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*Mutation Research (MR)* 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.

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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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