**Podicin - interdependece of pathogenicity, dimerization and molecular structure**

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We have shown that p.R286Tfs\*17 podocin is membrane-associated in human podocyte cell cultures, while the p.A317Lfs\*31,  p.F344Lfs\*4 and p.L346Yfs\*2 mutations cause internal retention of podocin, in accordance with their pathogenicity. Molecular-modelling results indicate that in case of p.R286Tfs\*17 podocin, the frameshift mutants form disulfide linkages with each-other, thus shielding their helical interaction surfaces from any association with the p.R229Q or WT variants. Expression of the C-terminal intracellular fragment of podocin (for crystallization and structure determination purposes) – bound to fusion proteins – has been successfully carried out. Coiled-coil domains of WT and mutants have been investigated using GFC, CD and NMR, and showed that structural consequences of pathogenicity are detectable already on this scale.