mended reviews as a basis for essays for final year honours students.

On a point of nomenclature I think that there could be some confusion in the way that words having had a specific meaning in molecular genetics for many years are used in quite a different way in this book. Heteroduplex analysis is a standard method for studying the degree of dissimilarity between DNA molecules in which single strands of related DNA are annealed and the DNA:DNA heteroduplexes examined in the electron microscope for non-homologous single-stranded regions. But here the word heteroduplex is used to refer specifically to a DNA:RNA complex to distinguish it from a DNA:DNA homoduplex. Likewise the word transfection which is used to describe the uptake into a cell of the naked DNA of a viral genome is used here to describe what traditionally is called transformation. Teaching genetics is difficult enough without having to cope with shifting definitions. These criticisms apart there is an awful lot of information in this book and I am glad to have a copy.

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Editor's Note
There are two reviews of John Hawkins' book, "Gene Structure and Expression". Dr. Moseley was asked to review the book for "Research and Development in Agriculture", but the editor felt that the subject was not really of interest to the readership of this journal. The review was, therefore, passed on to *Heredity*.

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 antimetabolites, 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 Melamede 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 reveal 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.

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Current developments in anthropological genetics. Vol. 3 Black Caribs. A case study in biocultural adaptation. M. H. Crawford (ed.). Plenum Press, New York. 1984. Pp. xvii+395. Price $59.50 US.

This is the third volume in this series, the first two volumes dealt with theory and methods, and ecology and population structure respectively. This volume differs from its predecessors in that it provides an in-depth survey of one particular group, the Black Carib peoples. The Black Caribs (Garifuna) were originally the products of intermixture between runaway West African slaves and Carib-Arawak Indians of the eastern Caribbean. The Black Caribs are to be found on St. Vincent Island and in Central America around the Bay of Honduras. They have an interesting history (see chapters 1 to 4) which needs to be understood for the reader to fully appreciate their evolution.

In brief, there are three main Carib groups; the South American Caribs who live north of the Amazon; the Island Caribs—those who migrated from the mainland initially onto the Lesser Antilles. After 200 or so years of European depredations they were confined to Dominica and St. Vincent. After a series of wars with the British, the Carib population was deported to Roatan Island, Honduras.

One hundred or fewer managed to avoid capture and their descendants constitute the contemporary Black Carib population on St Vincent. The 2500 Caribs deported to Roatan Island rapidly emigrated to the Central American mainland. The coast of Honduras already contained some Creole settlements, thus there was genetic admixture. The Black Caribs have now spread along the coast and today live in 52 villages and number about 70,000.

The book is divided into three parts, sociocultural and demographic (chapters 1 through 7); morphological (chapters 8 to 13); and genetic aspects (chapters 14 to 20). This review is concerned mainly with part III. The genetic studies undertaken by Professor Crawford and his colleagues have been designed to determine the extent of admixture and genetic microdifferentiation of Black Carib communities over a relatively short geographical separation. The analyses reported in chapters 14 to 17 cover these aspects in detail. Evidence from blood group, haemoglobin and enzyme markers suggests contributions of about 75·5 per cent African, 24 per cent American Indian and 0·5 per cent European ancestry. The study of immunoglobulin phenotypes confirms these admixture figures as well as throwing light on the extent of the St Vincent–Honduran similarities and differences. To summarise, Black Caribs of Belize have more African—and St Vincent more American—haplotypes. The *Gm* haplotypes permit some reconstruction of the past population structure and it would seem that the majority of the components were present in the founding populations prior to deportation from St Vincent. The St Vincent populations are genetically more heterogeneous due primarily to variation in amounts of admixture among local subpopulations and the isolation of communities from one another.

The other area of specific genetic interest relates to abnormal haemoglobins. The Black Caribs have remained until quite recently under heavy malarial pressure both from *Plasmodium vivax* and *Plasmodium falciparum*. Sickle-cell trait heterozygote carriers have been shown to be more resistant to malarial infestation than normal homozygotes and in particular, children who are carriers appear resistant to cerebral malaria. Work by Firschein in Belize published in 1961 revealed a very high sickle-cell carrier rate and he suggested that the haemoglobin polymorphism is maintained by differential fertility rather than by differential mortality. The hypothesis was retested by Custodio and Huntsman (chapter 17) but unfortunately their data are not conclusive.

This is a wide ranging and full study although there are clearly some topics awaiting future research (see chapter 19). Even so an excellent book—highly recommended.

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Aetiological studies of isolated common congenital abnormalities in Hungary. A. Czeizel and G. Tusnády. Akadémiai Kiadó, Budapest. 1984. Pp. xv+359. Price unspecified.

Congenital malformations are now the major cause of anxiety for prospective parents, and provide a substantial proportion of the deaths and disabilities in infancy and childhood. In most of the common forms, surgery has a major contribution to offer, varying from cure with a minor scar in pyloric stenosis to the replacing of natural death by unnatural life in severe spina bifida.

For reasons which are not clear, but probably go no deeper than the semantic confusion of congenital and genetic, geneticists have taken a greater interest in common fetal disease than in common disorders arising after birth, yet malformations receive priority among the consequences to be expected from any increase in the mutation rate. Both the BEIR and UNSCEAR reports allow this group of disorders pride of place, virtually ignoring recessive disorders, which might seem the natural leaders of this host of death and destruction. If UNSCEAR were