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Asymmetric stem cell division in development and cancer.

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Asymmetric stem cell division leads to another stem cell via self-renewal, and a second cell type which can be either a differentiating progenitor or a postmitotic cell. The regulation of this balanced process is mainly achieved by polarization of the stem cell along its apical-basal axis and the basal localization and asymmetric segregation of cell fate determinants solely to the differentiating cell. It has long been speculated that disturbance of this process can induce a cancer-like state. Recent molecular genetic evidence in Drosophila melanogaster suggests that impaired polarity formation in neuroblast stem cells results in symmetric stem cell divisions, whereas defects in progenitor cell differentiation leads to mutant cells that are unable to differentiate but rather continue to proliferate. In both cases, the net result is unrestrained self-renewal of mutant stem cells, eventually leading to hyperproliferation and malignant neoplastic tissue formation. Thus, deregulated stem cells can play a pivotal role in Drosophila tumor formation. Moreover, recent evidence suggests that so-called cancer stem cells may drive the growth and metastasis of human tumors too. Indeed, cancer stem cells have already been identified in leukemia, and in solid tumors of the breast and brain. In addition, inappropriate activation of pathways promoting the self-renewal of somatic stem cells including defects in asymmetric cell division has been shown to cause neoplastic proliferation and cancer formation. Taken together, these data indicate that evolutionary conserved mechanisms regulate stem and progenitor cell self-renewal and tumor suppression via asymmetric cell division control.

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