

Overall this is a valuable publication, despite the fact that the title is merely a description of a common theme, rather than a clear focus for the organisation of a balanced collection of papers.

REFERENCES

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ROBERT BENSTED-SMITH

*Department of Pure and Applied Biology
Imperial College of Science and Technology*

Genetic consequences of nucleotide pool imbalance. Frederick J. de Serres (ed.). Plenum Press, New York and London. 1985. Pp. x+523. Price \$69.50 US.

Usually there are several good reasons for consigning a symposium-linked volume such as this one to the obscurity of a top shelf or worse. The reasons have to do with lax or non-existent editing, the time elapsed since the actual meeting, and the ephemeral data of many contributions.

However, it is difficult to level such criticisms at this particular volume which consists in the most part of a well-written series of chapters connected by a common theme and placed in wider perspective by contributions from Haynes, Mathews, Drake, Glickman and Holliday.

"Genetic Consequences of Nucleotide Pool Imbalance" arose out of a conference of the same name held in 1983. The theme proves to be an extremely unifying one, bringing together contributions from the fields of DNA precursor production, mechanisms of DNA synthesis and repair and the genetic consequences of pool imbalance in a variety of bacterial, fungal and mammalian cells. It is a general finding that disturbance to DNA precursor metabolism resulting in the presentation of an inappropriately balanced cocktail of deoxyribonucleotide triphosphates results in cell-killing and mutation. Much of the volume is preoccupied with showing that precursor imbalance can arise from the use of anti-metabolites, excess nucleosides or base analogues—all situations that have long been known to generate genetic instability. It is a pity, therefore, that recent data on reinitiation of *S* phase with consequent gene amplification following precursor synthesis inhibition by hydroxyurea post-dated the meeting. Such information strongly reinforces the view advanced in this book that precursor synthesis is intimately connected with the actual replication mechanism.

Precursor synthesis and the complex role of the key enzyme ribonucleotide reductase are reviewed extensively, and I found these topics especially useful since they underpin many of the other contributions. There are excellent chapters by Reichard, Mathews, and Melamed and Wallace, and, despite the still controversial and speculative nature of DNA replicase complexes and facilitated precursor channelling, these ideas will stimulate the search for useful mutants with which to dissect mechanism. One personal disappointment was the lack of any information on the possibility that there are distinct precursor pools for DNA repair. Unifying repair and replication demands has not been attempted in this volume except to register that the pool serving repair can be depleted by inhibitors (Snyder) and that pyrimidine auxotrophs of *Ustilago* are UV sensitive and have difficulty in completing repair (Holliday).

DNA repair is, of course, likely to make far fewer demands on precursor production than replication and several excellent contributions on the thymineless state from bacteria to mammalian cells reinforce how important it is to maintain an adequate pyrimidine pool. Failure to do so results in DNA degradation, chromosome aberrations, cell-killing, and a wide range of genetic changes. The stressed situation is very complex, however, and the excellent chapter by Little on yeast corrects the impression that the thymineless state is entirely understood. Nevertheless the analysis of useful yeast and mammalian cell mutants promises to open up the field. My experience is with mammalian cells and I particularly appreciated the mutant analysis of Seno and of Meuth and their colleagues which reveals the enormous and complex genetic instability associated with mutations in the DNA precursor pathway.

More than anything else this work should help us to understand that mutation is the result of an extremely complex series of cellular perturbations and that it is not enough to consider simply DNA damage and repair capabilities. Now it is essential to realise that precursor pools can shift dramatically after exposure to genotoxic agents resulting in reduced replication fidelity and increased possibilities of mutation.

This is an enjoyable, thought-provoking and useful volume. Inevitably there is unevenness among the contributions but the general standard is good and most authors have removed blinkers and allowed themselves to relate their work to wider issues. There are several good perspective chapters, that of Haynes being especially helpful. Good meetings do not invariably generate good books. This one has and it should be made widely available to provide a glimpse into a complex and fascinating field.

R. T. JOHNSON

*Cancer Research Campaign Mammalian Cell DNA
Repair Group
Department of Zoology
Cambridge University*