Introduction to Molecular Embryology Second, totally revised and enlarged edition. By J. BRACHET and H. ALEXANDRE. Berlin, Heidelberg, New York, Tokyo: Springer-Verlag, 1986. 229 pages. Soft cover DM 39, ISBN 3 540 16968 7.

This book presents an up-to-date account of how the techniques of molecular biology are being applied to the complex problems of embryology. The discussion follows the natural order of events from gametogenesis to the early stages of embryonic development and differentiation, with due attention to diverse aspects of nuclear cytoplasmic interaction. The comparative approach addresses the classic problems of embryology, drawing on evidence from *Acetabularia*, mammals, amphibia, echninoderms and other invertebrates, although *Drosophila* is disposed of rather briefly. The authors are eager to develop the implications of the flood of new experimental evidence but they do not conceal how much of the mystery remains, sufficient to tax the ingenuity and imagination of experimental biologists for the foreseeable future.

The senior author (J.B.) has been active in the game for a long time, since the introduction of Dische's diphenylamine method of estimating DNA and the time when RNA was thought to be confined to plant cells, and has lived through a remarkable transformation of his subject, never faster than since the first edition of this work about a decade ago. This personal involvement imparts a unity and coherence to the account, so often lacking in research reviews. There is also a sense of historical perspective since the early workers are not forgotten. It is worth being reminded how far the theoretical views of say T. H. Morgan on embryonic differentiation have been vindicated half a century later.

The style of presentation is succinct and readable, accessible to the non-specialist as well as the student. The illustrations are clear. Although the reference list is very modest the recommendations for further reading at the end of each chapter compensate well enough. This is certainly a book to add to the library list.

FORBES W. ROBERTSON
41 *Braid Farm Road*
Edinburgh

Developmental Mutants in Higher Plants. Edited by HOWARD THOMAS and DONALD GRIERSON. [Society for Experimental Biology Seminar Series No. 32. Cambridge University Press, 1987. 287 pages £25.00 ($39.50), ISBN 0 521 32844 6.

The thirteen papers in this symposium deal with different aspects of plant development, which have been revealed by the study of mutants. The number of mutants already known in both crop and ornamental species is very large. They span a wide range of effects on plant morphology, physiology and biochemistry. As the editors note, by analogy with animals and micro-organisms, there is immense potential scope for integrating the techniques and concepts of the different disciplines in unravelling the effects of mutant genes. Given the role of selection for mutants in plant domestication and their relevance to taxonomic distinctions, such information will also add a further dimension to evolutionary interpretations and will often prove directly relevant to agricultural or horticultural practice. So, on all counts, the publication of the present volume is timely.

The stated intention was not to aim at a comprehensive survey but rather to direct attention to general biological principles and also the respects in which plant development differs intrinsically from that of animals. It is no surprise that the level of analytical sophistication varies widely, according to the aspect of development or physiology chosen for comment, ranging from basic description of the phenotypic effects of mutant genes to application of the latest molecular techniques. The style of presentation also varies. Some of the papers carry a lot of technical information, others are cast in a more theoretical mould. Perhaps the best way to indicate the scope and variety of the contributions is to summarize briefly the main themes.

Gottlieb and Ford advocate an integrated developmental and genetic study of discrete, morphological differences of taxonomic significance, to determine their often simply inherited primary developmental causes. This approach is illustrated by their investigation of the differences between two closely related, annual, diploid Californian composites, *Layia discoidea* and *L. glandulosa*. The more widespread putatively ancestral *L. glandulosa* has conspicuous ray florets which *L. discoida* lacks. There are also other floral differences in the involucral bracts, disc floret number and pappus size. Electrophoretic comparisons indicate a high degree of genetic similarity between the species, which are fully interfertile. The genetic analysis and developmental comparisons are presented in some detail. Two loci are involved in the determination of presence or absence of ray florets, although additional genetic differences affect floret number and size. On present evidence the authors favour a model in which a difference in the timing of growth of the peripheral florets, relative to their bracts, determines whether or not they become accessible to ray inducing signals.

Martin, Carpenter, Coen and Gerats present anthocyanin synthesis as a relevant model for understanding morphogenetic regulation. This theme is developed in comparisons of mutations in *Antirrhinum*-which affect the steps from the intervention of chalcone synthase to the formation of the anthocyanidin 3- rutinosides, a sequence which includes at least seven enzymes. Transposable element insertion at two loci, coding for either chalcone synthase or an, as yet unknown, enzyme are proving valuable sources of informative

mutants. Under laboratory conditions, variation in pigment biosynthesis appears not to affect survival, so the authors think it may be possible to recover a sufficient variety of mutants to elucidate fully the course of biosynthesis. They discuss the regulation of anthocyanin synthesis both by environmental agents, such as temperature and light, and by genetic mechanisms, especially cis- acting control systems. Insertion of a 3·5 kb element in the promoter region of the chalcone synthase locus allows partial gene expression. Excision is accompanied by loss of small segments of the promoter sequence and correlated pattern differences in the distribution of pigment in the corolla, suggesting that promoter sequence regions may contribute to the control of spatial differences in gene expression. Trans-acting regulation is discussed in relation to mutants in which pigment intensity is altered or pigment is limited to particular parts of the corolla. Evidently the story of plant pigmentation, which started at such a cracking pace in the early days of genetics, is unfolding into a tale of control mechanisms in a molecular context.

Bailey-Serres, Isaac, Small and Leaver are concerned with the properties and functions of the mitochondrial genome and have chosen cytoplasmic male sterility (CMS) as a relevent system. Aberrant intra- and/or inter- molecular recombination in mitochondrial DNA is regarded as a likely origin of CMS. Given the presence of a promoter and a transcriptional control sequence, the consequent chimeric sequences could code for a novel polypeptide, biosynthesis of which might have a variety of consequences. But, at microsporogenesis, a stage of increased production of mitochondria and rates of respiration, its difference could become crucial and lead to the characteristic mitochondrial degeneration. Restorer genes, introduced from an out-cross, could either suppress biosynthesis of the aberrant polypeptide or make good its deficiency. Circumstantial evidence in favour of this scenario refers to rearrangement within or adjacent to the cytochrome *C* oxidase subunit 1 gene in the mitochondrial genome of maize and sorghum. Although these particular sequence alterations apparently are without effect on either cytochrome oxidase activity or seedling growth, they provide a model for the possible origin of CMS.

Grierson, Purton, Knapp and Bathgate consider the genetic control of ripening in the fruit of tomato, a phase of development adapted to secure seed dispersal by making the fruit both attractive and palatable. Constituent processes include an increase in ethylene synthesis and in respiration, solubilization of starch, the conversion of chloroplasts into chromoplasts, with loss of the machinery of photosynthesis, modification of the pectins and cellulose in cells walls, accumulation of sugars etc., accompanied by changes in the make-up of the mRNA population. Many mutant genes are known which apparently affect only the ripening phase by inhibiting or altering one or more of the ripening processes. Current research is focused on the changes in composition of the mRNA during ripening to identify the primary differences between wild-type and the mutants. Particularly relevant is the control of synthesis of ethylene, which plays a key role in ripening, and, especially, the control of activity of two enzymes which are of prime importance in the autocatalytic production of ethylene. The authors consider the possible effects of mutant genes and present a model for the regulation of gene expression during ripening.

Shewry, Williamson and Kreis are concerned with the biosynthesis of prolamins (alcohol-soluble storage proteins), which, together with starch, are the major storage components of cereal endosperm. Apart from general quantitative and qualitative variation, the nutritional quality of major cereals, other than oats and rice, is limited for man and monogastric livestock by low levels of particular essential amino-acids, such as lysine and threonine in barley and lysine and tryptophan in maize. Mutant genes which influence the synthesis of starch and prolamins are known in barley, sorghum, rice and, especially, maize, but particular attention is here devoted to the mutant genes which increase the lysine content of barley endosperm. Barley prolamins comprise a complex mixture of 20–40 polypeptides which can be classified into three groups, distinguished respectively as high molecular weight (HMW), S-rich or S-poor. Each group is controlled by co-dominant alleles at single, complex, linked loci. The S-rich and S-poor loci are highly polymorphic with 20–30 gene copies per haploid genome, while the HMW category shows little polymorphism and has only a low copy number. Sequence comparisons point to a common ancestral gene. In barley a generally inverse relation between higher lysine content and reduced prolamin and starch content has proved a recurring feature of high lysine mutants. The authors compare the effects of different mutant genes, the amino-acid composition of the different proteins, evidence for genetic regulation and also the evidence from mRNA composition and restriction enzyme analysis, that an induced deletion, at least 85 kb long, at the S-rich locus, is associated with some 17% increase in lysine, smaller and fewer seeds, substantial reduction in prolamins and relative increase in other proteins.

Karssen, Groot and Koorneef review the control of seed dormancy and gravitropism, especially in *Arabidopsis thaliana* and the tomato. Mutant genes which influence the availability and/or concentration of gibberellins (GAs) or abscisic acid (ABA) are helping to clarify a rather confusing situation in which environmental factors, such as light and lower temperature, alter the sensitivity of seeds to GA. In *Arabidopsis* and tomato GA-deficient mutants require an application of GA for seed germination, while the dwarf growth of the seedlings can be corrected by GA sprays. One of the *Arabidopsis* mutants is known to

have greatly reduced capacity to synthesize *ent*-kaurene while, in the tomato, the GA pathway is blocked before *ent*-kaurene in one mutant and between *ent*-kaurene and GA_{12} in another. In addition to dwarf growth and lack of seed germination, GA-deficient mutants of *Arabidopsis* and tomato show reduced apical dominance, dark green leaves and abnormal flower development in which sepals and pistil are normal but petals and stamens are defective. ABA-deficient mutants have been detected by acute wilting because stomata fail to close in response to water stress, although this behaviour can be reversed by ABA sprays. ABA mutants display reduced seed dormancy. Mutants are also known which phenotypically resemble GA- or ABA-deficient mutants but which either fail to respond to sprays of GAs or ABA or require much higher concentrations. Evidence that the absolute requirement for induction of dormancy depends on the presence of ABA in the embryo and endosperm, independent of presence or absence of maternal ABA, rather calls in question the use of the term hormone in this context. The stage is now set for the application of molecular methods of analysis to the control of GA and ABA function.

Roberts presents a review of mutant genes which intervene in the co-ordinated response to gravity which includes the perception of gravity, transduction of the stimulus and growth reponse. Gravitropic mutants are described for *Pisum sativum*, *Arabidopsis thaliana*, maize, tomato and barley. Perception is believed to reside in the behaviour of sedimenting starch grains (amyloplasts) and their interaction with the endoplasmic reticulum. Comparative study of mutants in different species has associated impaired response to gravity with absence of amyloplasts, reduction in their size or their failure to sediment normally. But that is only part of the story. In one mutant of *Arabidopsis* the root, but not the shoots, are agravitropic while, in another, in early seedlings neither roots nor shoots respond. Maize mutants have provided evidence of growth inhibiting compounds in the root extension zone, while, in some maize cultivars, when grown in the dark the roots do not respond to gravity but do so after a brief light treatment. Light-sensitive protein synthesis as well as indole-acetic acid (IAA) are implicated. ABA is believed to play an important role by some workers although interpretation may have to take account of its concentration in particular cellular compartments of the root. The author emphasizes the need to separate events necessary for the induction of gravitropic sensitivity from those necessary for gravitropism to take place and rounds off the account with a model for hypocotyl bending, in which sedimenting amyloplasts act as non-specific barriers to compounds which regulate plant growth.

Stoddart deals with the genetic control of plant stature, especially in crop species. Plant height is determined by the number and/or extension rate of sub-apical internodes and can be altered in either the division or elongation phase of the cell cycle. Control ranges from events at single sites to complex interactions and generalizations are as yet hard to come by. The genetic control of the dwarf and semi-dwarf habit is compared with that of overgrowth in the so-called 'slender' mutants. In *Pisum* a number of interacting loci are involved, which either affect the extension rate of stem internodes throughout the growth cycle or only at particular stages, or influence internode number. The genetic intervention is in the GA pathway in the sub-apical region. In maize recessive genes at several loci can independently cause dwarfing. The GA pathway is implicated, with GA as the only active endogenous gibberellin. In wheat, interaction between dominant genes at three loci determines very short internodes and hence a dwarf plant with a perennial grass-like habit. Several loci are also known at which dominant alleles control the 'semi-dwarf' condition, which has been so important in agriculture. Unlike normal cultivars, seedlings of these genotypes are insensitive to GA treatment. In single recessive dwarfs in other species like rice, common bean, *Lolium* spp. and watermelon the dwarf condition is reversible by GA treatment. The overgrowth phenotype in *Pisum* is determined by several interacting loci. In barley an interesting mutant gene increases growth rate and also extends the lower limit of growth at low temperature by 10 °C. The reactions of pea and barley mutants to GA inhibitors and to environmental differences are compared. The author attaches significance to possible alteration in the chain of reactions after the primary sub-cellular reaction with GA and leaves the reader in little doubt that the study of genetically controlled over-growth is crucially important for a general understanding of the control of plant stature.

Cove and Knight are alone in not dealing with angiosperms but with reactions to gravity and light in the moss *Physcomitrella patens*, in which auxins and cytokinin have a role in the regulation of development and phytochrome is involved in the reactions to light. In the apical cells of primary chloronemata derived from germinating spores, at low light intensity filaments show positive phototropism or orient perpendicular to the electrical vector of polarized light. At high intensities the filaments grow perpendicular to the direction of incident light and orient parallel to the E vector. At least two loci are involved in the origin of mutants with an abnormal light response, but which have not lost the high/low intensity reaction, although the light intensity required to switch the response is lower than in wild type. The authors compare the properties of the apparently heterogeneous mutants and also comment on the interaction between gravitropism and phototropism, which suggests that light may not merely override the gravity response but actively inhibit it.

Taylor presents a cogent interpretation of ABA

synthesis in the tomato, with particular reference to three ABA-deficient mutants, with a 'wilting phenotype', which can be ranked in order of decreasing concentration of basal, endogenous ABA. The treble and double homozygous combinations extend the range of ABA deficiency, including controls, to eight genotypes responsible for different ABA concentrations, thus providing excellent material for examining the function of ABA in the regulation of plant growth and development, and also for elucidating the rather uncertain pathway of ABA biosynthesis and the control of accumulation. The two disputed routes of ABA synthesis are considered, namely the direct route via oxidative steps after cyclization of a C15 terpenoid precursor, farnesyl pyrophosphate, as in fungi which synthesize ABA, and the alternative route in which ABA is formed indirectly by oxidative cleavage of a larger C40 precursor, such as violaxanthin. The argument goes in favour of the indirect route, with emphasis on the nature of by-product formation and the measurement of by-product concentration which can shed light on rates of change of the C40 precursor. The author discusses the consequences of different, genetically determined lesions in the ABA pathway in the context of the indirect route.

A large number of mutant genes have been isolated in *Pisum*. Hedley and Wang review the main features of those which affect seed and foliar development. The normal, compound, pinnate leaf comprises leaflets, tendrils and 1/2 sessile stipules. Different mutant genes convert leaflets into tendrils and *vice versa*, reduce stipule size, transform the leaflets into a single leaf, separate the epidermal and palisade cell layers, alter the amount of wax on the leaf surface, alter chlorophyll content and hence leaf colour etc. Some of these, separately or in combination, have proved commercially valuable and novel combinations of mutant genes to meet changing demand can be anticipated. About 50 genes have been associated with variation in seed characters. Most refer to the maternally derived testa and only a few have been shown to affect embryo development although the true number may be greater if differences in seed shape, surface impressions etc. have an embryonic component. The familiar, wrinkled condition, due to homozygosity of the *r* allele at the *rugosus* locus, appeared as a mutant probably about 1600, was incorporated into commercial varieties in the 18th century by Knight, and, as we all know, was put to good use by Mendel. The wrinkled phenotype is part of a syndrome of effects which include smaller, fissured starch grains, about twice as much sugar in the seed as in the round seeded form, lower starch content of different composition, higher water content of developing embryos etc. The wealth of *Pisum* mutants which are distinguished by differences in the leaves, seed and other characters, offers promising material for research into the genetic control of development and morphology. With a few exceptions, such as Murfet's study of flowering and internode length, this mine of information awaits exploitation.

Thomas develops the case for senescence as a programmed, developmental process rather than a function of macromolecular obsolescence, and discusses the effects of mutants in maize, wheat, soybean and *Festuca*, which alter senescence rates. In many crops leaf expansion, senescence and duration of leaf area are major determinants of yield. The onset of senescence marks the functional transition from assimilation to redistribution, especially of nitrogen. In maize, the complex, heritable delay in senescence, known at 'Stay-green', is associated with high yield. Wheat displays polygenic variation in grain protein which is correlated with senescence rate in some cultivars. In soybeans mutant genes at several loci have been shown to influence senescence, evident in delayed or premature leaf yellowing. In one analysed case, homozygosity at four loci is required for expression of delayed senescence, which is also very susceptible to environmental variation. A remarkable, single locus mutant of *Festuca pratensis* is described in which chlorophyll is retained in circumstances in which degradation would normally occur, such as water stress, treatment with ABA and even infection by pathogens. Although mutant and normal plants share many features of senescence, they especially differ in the persistence in the mutant of components of the hydrophobic core of thylakoid membranes. Although the origin of the pleiotropic effects of the mutant gene is unknown the author favours an impairment of glycolipid turnover.

Horgan rounds off the contributions with an account of the ability of certain tissue cultures to grow on defined medium, without the need for added cytokinins and/or auxins. Such autonomous behaviour can be classified into three categories, according to whether the tissues are derived from either crown galls, or the tumours which are a feature of certain interspecific hybrids in some genera or from cell cultures in which the exogenous cytokinin and/or auxin requirement was either absent from the start or gradually acquired during culture. Only for the crown gall situation are the biochemical and genetic causes reasonably well understood. Particularly significant is the evidence for two T-DNA loci, *tms* and *tmr*, which affect tumour morphology. The consequences of insertional inactivation of either *tms* or *tmr* differ in the promotion of respectively either leafy shoots or roots, accompanied by correlated increase or decrease in the cytokinin/auxin ratio. The author considers the significance of such evidence in the context of currect ideas in this rapidly advancing field. In the other categories the origins of the autonomous growth of tissue cultures is quite obscure. In the second category, the nuclear endowment of the parent apparently regulates tumour induction in some species and tumour expression in others and only crosses between