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Laboratory of Chromosomal Stability

DNA damage response, DNA repair mechanisms, genomic instability syndromes



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Research topics

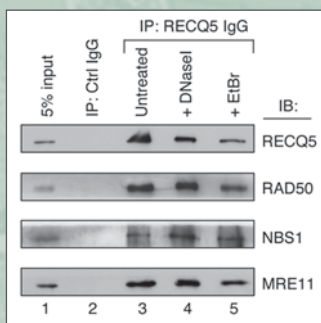
Cells are constantly under threat of the cytotoxic and mutagenic effects of DNA damage arising from DNA metabolism and following attack of endogenous and exogenous genotoxic agents. Failure to preserve genomic stability can lead to tumorigenesis. The research in our recently established laboratory is focused on understanding the mechanisms that control genomic stability in mammalian cells. Specifically, we study the role of human RecQ DNA helicases in DNA replication and DNA repair through analysis of their enzymatic activities and interactions with nuclear proteins. In humans, there are five RecQ homologues and deficiencies in three of them cause genetic disorders characterized by genomic instability, cancer predisposition, premature ageing and/or developmental abnormalities. Current studies in our laboratory address the function of the human RECQ4 protein, which is mutated in Rothmund-Thompson syndrome, a rare autosomal recessive disorder manifested by photosensitivity, skeletal abnormalities, aneuploidy, chromosomal rearrangements and predisposition to osteosarcomas. We also explore the cellular role of the RECQ5 helicase, whose deficiency in mice results in elevated level of homologous DNA recombination and increased incidence of cancer.

Current grant support

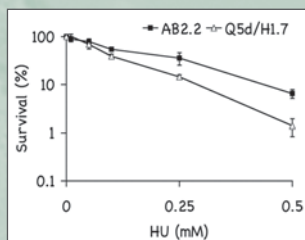
A start-up grant from the Institute of Molecular Genetics

Selected recent papers

A new group, so far no publications with IMG affiliation



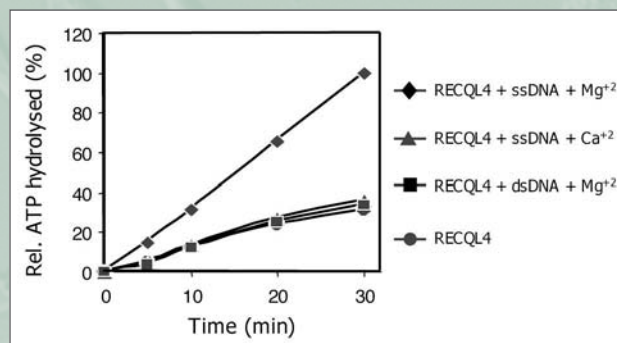
Co-immunoprecipitation of RECQ5 and the MRE11-RAD50-NBS1 complex from extract of human 293T cells



Hypersensitivity of RECQ5-deficient cells to hydroxyurea (HU) that inhibits DNA replication



Accumulation of the human RECQ5 protein at chromosomal breaks generated by laser-microirradiation



ATPase activity of purified human RECQL4 protein