**pathway -** Nicotinate and nicotinamide metabolism – GMPR2

**Gene involved:** NMRK2 **-** Nicotinamide riboside kinase 2.

**Function**

Catalyzes the phosphorylation of nicotinamide riboside (NR) and nicotinic acid riboside (NaR) to form nicotinamide mononucleotide (NMN) and nicotinic acid mononucleotide (NaMN). Reduces laminin matrix deposition and cell adhesion to laminin, but not to fibronectin. Involved in the regulation of PXN at the protein level and of PXN tyrosine phosphorylation. May play a role in the regulation of terminal myogenesis. <<https://www.uniprot.org/uniprot/Q9NPI5>>

**pathway - Mineral absorption –** GMPR2

**Genes involved:** CaBP9K ( S100G)

This gene is expressed in the intestine, placenta and uterus of the rat as a single 0.5kb long transcript. Exogenous 1,25(OH)2D3 triggers the rapid synthesis of CaBP9K mRNA and accumulation of translatable CaBP9K mRNA in the duodenum of vitamin D-deficient rats. Calcium also stimulates CaBP9K gene expression in this tissue. In contrast 1,25(OH)2D3 does not change the uterine concentration of CaBP9K but estrogen stimulates the transcription of the CaBP9K gene in the uterus. The promoter region of rat CaBP9K gene contains 1 TATA box and 4 CAAT box-type sequences and several steroid hormone regulatory elements. The CaBP9K gene is therefore a suitable model for studying the tissue-specific regulation of gene expression by steroid hormones. < <https://pubmed.ncbi.nlm.nih.gov/2291623/>>

**pathway - Vibrio cholerae infection - TM7SF3**

**Genes involved:** CFTR, ATP6V0A4, ATP6V0D2, ATP6V1B1 e ATP6V1G3

**CFTR** expression is downregulated in the UUO (unilateral ureteral obstruction)-induced kidney fibrosis mouse model and human fibrotic kidneys. Dysfunction or downregulation of CFTR in renal epithelial cells leads to alteration of genes involved in Epithelial-Mesenchymal Transition (EMT) and kidney fibrosis. In addition, dysregulation of CFTR activates canonical Wnt/β-catenin signaling pathways, whereas the β-catenin inhibitor reverses the effects of CFTR downregulation on EMT marker. More interestingly, CFTR interacts with Dishevelled 2 (Dvl2), a key component of Wnt signaling, thereby suppressing the activation of β-catenin.

< <https://pubmed.ncbi.nlm.nih.gov/28701694/>>

**pathway - Synaptic vesicle cycle - TM7SF3**

**Genes involved:** SLC1A6, SLC6A2, ATP6V0A4, ATP6V0D2, ATP6V1B1 e ATP6V1G3

**SLC1A6 and SLC6A2**

Sodium-dependent, high-affinity amino acid transporter that mediates the uptake of L-glutamate and also L-aspartate and D-aspartate (PubMed:[7791878](https://www.uniprot.org/citations/7791878)). Functions as a symporter that transports one amino acid molecule together with two or three Na+ ions and one proton, in parallel with the counter-transport of one K+ ion. Mediates Cl- flux that is not coupled to amino acid transport; this avoids the accumulation of negative charges due to aspartate and Na+ symport (By similarity). Plays a redundant role in the rapid removal of released glutamate from the synaptic cleft, which is essential for terminating the postsynaptic action of glutamate (Probable). < <https://www.uniprot.org/uniprot/P48664>>