Cell division

Mitotic activity refers to the process of cell division, specifically mitosis, which is essential for growth, development, and tissue repair in multicellular organisms. Mitosis involves the replication and division of a cell's chromosomes into two identical sets, resulting in the formation of two daughter cells that are genetically identical to the parent cell.

Mitotic activity can be observed and measured in tissues undergoing active cell division. It is often assessed in medical and research contexts to evaluate tissue health, growth rates, and pathological conditions such as cancer. High mitotic activity may indicate rapid cell proliferation, which can be characteristic of cancerous growth, while low mitotic activity may indicate normal tissue turnover or a state of quiescence. Mitotic activity is commonly quantified by counting the number of dividing cells within a given area of tissue under a microscope.

Signaling pathways in a cell work together to control one or more cell functions, such as cell division or cell death.

Cell division cycle 2 (CDC2) is always overexpressed in malignant tumor cells and is correlated with chemosensitivity, but it is unclear whether CDC2 overexpression contributes to the chemoresistance potential of glioma cells.

In a study, the glioblastoma U87 and U251 cell lines were steadily transfected with a lentivirus vector expressing a short hairpin RNA-targeting CDC2. Expression of CDC2 was evaluated in glioblastoma and cell lines by immunohistochemistry and Western blot analysis. The relationship between CDC2 expression and clinicopathological characteristics was analyzed. Using RNA interference, the effects of CDC2 on chemosensitivity to temozolomide (TMZ) were investigated in U87 and U251 cell lines in vitro. Combined CDC2 knockdown and TMZ treatment inhibited cell proliferation and induced apoptosis in vitro more effectively than either treatment alone. qRT-PCR and Western blot analysis showed that cells underexpressing CDC2 revealed lower expression of the anti-apoptotic protein B cell lymphoma-2 and increased expression of the apoptosis effector caspase-3 compared to U87 and U251 cells transfected with a control vector. Furthermore, expression levels of CDC2 in U87 and U251 cells were related to the IC50 of the antitumor drug TMZ. Knockdown of CDC2 expression was associated with decreased expression of Ral-binding protein 1, a classical chemotherapy drugs transporter. These results indicate that the ability to suppress the malignant phenotype by down-regulating CDC2 expression may provide a new gene therapy approach for overcoming CDC2-associated chemoresistance in patients with malignant glioma [1)].

Tumor Treating Fields disrupt cell division through physical interactions with key molecules during mitosis. This non-invasive treatment targets solid tumors.

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Zhou B, Bu G, Zhou Y, Zhao Y, Li W, Li M. Knockdown of CDC2 expression inhibits proliferation, enhances apoptosis, and increases chemosensitivity to temozolomide in glioblastoma cells. Med Oncol. 2015 Jan;32(1):378. Epub 2014 Nov 30. PubMed PMID: 25433945.