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Myc-Driven Tumorigenesis and Cell Death

Guest Editor:

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Message from the Guest Editor

Dear Colleagues,Myc has a central role in almost every aspect of the oncogenic process, orchestrating proliferation, apoptosis, differentiation, and metabolism. The Myc family oncogene is deregulated in >50% of human cancers, and this deregulation is frequently associated with poor prognosis and unfavorable patient survival. Notably, Myc is frequently overexpressed in hematological malignancies due to gene amplification or translocation. Apoptosis is the main mechanism that counteracts cell proliferation driven by oncogenes, thus limiting cancer development. Myc is a unique oncogene which, alone, can potently induce cell death and carcinogenesis in transgenic animals. Of note, in an Emu-myc transgenic mouse model of B-lymphomagenesis, the pre-cancerous state is characterized by aberrant proliferation of B-lymphoid cells, which is initially offset by pro-apoptotic action of Myc. The resistance of pre-cancerous B cells to Myc-induced apoptosis must occur to proceed toward malignancy. Both basic and translational cancer research have greatly benefited from the use of suitable cellular and animal models, which have helped to characterise the molecular basis of My-driven cancers.

We invite investigators to contribute original research articles and review articles describing and discussing the molecular mechanisms at the basis of cooperative Myc-driven tumourigenesis, using basic and translational experimental models.

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Message from the Editorial Board

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