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Axl glycosylation mediates tumor cells proliferation, invasion and the lymphatic metastasis in murine hepatocarcinoma

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Aim: To investigate the possible effect of Axl deglycosylation on regulation of tumor lymphatic metastasis in mouse hepatocarcinoma cell lines.

Methods: Western blot analysis was used to evaluate the expression profile of Axl glycoprotein in the mouse hepatocarcinoma cell lines Hca-F which were treatmented with tunicamycin and PNGase F. MTT assay, ECM invasion assay in vitro and tumor metastasis assay in vivo were utilized to evaluate the effect of Axl deglycosylation on the Hca-F cells proliferation, invasion and the lymphatic metastasis.

Results: By treatment with tunicamycin and PNGase F, the expression level of Axl glycoprotein was markedly changed. The new bands that appeared were consistent with the size of the core protein, which were the result of tunicamycin and PNGase F inhibiting Axl glycosylation. Further analysis of the regulation of Axl glycosylation by tunicamycin or PNGase F treatment in Hca-F cells showed partial inhibition of biosynthesis, as well as led to the decreased proliferation, invasion and metastasis to peripheral lymph nodes both in vitro and in vivo ($aP<0.05$).

Conclusion: The alterations of Axl glycosylation are responsible for overcoming tumor cell lymphatic metastasis, and the Axl N-glycans could be a universal target for chemotherapy.

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