serious problems of our ageing population. At one time it was considered a form of presenile dementia confined to the fifth and sixth decades of life. But it is now becoming clear that this, the commonest form of dementia, may begin more often after the age of 60. Much has been written about this fascinating disorder and this current text, the edited proceedings of a meeting held in Paris in 1988, summarizes many of the recent findings.

There is no doubt that the disorder is often familial but genetic studies are difficult because of the late age at onset and difficulties in clearly defining the disease on the basis of clinical criteria. Furthermore, there is increasing evidence of heterogeneity with earlier onset in some families and later onset in others. Also relatives of individuals with what is referred to as the AAAA syndrome (Amnesia, Aphasia, Apraxia and Agnosia) have a higher risk of becoming demented than relatives of cases with pure dementia. Dermatoglyphic abnormalities have been found more commonly in some affected families than others. But undoubtedly the most exciting findings have been in regard to linkage studies and the relationship to Down's syndrome.

Individuals with Down's syndrome who survive to middle age often develop an Alzheimer-like disorder and autopsy studies have revealed neuropathological changes in the brain similar to AD. In particular there is an accumulation of amyloid A4 protein (beta protein). The gene locus for this protein has been localised to chromosome 21 and it was therefore tempting to believe that this might also prove to be the locus for AD. Linkage studies have now shown that the gene for early onset AD (mean age of onset less than 60) is located on chromosome 21, but not at the locus for amyloid protein. The AD gene locus is *not* identical with the amyloid locus. Furthermore, familial late onset AD (mean age of onset greater than 60), which is a more common disorder, may not be located on chromosome 21 [but see *Lancet* 1, 352–354 (1989)]. AD would therefore appear to be genetically heterogeneous.

In many families where the disease affects individuals in several generations, the disease does not always appear to be fully penetrant. Sometimes this may be because a gene carrier died from some other cause at an age before AD would have become manifest. But it also seems very likely that some, as yet unrecognized, environmental factors are involved.

The final part of the book is devoted to several papers which examine the nature, molecular structure and function of amyloid protein. It is already becoming clear that studies along these lines may well help us to understand more of the pathogenesis of this tragic disease which now affects around 1% of the population.

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The Use of Plant Genetic Resources. Edited by A. H. D. BROWN, D. R. MARSHALL, O. H. FRANKEL and J. T. WILLIAMS. International Board for Plant Genetic Resources: Cambridge University Press, Cambridge, UK. 1989. 382 pages. Paperback, $17.95 ISBN 0 521 36886 3; Hardback, $49.50 ISBN 0 521 34584 7.

This book is the result of a workshop convened by the International Board for Plant Genetic Resources (IBPGR) in 1986, with the intention of exploring how the use of collections, primarily of seed samples from designated source populations, is limited or can be facilitated. The 22 papers included are grouped into 6 sections: the first two sections describe the uses to which collections have been put, the third discusses the size and design of collections, the fourth discusses some factors and principles for evaluating them, the fifth considers the special problems of creating collections of wild relatives of domesticated species, and the last reviews new technologies that may affect the utility of collections

A persistent theme is that with notable exceptions, breeders have not used the collections very extensively despite increased efforts over the past 20 years. Large samples are now available in seed banks and other storehouses, but their genes are not often seen in breeding populations or in released varieties of the major crop species and their relatives. If the lack of use is due to a longer than expected lag time in varietal development, or to inefficiencies in disseminating seeds or information, then merely increasing breeding efficiency will solve the problem. However, as suggested by Frankel in Ch. 15, '... the prevailing strategy for evaluation has not been altogether successful. And the proposed cure, more of the same, scarcely inspires confidence.' Apparently, breeders see the bottleneck to the potential outpouring of benefits from these collections as a lack of evaluation and other prebreeding activities. While this problem may hardly seem of interest to geneticists other than plant breeders, one wonders whether there is, in fact, a genetical problem. If genotypes or populations can be characterized by standard descriptors, and standard tests can be used to analyze performance, then evaluation problems only involve efficient screening through large sets of materials. In this vein, methods are discussed for sampling and reducing collections to small 'core' sets for maximum differentiation and for screening entries by correlated measures either of their source environment, or of detectable linked loci. Further, it is asserted in the last chapters that many gene actions have major and direct effects on traits of economic importance that are consistent when transferred among species even across different kingdoms. Therefore, evaluation should be only a matter of more detailed gene cataloging.

Obviously however, evaluation is not easily reducible to observations of genes or of environmental

descriptors. Frankel (ch. 15), Burdon and Jarosz (ch. 17), Ladzinsky (ch. 18), and P. H. Williams (ch. 20) point out that performance and potential breeding value usually depend on multigenic effects in the genetic background, requiring prebreeding as well as simple evaluation of accessions. Evaluation is as much a question of physiology, pathology, entomology, and ecology as it is of understanding single gene functions. Since the utility of most traits sought by breeders depends on interactions within and among organisms, the level of organization at which gene effects must be studied is higher than the single gene. Especially for traits involved in biotic resistances where breeders may seek to influence the coevolution of host and pathogen, evaluation is not reducible to even the individual level. Therefore, by identifying evaluation as the major problem in the use of collections, plant breeders are implying that various forms of genotype–environment interactions and interorganism effects are important to understand and that molecular level reductionism is not sufficient for their needs. Examination of these interactions is an area in which breeders apparently need more genetical research.

This book is a valuable overview of the current uses to which collections of plant materials of the major crops can be put. Details in individual species are not provided, but opportunities and problems in the utility of collections are identified. Uses and structure of collections and problems in evaluation are constructive indicators of the next steps to be taken in more fully utilizing conserved plant genetic resources.

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Introduction to Quantitative Genetics, by D. S. FALCONER. Harlow, Essex: Longman Group UK, 1989. 438 pages. $14.95 soft cover. ISBN 0 582 016428. Also published in the United States by John Wiley & Sons, New York, as ISBN 0 470 21162 8.

This is the third edition of a well-known and long-running textbook, first published in 1960, revised in 1980 and now again in 1989. Quantitative Genetics, as most of us know, stems from the work of Fisher, Haldane and Wright in 1918–1921, and is concerned with the genetics of biometrical or quantitative characters, which form the main raw material of plant and animal breeding research and are also of primary importance in evolutionary theory. Many genes, of generally small effect which is overlaid by environmental variations during development, contribute to the end points forming these characters, so that the individual genes cannot be identified and mathematical analysis of means, variances, selection progress, etc. has had to replace the Mendelian genetic approach. This all makes it difficult for new students in this research area to find their way.

In the late 1950s Douglas Falconer, already heavily involved in long-term experiments on quantitative characters in mice and with colleagues working on similar problems in *Drosophila*, decided to set out his lectures to genetic students on quantitative genetics in book form for a wider audience. Taking a modest view of his own mathematical ability and that of his readers, he restricted himself to fairly simple algebra and standard statistical concepts, and the result was the first edition of the book under review. Its organization was so well chosen that it was possible for the two revised editions to retain the same structure down to the 20 chapter headings and most of their subheadings; references, paragraphs and sections have been added where they seemed appropriate.

Rereading the book in its new edition, I am struck by the clarity of the writing and the logical arrangement and interlinking of the topics, which make it a pleasure to read. The essence of the subtle mathematical concepts developed to relate the conjectural dances of the polygenes in their environmental soup to the characters which can be measured are very well explained; and the new reader should not have too much difficulty with coefficients of co-ancestry and inbreeding, effective population number, linkage disequilibrium (which, confusingly, applies to loci that are not linked), mutation rates affecting quantitative characters, analysis of threshold characters, and so on.

The main changes in this edition are 3 pages added on the effects of selection on variance in chapter 11, a brief discussion of the actual achievements of selection applied to farm animals in chapter 13, a revised and extended discussion of the effects of mutation in chapter 15, additions to the discussion of threshold characters in chapter 18 and a section on the origin of variation by mutation in chapter 20. Many up-to-date references have also been included.

After the second edition a set of problems was compiled and published separately, but I doubt whether many readers got to work on them. The new edition has these problems, probably with some new ones added, in their proper places at the end of each chapter. These 140 problems are often ingeniously designed to be of varying difficulty and their solutions are very amply explained at the end of the book so that they take up 56 pages. These seem to me excellent value and I trust that every reader will test his skill on them.

I think this book will have many years of useful life yet, in its new edition, particularly as the subject is acquiring new interest from the attempts of gene technologists to identify individual polygenes on the chromosome and gather them together to make much improved strains. We shall wait impatiently to learn how many of these genes have large enough effects for them to be transportable.

A point in the book which particularly caught my attention is Figure 15.1 on page 266. This shows 20