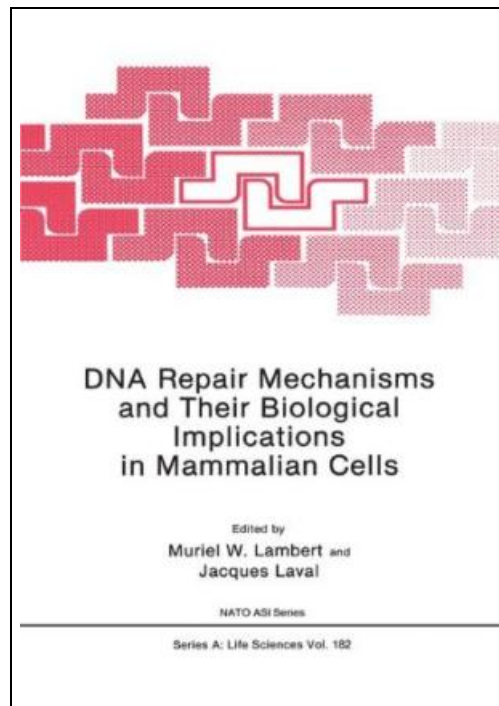


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(Matteo Johnson)

DNA REPAIR MECHANISMS AND THEIR BIOLOGICAL IMPLICATIONS IN MAMMALIAN CELLS



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Book Condition: New. Publisher/Verlag: Springer, Berlin | This volume contains edited contributions from the speakers at the NATO Advanced Research Workshop on "DNA Repair Mechanisms and Their Biological Implications in Mammalian Cells" held October 1-6, 1988, at the Abbaye Royale de Fontevraud, Fontevraud France. The meeting was dedicated to Paul Howard-Flanders (Yale University, New Haven, CT., 1919-1988), whose seminal contributions to the DNA repair field include the co-discovery of the excision repair pathway, the elucidation of post-replication repair in *E. coli*, the isolation of the *lexA* and *recC* mutants, and his extensive work on the enzymology of *RecA*. A plethora of recent developments in DNA repair mechanisms and related processes in mammalian cells have advanced our understanding of this field in a number of different areas and have given new emphasis to the ways these systems both resemble DNA repair processes in other groups of organisms in some respects yet are strikingly different from them in others. Within the past decade there have been a number of international conferences on DNA damage and repair mechanisms but none has been focused on these processes in mammalian cells. | Repair of Alkylation Damage in DNA.- Structure-Function Relationships in Alkylation Damage and Repair.- The Roles of Beta- and Delta-Elimination in the Repair of AP Sites in DNA.- Imidazole Ring-Opened Purines: Occurrence and Repair in *Escherichia coli* and Mammalian Cells.- Substrate Specificity of the Formamidopyrimidine-DNA Glycosylase of *E. coli*: Repair of the Imidazole Ring-Opened Form of N-Hydroxy-2-Amino-Fluorene-Guanine Adducts in DNA.- Investigation of Sequence Specificity in DNA Alkylation and Repair Using Oligodeoxynucleotide Substrates.- Towards a Role for Promutagenic Lesions in Carcinogenesis.- Acquisition of Resistance to Alkylating Agents by Expression of Methyltransferase Gene in Repair-Deficient Human Cells.- The Mer Minus Phenotype, Patient Response to Nitrosoureas, and Protooncogene Activation in Human Glioblastomas.- The...



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