Karyopherin

The karyopherin protein family comprises importins and exportins which are nucleocytoplasmic shuttling receptors. Increased levels of karyopherin a2 and chromosome region maintenance protein 1 correlate with a higher WHO grade and a poorer prognosis in patients with infiltrative astrocytomas.

Gousias et al., semiquantitatively analyzed nuclear expression of karyopherin a2, chromosome region maintenance protein 1 and the MIB1 index using immunohistochemistry in 108 primary (44 meningiomas WHO grade I, 48 meningiomas WHO grade II, 16 meningiomas WHO grade III) and 13 recurrent meningiomas. Statistical analysis was performed using standard techniques. Karyopherin a2 ($p < 0.001$) and chromosome region maintenance protein 1 ($p = 0.002$) expression correlated significantly with the histological grade. Karyopherin a2 expression correlated with proliferative activity as assessed by the MIB1 index ($p < 0.001$). Recurrent tumors expressed significantly higher levels of karyopherin a2 ($p = 0.045$) when compared to primary growths. Multivariate analysis of the overall series as well as of patients with atypical meningiomas identified higher karyopherin a2 (≥5 vs. <5 %) and chromosome region maintenance protein 1 (≥60 vs. 60 %) expression as independent predictors of tumor recurrence. Karyopherin a2 and chromosome region maintenance protein 1 expression may have potential as novel biomarkers for meningiomas [1].

Karyopherinβ1 (KPNB1), one of the cytosolic factors involved in the selective protein transport across nucleus, docked at nuclear pore complex and transported through nuclear envelope in an ATP-dependent style, assisting proteins to be recognized as import substrates. It has been reported to be bound up with the origination and progress of lung cancer, cervical cancer, head and neck cancer and hepatocellular carcinoma. In current study, we demonstrated for the first time that the role of KPNB1 in human glioma. KPNB1 was over-expressed as the well-known trend of Ki-67($p < 0.01$) and tightly closed to poor prognosis, as an independent prognostic factor. In vitro, up-regulation of KPNB1 was accompanied by certain rising levels of proliferation markers, employing U251 and U87MG cells as serum-starve models. Silencing KPNB1 in U251 and U87MG led to G1 phase arrested directly via flow cytometry analysis. In the nucleus of KPNB1-depletion cell models, the decreasing expression of KPNB1 and β-catenin was detected respectively, which indicated that KPNB1 functioned via β-catenin signal. Besides, the interaction between KPNB1 and β-catenin was proved clearly by immunoprecipitation. Taken together, it showed that KPNB1 might enhance human glioma proliferation via Wnt/β-Catenin Pathway [2].

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2)
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