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PhD Program in Molecular Biomedicine

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Seminar room 121d, Building Q – Via Giorgieri, 5

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HMGA1 and the regulation of stem cell asymmetric division

Stem cells (SCs) provide tissue homeostasis by appropriately choosing between two different division modes: symmetric division, which generates two daughter SCs and asymmetric division, which generates one self-renewing SC and one differentiating cell. The choice between these two modes has to be finely regulated: deregulation of genes controlling asymmetric division induces increase of the SC pool, hyperproliferation and cancer. Conversely, recovering asymmetric division ability is onco-suppressive. The mechanisms leading to asymmetric division as well as the asymmetric partitioning of cell fate determinants, are not completely understood. Brain tumor stem cells (BTSCs), as other cancer stem cells, have reduced ability to divide asymmetrically and they mostly divide symmetrically. HMGA1 is an architectural transcription factor, overexpressed in many malignant tumors and stem cells, including BTSCs. Here, we provide evidence that HMGA1 can take part in the decision between symmetric and asymmetric division, affecting non-random chromosome segregation as well as NUMB expression and localization.

