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EXAMINING THE GENOMIC ARCHITECTURE OF NEURONAL GLUCOSE TRANSPORTER 3 FROM AN EVOLUTIONARY PERSPECTIVE

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Neuronal glucose transporter 3 (Glut3, *SLC2A3*) is essential in providing energy supply for neurons. Using mice as disease-models homozygous *slc2a3* knock-out was found to be lethal in early embryonic phase while heterozygous form was associated to autism spectrum disorders.

The genomic region where *SLC2A3* is located (12p13.3) is investigated intensely since *NANOG* – a homeobox gene critical in embryogenesis – and one of its impressive number of pseudogenes are also located here. Interestingly, this region also codes the gene *SLC2A14* which is selectively expressed in testis and lung but shows 95% homology with *SLC2A3*. A tandem duplication event involving *NANOG* and *SLC2A3* was proposed as a possible explanation to the current genomic architecture.

Since *SLC2A3* was associated to a number of mental disorders this study aimed to examine *SLC2A3* and the related region in an evolutionary perspective. Ensembl BLAST search and multi-species alignment were used to provide a comparison.

Our results support the notion that the region of 12p13.3 underwent a tandem duplication event which gave rise to new paralogues of *NANOG* and *SLC2A3*. Out of these *SLC2A3* and *SLC2A14* both remained functional. We argue that the region can be still a genomic spot susceptible to further rearrangements.

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