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ERRATUM

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The sodium/glucose cotransport family SLC5

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Figure 1 and Table 1 have been revised to reflect the recently approved HUGO gene names for newer members of the SLC5 family, namely those for AIT (SLC5A8) and SGLTs 4-6 (SLC5A9-11). In addition, a reference has been added documenting the function of SGLT3 (SLC5A4) as a glucose sensor.

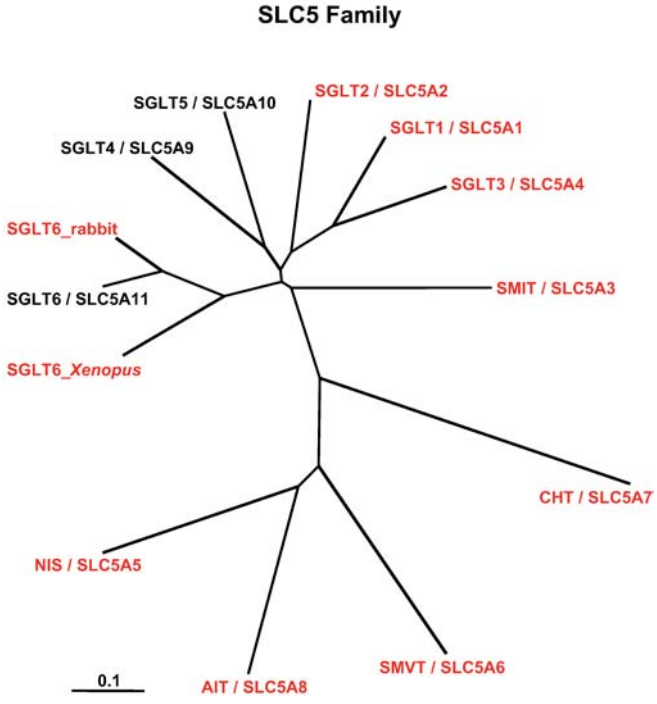

Fig. 1 Unrooted phylogenetic tree of the 11 human members of the SLC5 family of cotransporters and two other vertebrate members of known function. The alignment program CLUSTAL W (http://www.ebi.ac.uk/clustalw/) and the phylogenetic display program TreeViewPPC (http://taxonomy.zoology.gla.ac.uk/rod/treeview. html) were used to generate the tree. Members for which transport functions have been demonstrated experimentally are shown in *red*. Members shown in *black* have yet to have their function determined. In a pairwise basic local alignment search tool (BLAST) analysis the amino acid identities relative to SGLT1 (SLC5A1) were 59% SGLT2, 70% SGLT3, 56% SGLT4, 57% SGLT5, 50% SGLT6, 55% SMIT, 24% SMVT, 24% CHT, 21% AIT, and 21% NIS (*SGLT* Na^+/glucose transporter, *SMIT* Na^+/*myo*-inositol transporter, *SMVT* Na^+/multivitamin transporter, *CHT* choline transporter, *AIT* apical iodide transporter, *NIS* Na^+/iodide transporter)

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Table 1 The SLC5 sodium glucose cotransporter family (*OMIM* Online Mendelian Inheritance in Man database, *TMH* transmembrane helix, *aa* amino acids)

Human gene name	Protein name	Aliases	Predominant substrates	Transport type/coupling ion*	Tissues distribution and cellular/subcellular expression[T]	Link to disease	Human gene locus	Sequence accession ID	Splice variants and their specific features[S]
SLC5A1	SGLT1	None	Glucose and galactose (urea and water)	C/Na^+ (H^+) F/Na^+ (H^+). Channel: urea and water	Small intestine>>>>trachea, kidney and heart; plasma membranes	Glucose galactose malabsorption[G] OMIM 182380	22q13.1	NM_000343	
SLC5A2	SGLT2	None	Glucose	C/Na^+	Kidney cortex	Familial renal glycosuria[G] OMIM 182381	16p12-p11	NM_003041	
SLC5A3	SMIT	None	*myo*-inositol (glucose)	C/Na^+	Brain, heart, kidney and lung; plasma membranes	Down's syndrome? OMIM 600444	21q22.12	NM_006933	Splicing within, and distal to exon 2 leads to 3 transcripts (SMIT1, SMIT2, SMIT3). SMIT2 and -3 lack the 14th TMH
SLC5A4	SGLT3	SAAT1	Na^+ (H^+)	Glucose sensor[U]	Small intestine (cholinergic neurons), skeletal muscle, kidney, uterus and testis; plasma membranes		22q12.2-q12.3	NM_14227	
SLC5A5	NIS		I^-(ClO_4^-, SCN^-, NO_3^-, Br^-)	C/Na^+ Uniporter Na^+ Channel Urea water	Thyroid, breast, colon and ovary; plasma membranes	Thyroid hormon ogenesis[G] OMIM 601843	19p13.2-p12	NM_ 000453	
SLC5A6	SMVT		Biotin, lipoate and pantothenate	C/Na^+	Brain, heart, kidney, lung and placenta; plasma membranes		2p23	NM _021095	
SLC5A7	CHT	CHT1	Choline	C/Na^+/Cl^-	Spinal cord and medulla (intracellular vesicles)		2q12	NM _021815	
SLC5A8	AIT		Iodide	?	Thyroid, apical plasma membrane		12q23.1	NM_145913	
SLC5A9**	SGLT4				Small intestine, kidney; liver, lung and brain		1p32	HCT1951464	Alternative splice in exon 14 yields either 38 or 53 aa between TMHs 13–14
SLC5A10	SLGT5	Rabbit RK-D			Kidney		17p11.2	XM_064487	First 16 aas of exon 10 may be spliced out. An additional 37 aa exon may bge spliced in between exons 11 and 12

Table 1 (continued)

Human gene name	Protein name	Aliases	Predominant substrates	Transport type/coupling ion*	Tissues distribution and cellular/subcellular expression[T]	Link to disease	Human gene locus	Sequence accession ID	Splice variants and their specific features[S]
SLC5A11	SGLT6	KST1 Rabbit ST1 Rabbit SMIT2	Rabbit ortholog: *myo*-inositol, chiro-inositol, glucose and xylose. *Xenopus laevis* ortholog: *myo*-inositol and glucose	C/Na^+	Small intestine, brain, kidney, liver, heart, and lung		16p12.1	NM_052944	Splicing eliminates exon 6 and TMH 4

* *C* cotransporter; *F* uniporter. Function based on results obtained with heterologous expression systems

** Provisional SGLT4 exons identified by mining the Celera databases

[T] Tissue distribution of SGLT1–6 was determined by RNAase protection assays using gene specific probes (Bing M, Turk E, Martin MG, Wright EM, in preparation). Also includes data from the original cloning papers and GeneCard (EST, and/or DNA array)

[G] Gene defect

[S] Potential alternative splice sites identified by Roll et al. (2002), Porcellati et al. (1999) and Turk (unpublished)

Original references may be obtained through the Accession numbers and/or the text

[U] Diez-Sampedro A, Hirayama BA, Osswald C, Gorboulev V, Baumgarten K, Volk C, Wright EM, Koepsell H (2003) A glucose sensor hiding in a family of transporters. Proc Nat Acad Sci (USA) 100:11753–11758

Note: single nucleotide polymorphisms (SNPs) and variants in the NCBI SNP database are not included