Selenocystine

Selenocystine (SeC) a natural available Selenium-containing aminoacid showed novel anticancer potential through triggering oxidative damage-mediated apoptosis. However, whether TrxR-mediated oxidative damage was involved in SeC-induced apoptosis in human glioma cells has not been elucidated yet.

SeC-induced human glioma cell apoptosis was detected in vitro, accompanied by PARP cleavage, caspases activation and DNA fragmentation. Mechanically, SeC caused mitochondrial dysfunction and imbalance of Bcl-2 family expression. SeC treatment also triggered ROS-mediated DNA damage and disturbed the MAPKs and AKT pathways. However, inhibition of ROS overproduction effectively attenuated SeC-induced oxidative damage and apoptosis, and normalized the expression of MAPKs and AKT pathways, indicating the significance of ROS in SeC-induced apoptosis. Importantly, U251 human glioma xenograft growth in nude mice was significantly inhibited in vivo. Further investigation revealed that SeC-induced oxidative damage was achieved by TrxR1-targeted inhibition in vitro and in vivo.

The findings validated the potential of SeC to inhibit human glioma growth by oxidative damage-mediated apoptosis through triggering TrxR1-targeted inhibition [1)].

1)
Fan CD, Fu XY, Zhang ZY, Cao MZ, Sun JY, Yang MF, Fu XT, Zhao SJ, Shao LR, Zhang HF, Yang XY, Sun BL. Selenocysteine induces apoptosis in human glioma cells: evidence for TrxR1-targeted inhibition and signaling crosstalk. Sci Rep. 2017 Jul 25;7(1):6465. doi: 10.1038/s41598-017-06979-2. PubMed PMID: 28743999; PubMed Central PMCID: PMC5526989.