MUTATION RESEARCH - FUNDAMENTAL AND MOLECULAR MECHANISMS OF MUTAGENESIS
A section of Mutation Research

AUTHOR INFORMATION PACK

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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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**Carlos F. M. Menck**, Universidade de São Paulo (USP), São Paulo, Brazil
DNA repair, xeroderma pigmentosum, DNA damage, translesion synthesis, ultraviolet, gene therapy, autophagy, chemotherapy

**Sankar Mitra**, Houston Methodist Research Institute, Houston, Texas, USA
Oxidative damage in mammalian genome and its linkage to transcription and involvement of non-canonical factors, microhomology-mediated end-joining of DNA double-strand breaks in tumor cells

**Cristina Montagna**, Albert Einstein College of Medicine, Bronx, New York, USA
Genomic instability, Aneuploidy, Fluorescence in situ hybridization, Cancer, Aging, Mammary gland development, Breast tumorigenesis, Cervical cancer, Cell free DNA

**Lee Moore**, National Cancer Institute (NCI), Bethesda, Maryland, USA
Carcinogens, molecular epidemiology

**Gerd Pfeifer**, Van Andel Research Institute, Grand Rapids, Michigan, USA
DNA methylation, cancer mutations, 5-hydroxymethylcytosine, epigenetics, tobacco carcinogenesis

**Robert H. Schiestl**, University of California at Los Angeles (UCLA), Los Angeles, USA
DNA Repair, Carcinogenesis, Mutagenesis, intestinal microbiota

**John Schimenti**, Cornell University, Ithaca, New York, USA
Genetics; mouse; meiosis; DNA repair; reproduction; DNA replication

**Changshun Shao**, Shandong University, Shandong, China
DNA repair, genomic instability, oxidative stress, cell senescence

**Richard R. Sinden**, South Dakota School of Mines and Technology, Rapid City, South Dakota, USA
DNA conformations, G-quadruplex, genomic instability

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DNA Repair, base excision repair, DNA damage response, ADP-ribose, Poly-ADP-ribose polymerase, DNA polymerases, NAD+ metabolism

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Functional genomics of aging, genetics of age-relate disease, genetic and epigenetic variation, miRNAs, role of non-coding variants in human age-related traits, high-throughput genomic technologies

**Masako Suzuki**, Albert Einstein College of Medicine, Bronx, New York, USA
Epigenomics, DNA methylation, developmental origins of health and disease (DOHaD), assay development

**Joann Sweasy**, Yale University School of Medicine, New Haven, Connecticut, USA
Mutagenesis, autoimmunity, genomic instability, DNA damage, DNA repair

**Jay Tischfield**, Rutgers University, Piscataway, New Jersey, USA
Psychiatric genetics, addiction biology, gene regulation, induced pluripotent stem cell (iPSC) and engineered mouse genetic models

**Mitchell Turker**, Oregon Health and Science University (OHSU), Portland, Oregon, USA
Mutagenesis, DNA repair, ionizing radiation, epigenetic gene silencing, environmental epigenetics

**Marc Vermulst**, Children’s Hospital of Philadelphia, Philadelphia, Pennsylvania, USA
Mechanistic basis of aging, proteotoxic stress, stem cell biology, mitochondrial DNA dynamics, carcinogenesis

**Altaf Wani**, The Ohio State University, Columbus, Ohio, USA
DNA damage, Xeroderma pigmentosum, cell transformation, neoplastic, ovarian cancer, breast cancer, Li-Fraumeni syndrome

**Kandace Williams**, University of Toledo, Toledo, Ohio, USA
DNA damage and repair, mismatch repair, mammalian cell cycle, chemotherapy resistance, glioblastoma

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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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