

BiolSI Research Seminar

A New Role for Arl13b in Cancer Cell Migration and Tumor Formation



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When: July 5, Thursday - 11h30
Where: Building C1 Room 1.3.33 A

Cell migration is essential for homeostatic processes, such as wound healing. Moreover, cell migration has an important role in tumor dissemination, a process by which tumor cells leave the primary tumor and migrate to distant tissues where they form metastases. Therefore, it is essential to understand the mechanisms underlying cell migration in order to develop new targeted therapeutic strategies for controlling invasive tumor cells.

Cancer cell migration and invasion are regulated by intracellular proteins of the Ras superfamily of small GTPases. Among these, proteins of the ADP-ribosylation factor (Arf) family, such as Arf6 and Arf1 have been associated with actin remodeling, vesicle formation and cancer progression. Arl proteins are a subfamily of the Arf family of proteins that regulate cytoskeleton organization and vesicle tethering to acceptor membrane compartments. In particular, Arl13b has been associated with cilogenesis and sonic hedgehog signaling, which plays a critical role in aggressive cancers. Previously, we described that Arl13b regulates endocytic recycling traffic and that it interacts with the non-muscle myosin heavy-chain IIA (NMIIA) in a GTP-dependent manner to regulate actin cytoskeleton remodeling and fibroblast migration. Moreover, NMIIA was described to regulate breast cancer cell spreading and migration.

In this study, we used breast cancer cells to define the role of Arl13b in cancer cell migration and invasion. We found that the silencing of Arl13b leads to the impairment of breast cancer cell migration and invasion, as well as cell adhesion defects. Moreover, we observed that Arl13b depletion inhibits tumor growth and metastasis formation in mice. In contrast, Arl13b overexpression enhances breast cancer cell migration and invasion, as well as tumor growth and metastasis in mice. Furthermore, our data indicates that Arl13b is overexpressed in highly invasive breast cancer cell lines and cancer patient breast tissue samples. Altogether, our findings suggest that Arl13b regulates migration and invasion of invasive cancer cells, acting as an oncogene.