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Aurora-A: To be or not to be an oncogene or a tumour suppressor

In 1997 Aurora-A was discovered overexpressed in cancers, since then the kinase has been considered by pharmaceutical companies as a priority target to develop inhibitors to be used in cancer therapy.

In 1998 the function of the kinase in mitotic bipolar spindle assembly was identified together with its oncogenic activity. Overexpression of the kinase is sufficient to transform cell and induce tumour formation in nude mice. A direct relationship between Aurora-A and tumour formation was discovered in 2006 by the demonstration that overexpression of the kinase in mouse mammary epithelium was sufficient to induce tumour formation. However, the same year, it was also reported that a loss of Aurora-A kinase activity in Drosophila neuroblasts led to tumour formation identifying this time Aurora-A as a tumour suppressor. This function of Aurora-A was also observed in mice. How does Aurora-A fulfil this dual function? This must be taken into account when using Aurora-A inhibitors in chemotherapies. Indeed, a large number of Aurora-A inhibitors has been identified since the discovery of the kinase, they show, as usual, variable specificities, and some of them are at present in clinical trials.

Biography

Claude Prigent is the Director of Microscopy- Rennes Imaging Center and Head of the Cell Cycle team at Research CNRS at Univ Rennes1, France. He is studying mechanisms that regulate different aspects of mitosis. He is particularly interested in the main mitotic structures, which are the centrosomes, the mitotic spindle and the chromosomes. For instance, he is asking questions like how centrosome maturation proceeds, how bipolar spindles assemble and how chromosomes condense. In parallel, he is also studying regulatory mechanisms such as cell cycle checkpoints, in relation with DNA repair and sister chromatid cohesion. He is currently focusing on late mitotic events, post metaphase stages. The approaches we use to unravel these mitosis mysteries mainly focus on post-translational modifications of proteins. In particular, they invest a lot of energy to uncover the role of phosphorylation, and ubiquitination, in the regulation of mitosis. His team is also deeply engaged in cancer research. Indeed, defects in cell cycle controls are frequently associated with human cancers, and the proteins that regulate cell cycle appeared to be excellent targets for drug design. Not only do they search for inhibitors of cell cycle controls but also they try to understand how misregulation of these control mechanisms participates to carcinogenesis.

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