Ampelopsin

Ampelopsin (Amp), an effective component of the traditional Chinese herb of Ampelopsis grossedentata, is reported to have important biological properties, including anti-inflammatory, anti-cancer, and anti-oxidant activity; however, its effects on human glioma are poorly understood. Here, the in vitro and in vivo study was performed to investigate the anti-glioma ability of Ampelopsin. Human glioma cell lines of U251 and A172 were treated with Ampelopsin (0, 25, 50, and 100 uM) for 24 h, followed by various analysis. And human glioma xenograft models were established by injecting U251, accompanied with administration of Ampelopsin at 50 and 100 mg/kg to confirm the anti-cancer role of Ampelopsin. We found that Ampelopsin could suppress the glioma cell proliferation by modulating G1 and S phase arrest. Incubation with Ampelopsin led to the activity of Caspase-8, Caspase-9, Caspase-3 and poly (ADP-ribose) polymerases (PARP), indicating that Ampelopsin induced apoptotic response via both intrinsic and extrinsic signaling pathways. Additionally, autophagy was also observed in Ampelopsin-treated cancer cells, which is evidenced by autophagosome formation and LC3B-II accumulation. Ampelopsin-caused cancer cell death was obviously regained by apoptosis inhibitors. Further, Ampelopsin activated c-Jun N-terminal protein kinase (JNK) expression and enhanced reactive oxygen species (ROS) generation. Suppressing JNK markedly ameliorated Ampelopsin-induced apoptosis and autophagy, and ROS scavenger exhibited similar results. In vivo, Ampelopsin inhibited tumor growth and progression in mouse xenograft models. In conclusion, our findings indicated that Ampelopsin led to G1 and S phase arrest, triggered apoptosis and autophagy through potentiating ROS generation and JNK activation in human glioma cells. Thus, Ampelopsin might be a promising candidate against human glioma [1)].

1)
Guo Z, Guozhang H, Wang H, Li Z, Liu N. Ampelopsin inhibits human glioma through inducing apoptosis and autophagy dependent on ROS generation and JNK pathway. Biomed Pharmacother. 2019 May 16;116:108524. doi: 10.1016/j.biopha.2018.12.136. [Epub ahead of print] PubMed PMID: 31108349.