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## **Genetic alterations and signaling pathways in the evolution of gliomas.**

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Gliomas are the most common primary brain tumors. They account for more than 70% of all neoplasms of the central nervous system and vary considerably in morphology, location, genetic alterations, and response to therapy. Most frequent and malignant are glioblastomas. The vast majority (>90%) develops rapidly after a short clinical history and without evidence of a less malignant precursor lesion (primary or de novo glioblastoma). Secondary glioblastomas develop more slowly through progression from low-grade or anaplastic astrocytoma. These glioblastoma subtypes constitute distinct disease entities that affect patients of different age, develop through distinct genetic pathways, show different RNA and protein expression profiles, and may differ in their response to radio- and chemotherapy. Recently, isocitrate dehydrogenase 1 (IDH1) mutations have been identified as a very early and frequent genetic alteration in the pathway to secondary glioblastomas as well as that in oligodendroglial tumors, providing the first evidence that low-grade astrocytomas and oligodendrogliomas may share common cells of origin. In contrast, primary glioblastomas very rarely contain IDH1 mutations, suggesting that primary and secondary glioblastomas may originate from different progenitor cells, despite the fact that they are histologically largely indistinguishable. In this review, we summarize the current status of genetic alterations and signaling pathways operative in the evolution of astrocytic and oligodendroglial tumors. (*Cancer Sci* 2009; 00: 000-000).

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