Aggressive metastasis of tumor cells assumed a constructive role in strengthening the chemoresistance of tumors, so this investigation was intended to elucidate if lncRNA CCAT2 sponging downstream miR 424 regulated chemotolerance of glioma cells by boosting metastasis of glioma cells.

One hundred and twenty-eight pairs of glioma tissues and corresponding adjacent tissues were resected from glioma patients during their operation, and we also purchased a series of glioma cell lines, including U251, U87, A172 and SHG44. Furthermore, pcDNA3.1-CCAT2, si-CCAT2, miR-424 mimic and miR-424 inhibitor were transfected into SHG44 and U251 cell lines, so as to evaluate impacts of CCAT2 and miR-424 on chemosensitivity of the glioma cells. Besides, proliferation, invasion, and metastasis of the cells were determined through the implementation of colony formation assay, transwell assay and scratch assay.

Glioma tissues and cells were monitored with higher CCAT2 expression and lower miR-424 expression than adjacent normal tissues and NHA cell line ($P<0.05$). Among the glioma cell lines, the SHG44 cell line showed the strongest resistance against teniposide, temozolomide and cisplatin ($P<0.05$), whereas the U251 cell line was more sensitive to teniposide, temozolomide, vincristine and cisplatin than any other cell line ($P<0.05$). Besides, pcDNA3.1-CCAT2 and miR-424 inhibitor could enhance tolerance of glioma cell lines against drugs ($P<0.05$). Moreover, in-vitro transfection of si-CCAT2 and miR-424 mimic could significantly retard proliferation, invasion and migration of SHG44 and U251 cells ($P<0.05$), and CCAT2 was found to negatively regulate miR-424 expression by sponging it ($P<0.05$). In addition, CHK1 was deemed as the molecule targeted by upstream miR-424, and its overexpression can changeover the effects of miR-424 mimic on proliferation and metastasis of SHG44 and U251 cells.

lncRNA CCAT2/miR-424/Chk1 axis might serve as a promising target for improving chemotherapeutic efficacies in glioma treatment [1].

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