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The Role of ROS in Cell Transformation and Cell Cycle Checkpoint Control

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One of the critical determinants of cellular response to exogenous stimuli is the cellular redox status. Intracellular generation of reactive oxygen species (ROS) is tightly regulated by the intrinsic anti-oxidant defense systems. Nevertheless, oxidative stress and damage that results from it has been implicated in a wide number of disease processes including autoimmune disorder, neuronal degeneration, and cancer. Experimental evidence have been accumulated over the last decade that ROS also play an important role as signaling molecules in diverse physiological processes. Indeed, low levels of intracellular ROS have been linked to cellular proliferation and cell cycle progression, which provides an explanation for the pro-oxidant state invariably associated with the transformed phenotype. Contrary to that are our recent observations implicating increase of intracellular ROS level controlling apoptotic cell death and cell cycle checkpoint signals delivered upon exposure to certain anti-cancer drug. Here we present our recent data on the role of ROS in oncogenic K-Ras-induced cell transformation and anticancer drug-induced mitotic checkpoint control.