

# Selenium and glioma

Reliable supply of [selenium](#) is important since selenium compounds can affect [tumor microenvironment](#) and [neoangiogenesis](#) in [malignant gliomas](#) via induction of [apoptosis](#) and alteration of [matrix metalloproteinases](#) expression.

In 1990 Philipov and Tzatchev added selenium tablets to the diet of 15 patients with malignant brain tumors. In twelve patients with [glioblastoma multiforme](#) this treatment didn't prolong the postoperative survival <sup>1)</sup>.

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Yakubov et al. summarized findings focusing on the anti-toxicity and cancer-preventive properties of selenium and their implication in current multimodal therapies including [temozolomide](#) (Temodal), cyclophosphamide (Endoxan), and cisplatin (DDP, Platiblastin, and Platinol).

They shedded light on unintended side effects in [chemotherapy](#) and the developments of novel combinatorial chemotherapeutics with selenium compounds. They found that selenium and selenium compounds have dual action profiles with direct anti-cancer and chemotherapy-intensifier effects as well as neuroprotective and cytoprotective agents <sup>2)</sup>.

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[Thioredoxin reductase](#) (TrxR) as a [selenium](#) (Se)-containing antioxidantase plays key role in regulating intracellular [redox](#) status. Selenocystine (SeC) a natural available Se-containing amino acid 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. Herein, 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 <sup>3)</sup>.

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In a case-control study of glioma, Peeri et al., examined the associations of selenium in toenails and genetic variants in the [selenoenzyme pathway](#) with the risk of glioma and patient survival. A total of 423 genetic variants in 29 candidate genes in the selenoenzyme pathway were studied in 1547 glioma cases and 1014 healthy controls. Genetic associations were also examined in the UK Biobank cohort comprised of 313,868 persons with 322 incident glioma cases. Toenail selenium was measured in a subcohort of 300 glioma cases and 300 age-matched controls from the case-control study.

None of the 423 variants studied were consistently associated with glioma risk in the case-control and

cohort studies. Moreover, toenail selenium in the case-control study had no significant association with glioma risk (p trend = 0.70) or patient survival among 254 patients with high grade tumors (p trend = 0.70).

The present study offers no support for the hypothesis that selenium plays a role in the onset of glioma or patient outcome <sup>4)</sup>.

## References

<sup>1)</sup>

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<sup>2)</sup>

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<sup>4)</sup>

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