Title of the abstract
The role of Retinal Degeneration Protein 3 in visual dysfunction and tumor development

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1. Main Text

Photoreceptor guanylate cyclases (GCs) are key enzymes in photoreceptor physiology, since they synthesize cGMP, the intracellular messenger of photoreceptor excitation. GCs are under Ca^{2+}-dependent negative feedback control by guanylate cyclase-activating proteins (GCAPs). Numerous missense mutations in the genes *GUCY2D* and *GUCA1A* coding for GC-E and GCAP1, respectively, cause retinal diseases like Leber congenital amaurosis or cone-rod dystrophy. One of the main cellular consequences of these mutations seems to be an imbalance of the two secondary messengers, cGMP and Ca^{2+} [1]. The retinal degeneration protein RD3 adds a further twist to the regulatory processes of the GC-GCAP complex in photoreceptor cells. Among its main functions is the inhibition of photoreceptor GCs during trafficking from the inner to their final destination in the outer segment [2] and mutations in RD3 correlate with Leber congenital amaurosis type 12 [3]. However, RD3 is also found in non-retinal tissue and can control the activities of hormone receptor guanylate cyclases GC-A and GC-B [4]. Loss of RD3 correlates with the development of aggressive neuroblastoma cancer [5]. On the other hand, overexpression of RD3 decreases cell viability leading to cell-cycle arrest and induction of cell apoptosis. RD3 expression level seems to be important for a balance of cell death and cell survival [6].

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3. References

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