Life Sciences Seminar

What a bacterial carboxylate transporter structure has told us about the pathogenesis of early-onset epileptic encephalopathy

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In the neuron, citrate functions as an energy source and a precursor for lipid synthesis. The concentration of cytosolic citrate partially depends on direct import across the plasma membrane via the Na+-dependent citrate transporter (NaCT). Mutations in NaCT (encoded by the SLC13A5 gene) cause early onset epilepsy encephalopathy and developmental delays, and the disease is thus named SLC13A5 Deficiency. Based on their cellular expression patterns, the mutations have been classified as Type I and Type II mutations. NaCT and its bacterial homologs belong to the divalent anion/sodium symporter family. Our lab previously determined the crystal structure of a bacterial NaCT homolog (VcINDY). The structure reveals a VcINDY dimer, where each monomer consists of an anchoring domain and a transport domain. This VcINDY structure, and the transport mechanism it suggested, have proven to be largely applicable to human NaCT and can explain the molecular defects caused by SLC13A5 mutations.

Monday, January 14, 2019 02:30pm - 03:30pm
Mondi Seminar Room 3, Central Building

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