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Modeling of β-catenin signaling in Medulloblastoma

Medulloblastoma is a brain tumor that mainly affects children and is caused by several mutations. Our research is devoted to understanding of the role of monosomy and trisomy of the *6* chromosome. Each perturbation is characterized by extremely different prognosis. Trisomy is found to have a very bad prognosis and monosomy surprisingly good after medical treatment. *6q loss* and *6q gain* are related with difference in expression of cMyc, SGK1, which are target genes of β-catenin signaling in mutated cells. Our observations suggest that disruption in chromosome balance strongly affects the mentioned signaling pathway. However, the mechanism is still not explained. We can only see consequences which result in different mRNA levels of cMyc and SGK1. It is also not well understood how these differences influence the prognosis. Thus investigation of particular interactions between proteins is so interesting. We propose an ODE model describing complicated dynamics of chosen genes, concerning transcription, translation as well as transport between cytoplasm and nucleus. We calibrate models based on clinical data for both types of medulloblastoma. Simulations lead to a better understanding of the process. In particularly, the model indicates the important role of SGK1 gene in the process of oncogene cMyc production leading to cancer relapse.