Taurolidine

The antibacterial aminoacid derivative taurolidine (TAU) has been recently shown to exhibit antineoplastic activity based on a mechanism, which is still unknown in detail. Cytotoxicity and clonogenic assays were performed and the impact of apoptosis modulators, a radical scavenger, autophagy inhibitors, silencing of apoptosis inducing actor (AIF) and cytochrome-c (Cyt-C) by siRNA, and knockdown of autophagy related genes were evaluated in vitro. The intracellular ATP-content, release of AIF and Cyt-C, and DNA-laddering were investigated. This study could demonstrate cell killing, inhibition of proliferation, and inhibition or prevention of colony formation in human glioma cell lines and ex vivo glioblastoma cells after incubation with TAU. This effect is based on the induction of a mixed type of programmed cell death with the main preference of autophagy, and involvement of senescence, necroptosis and necrosis. This mechanism of action may open a new approach for therapeutic intervention [1)].

Taurolidine enhances the oxidative stress (ROS) selectively in tumor cells. Its cytotoxicity for various tumor cells in vitro and in vivo, which includes tumor stem cells, is based on the induction of programmed cell death, largely via apoptosis but also necroptosis and autophagy [2)]

1)
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2)
Möhler H, Pfirrmann RW, Frei K. Redox-directed cancer therapeutics: Taurolidine and Piperlongumine as broadly effective antineoplastic agents (review). Int J Oncol. 2014 Oct;45(4):1329-36. doi: 10.3892/ijo.2014.2566. Epub 2014 Jul 28. PubMed PMID: 25175943; PubMed Central PMCID: PMC4151817.