasma at approximately 50 μM , almost exclusively in the reduced form, and is transported to tissues to play a variety of roles. Svct2 imports reduced ascorbate from the plasma into very active tissues like the brain. Deletion in mice of the gene for Svct2 revealed that ascorbate is required for normal development of the lungs and brain during pregnancy. A high concentration of vitamin C in neurons of the developing brain may help protect the developing brain from free radical damage. The oxidized form of ascorbate, dehydroascorbic acid, is transported into a variety of cells by the glucose transporter Glut-1. Glut-1, Glut-3 and Glut-4 can transport dehydroascorbate, but may not transport significant quantities of ascorbic acid *in vivo*.