MUTATION RESEARCH - FUNDAMENTAL AND MOLECULAR MECHANISMS OF MUTAGENESIS
A section of Mutation Research - only available as part of a subscription to Mutation Research/Full Set

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DESCRIPTION

Mutation Research provides a platform for publishing all aspects of DNA mutations and epimutations, from basic evolutionary aspects to translational applications in genetic and epigenetic diagnostics and therapy. Mutations are defined as all possible alterations in DNA sequence and sequence organization, from point mutations to genome structural variation, chromosomal aberrations and aneuploidy. Epimutations are defined as alterations in the epigenome, i.e., changes in DNA methylation, histone modification and small regulatory RNAs. We are interested in: mechanisms of (epi)mutation induction, for example, during DNA repair, replication or recombination; novel methods of (epi)mutation detection, with a focus on ultra-high-throughput sequencing; the landscape of somatic mutations and epimutations in cancer and aging; the role of de novo mutations in human disease and aging; mutations in population genomics; interactions between mutations and epimutations; the role of epimutations in chromatin structure and function; mitochondrial DNA mutations and their consequences in terms of human disease and aging; and novel ways to generate mutations and epimutations in cell lines and animal models. Of special interest are basic mechanisms through which DNA damage and mutations impact development and differentiation, stem cell biology and cell fate in general, including various forms of cell death and cellular senescence. The study of genome instability in human molecular epidemiology and in relation to complex phenotypes, such as human disease, is considered a growing area of importance.

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INTRODUCTION

Mutation Research: Fundamental and Molecular Mechanisms of Mutagenesis broadly encompasses all aspects of research that address the detection of mutations, the mechanisms by which mutations in genes and chromosomes arise, and the modulation of mutagenesis by mutation avoidance pathways such as DNA repair, cell cycle control and apoptosis. It includes the role of genetic variation in the genesis and manifestation of mutations, ranging from the variable manner in which xenobiotics are metabolized to variations in the capacity of cells to replicate and repair damaged DNA. It also includes the contributions of these mechanisms, when perturbed, to animal disease models and to human disease, with particular emphasis on carcinogenic mechanisms. The Journal will publish articles on the genesis of aneuploidy and isodisomy, including the roles played by recombination, cell cycle checkpoints, spindle microtubules, centrosomes and kinetocore proteins, and agents that might disrupt them. Submission of appropriate epidemiological studies as well as consequences, including methods for high throughput SNP detection, whole genome and exonic sequencing, DNA microarrays, RNAseq approaches and proteomics are welcome. Submission of preliminary epidemiological studies that associate SNPs with a phenotype but provide no mechanistic insight is discouraged. The broader scope of the journal is a reflection of the rapid advances in the field of mutation research and the recognition that understanding of the mutagenic process requires full knowledge of the cellular response to DNA damage including DNA repair, cell cycle checkpoint arrest and apoptosis.

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