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Over-expressions of Golph3 family in ovarian carcinomas with significant clinical implications

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Background: Nowadays, two members were found at Golph3 family, Golph3 and its paralogue Golph3L. Golph3 has recently been reported toinvolve in the clinical progression in several human cancers including ovarian cancer. However, the expression status and function of Golph3L were rarely reported. We explored the expressions of Golph3 and Golph3L in ovarian cancer and their relationship with the disease.

Methods: The expressions level of Golph3 and Golph3L were detected by ELISA, quantitative PCR (Q-PCR), western blot (WB) and immunohistochemical staining (IHC). Their expression levels in ovarian tumors were compared with normal, borderline tissues and also correlated with clinicopathological parameters.

Results: No detectable Golph3 and Golph3L expression in serum. A continuous up regulation of Golph3 and Golph3L in the order of normal, borderline and malignant tissues was observed by Q-PCR, western-blot and IHC detection. Statistical analysis based on IHC detection showed significant difference ($P<0.001$ &$P<0.05$). Univariate and multivariate analysis indicate that overexpression of Golph3 and Golph3L are associated with clinical stage ($P=0.006$, $P=0.03$), T classification ($P=0.07$, $P=0.04$), N classification ($P=0.02$, $P=0.03$), chemo-sensitivity ($P=0.045$, $P=0.045$), tumor-free survival ($P=0.014$, $P=0.034$) and overall survival time ($P=0.023$, $P=0.037$). Univariate and multivariate analysis showed that Golph3 and Golph3L overexpression were, respectively, independent prognostic factor in ovarian cancer. Kaplan-Meier analysis revealed that patients with Golph3 and/or Golph3L over-expression experienced significantly disease-free and much shorter overall survival time (log-rank $P<0.001$ &$P<0.05$).

Conclusions: Both Golph3 and Golph3L were over-expression in ovarian cancer. Correlations with clinical parameters suggested that Golph3 and Golph3L are both independent prognostic factors for ovarian cancer.

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Synthesis of humanin and its derivatives to treat traumatic brain injury in mice

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Humanin is a 24-amino acid peptide known for its anti-apoptotic activity, especially against neuronal cell death caused by Alzheimer insults. Herein, we show a novel function of humanin and its derivatives, namely protection against necrosis, demonstrated both in vitro and in vivo. The synthesis of humanin is difficult due to hydrophobic amino acids that impose aggregation on the resin. Solid-phase peptide synthesis of humanin and its three derivatives, AGA-HNG, HNG and HN17 gave low yields. In order to avoid aggregation and overcome difficult sequences couplings, we developed efficient synthetic procedures that are based on fragment condensation in solution. Furthermore, native chemical ligation was applied to overcome resin aggregation for synthesis of peptides that contain cysteine. We found that humanin and its derivatives conferred protection in PC-12 and NSC34 cell lines in which necrosis was induced in glucose free medium by either chemohypoxia or upon staurosporine/oligomycin-A treatment. Moreover, in-vivo protective effect was shown in traumatic brain injury model in mice, where necrosis is the main mode of the neuronal cell death. We show that humanin derivatives antagonize the decrease in ATP levels associated with necrosis and also directly enhance the activity of isolated ATP synthase complex, indicating that humanin derivatives target the mitochondria, regulating ATP levels. The present findings could provide new therapeutic protocols for treatment of brain ischemic states, such as stroke, and traumatic brain injury, conditions for which no efficient drug-based treatment is currently available.

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