Seminar/Talk

Function and Regulation of Centrosomes during Immune Responses

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Host: Michael Sixt

Centrosomes function as the major microtubule-organizing center in most animal cells. They promote the assembly of the bipolar mitotic spindle, which segregates sister chromatids into two daughter cells. To assure spindle bipolarity, centrosome duplication is precisely controlled and limited to once per cell cycle. Defects during the duplication cycle lead to centrosome amplification a hallmark of almost all solid tumors and hematological malignancies. To allow cancer cells to proceed through mitosis despite their surplus of centrosomes, excess centrosomes tightly cluster into a pseudo-bipolar spindle configuration. Novel classes of anti-cancer therapies induce centrosome de-clustering thus driving malignant cells into a multipolar mitosis and subsequent cell death. Similarly to cancer cell, primary dendritic cells, which are the most potent antigen presenting cells of the innate immune system, exhibit an increased number of centrosomes. Excess centrosomes cluster during migration and pharmacological induction of centrosome de-clustering leads to polarization defects and loss of directed cell migration. Our data indicate that amplified centrosomes are not only a marker for malignancy but actually promote immune cell effector functions such as antigen presentation and T cell activation. We are further interested in the molecular basis of how amplified centrosomes enhance immune responses, which will critically impact the use of centrosome-based cancer therapies.

Tuesday, May 21, 2019 01:30pm - 02:30pm
Mondi Seminar Room 2, Central Building

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