A Reconsideration of Plant Teratology

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The significance to be attached to teratological phenomena in morphological and phylogenetical studies of flowering plants has long been a matter of debate. On the one hand there have been those who have regarded such phenomena as largely irrelevant, and not to be taken as necessarily providing concrete evidence in interpreting homologies of organs (Goebel 1897, 1928; Arber 1931; Bertrand 1947), while on the other many authors have unhesitatingly accepted teratological forms as often atavistic (Worsdell 1915, 1916; Hagerup 1938) and have even gone so far as to found theories of angiosperm flower structure — initially, at least — upon such evidence (Saunders 1931 etc.). Evidently, while we constrain ourselves to a consideration of morphology alone, there can be no reconciliation of these points of view. The interpretation of teratisms remains a matter of opinion, and if certain of them are accepted as reversionary and used as a basis for theories of homology and phylogeny, then such theories will always be regarded with scepticism by those to whom the premises are not acceptable.

A glance at any of the teratological manuals (of which the best known are those of Moquin-Tandon 1841, Masters 1869, Penzig 1921/23 and Worsdell 1915/16) shows that many types of structural anomaly have been listed as teratisms, including relatively minor departures from the presumed normal forms of species in numbers, positions and shapes of organs. Frequently such minor aberrations have no obvious effect on function, and indeed may be regarded as no more than extremes of the normal range of variation. As Goebel 1897 has remarked "We cannot say where a normal structure ends and an abnormal one begins, both being connected by the most imperceptible transitions." One difficulty, of course, lies in the concept of the n o r m a l, which is always to a large extent arbitrary. The normal never in actuality signifies the a v e r a g e condition in a population, since to arrive at a definition of the average, diseased, undernourished and otherwise "abnormal" individuals would necessarily be taken into account. It occasionally implies the m o d a l class of the population — that into which most individuals fall. But often the "normal" is a kind of subjective sublimation of the population, from which all individuals deviate in some major or minor respects. If the modal form is regarded as normal, then clearly what is

normal for one population of a species is not necessarily so for the next, and it is even more obvious that the normal of one species is often the abnormal of another. Particularly is this true of reproductive structures in the higher plants, where change — mainly reduction — in numbers of functional parts has undoubtedly been an evolutionary trend, and where neighbouring species are often found to have progressed to different levels.

At the other end of the scale of phenomena are the extreme forms of plant teratism which affect function, and are therefore to be regarded as pathological. Here are to be found the truly "monstrous" aberrations, involving gross distortions — often mechanical lysis — of tissues; malformations classed under the headings of proliferation and fasciation belong here. The majority of such aberrations are defects of g r o w t h rather than d e v e l o p m e n t, using the word "growth" to cover the purely quantitative changes occurring during the life of the individual, including increase in the size or numbers of cells and organs all of essentially similar types, and "development" to imply the qualitative changes involved in the passage of the individual from the embryo to the reproductive stage.

The tendency for extreme malformation of organs may be inherited, or may appear spontaneously in apparently normal individuals (cf. the classical work of de VRIES on *Crepis,* etc. 1910). Aberrations of the nature of hypertrophies are occasionally traceable to nutritional causes, and in particular to abnormalities of nitrogen supply. Certainly there is evidence that excessive luxuriance of growth made possible by the abundant availability of nutriment is often the prelude to loss of growth co-ordination. In this connection it is interesting to compare the recent words of WHITE 1948 reviewing the subject of fasciation with those of WORSDELL, written thirty-three years earlier. WORSDELL 1915 discussing the physiological cause of fasciation said, " . . . it is doubtless stimulated to appear by the presence of superabundant nutrition which produces a subtle diseased condition, thus giving rise to a hypertrophied growth which destroys the balance of the organism". WHITE 1948 concluded, "The basic cause of fasciation is a disturbed metabolism, involving excessive nutriment which mobilises energy that must be utilised. This energy, once accumulated, must go into growth, and it becomes 'wildly' expended in extravagant, abnormal and unpredictable tissue production, generally to the detriment of the plant." Both of these conclusions are, however, rather descriptive of the phenomenon than truly explanatory. Excessive nutrition in itself does not account for the abnormal element in fasciation and similar hypertrophic conditions: accummulation of nutrition does not invariably lead to hypertrophy, nor is it the case that hypertrophies are never produced by tissues with

normal availability of nutrition, or even for that matter, by those in a state of relative starvation, as shown for example by WHITES 1916 experiments with fasciated *Nicotiana*.

A proximate cause of many of these extreme abnormalities of growth is probably to be found in maldistribution of auxins. The plant growth substances so far isolated are remarkably unspecific, being physiologically effective in a wide range of tissues. Furthermore, they possess the notable capacities of provoking different responses from different tissues in the same effective concentrations, and conversely, of provoking different responses from the same tissue in different concentrations. The auxins themselves undoubtedly constitute important morphogenetic agents, in that their movement in the plant body, the concentration gradients which they form and their rates of deactivation in various tissues, all by local alterations in growth rate modify or control various aspects of external form.

Shapeless tissue proliferations, break-down of normal growth correlations resulting in abnormal patterns of branching and many similar teratological phenomena may be traceable directly to the maldistribution of the non-specific growth hormones. How such maldistribution arises is another matter. The clearest cases are those involving parasitism, where infesting organisms produce effects on the host out of all proportion to their size and often persisting after the direct influence of the parasite has ceased. The crown-gall problem has recently been reviewed by de ROPP 1951, who stresses the fact that crown-gall tissue contains a higher auxin content than normal tissue, and that the presence of crown-gall tissue in one part of the plant produces several responses in other parts (epinasty of petioles, bud inhibitions) similar to those produced by the application of high concentrations of synthetic growth substances. Proliferations strikingly similar to crown galls can be produced by hormone treatment (BROWN and GARDNER 1936, 1937), while on the other hand they may arise from innate genetical causes without any form of external provocation, as in the case of tumours produced by certain *Nicotiana* hybrids (WHITAKER 1934). This is another aspect of the capacity of plant cells to throw off co-ordinating influences and acquire growth autonomy, possibly as de ROPP suggests through the auto-catalytic renewal of growth substances within them.

Abnormalities of auxin distribution at physiological rather than at pathological levels produce growth anomalies in which integration of cellular activities is retained. A classical example of this is the *nana* form of maize ("teratological" from one point of view) in which dwarf growth is imposed by the rapid oxidative destruction of auxin in the tissues: the abnormality is hereditary, and due to a single gene

(van OVERBEEK 1935). There seems little doubt that many teratisms involving branching and phyllotactic abnormalities arise from the breakdown of the growth co-ordination imposed by the polar transport of auxins in the plant. Correlation of growth of leading and lateral buds and shoot is brought about through the inhibitory effect on laterals of auxins produced at the stem apex (SNOW 1937, THIMAN 1939). Parasitic (or other) destruction of apical buds can thus greatly modify branching patterns, the effects being evident in parts of the plant quite remote from the point of infection. The influence of growth substances on the production of leaf primordia at shoot apices has been investigated by SNOW and SNOW 1937 and BALL 1944, and it has been demonstrated that both phyllotaxis and branching patterns can be modified by such treatment. COULT 1946 has interpreted branching and phyllotactic anomalies in *Salvia* in terms of maldistribution of growth substances, and there seems little doubt that the changes in distributions of leaves and lateral shoots produced by parasites (as for example by *Perrisia crataegi, Eriophyes rudis)* arise from traumatic disturbances of auxin distribution.

A branching anomaly of a different order — dichotomy — is of rather frequent occurrence in flowering plants. Bifurcated inflorescences in plants which usually bear simple racemes are illustrated in all of the older teratological manuals and are still being recorded (e. g., in *Orchis maculata* by SUNESON 1951). Such branching bears no direct relationship with the normal branching pattern of the plant, and seems to arise through chance division of the growing point into two, each of which continues independent and normal growth. The exact nature of the developmental "accident" is naturally likely to be untraceable by the time its effects become apparent, but in many instances it may again be traumatic. Some forms of fasciation can be interpreted as incomplete or incipient dichotomy, or this may result from the lateral expansion of a single growing point. A full discussion of this very common form of plant teratism is given by WHITE 1948. A striking feature of teratisms involving anomalies of stem growth is that their occurrence does not normally seriously influence development. The orderly progression from vegetative to reproductive state may be unimpeded, and the flowers produced may be normal, or at least as well integrated as the new mechanical limitations will allow. Alternatively there may be associated malformation of flowers *(Nicotiana,* WHITE 1916) but even this does not lead to a very great reduction in fertility.

Between minor abnormalities, classed often as such only because of an excessively restricted view of what constitutes the normal, and the class of extreme growth aberrations just discussed lies the controversial field of teratism. Here appear the great mass of floral aberrations

which have convinced observer after observer of their importance to comparative morphology, mainly because of their orderliness of form and frequency of appearance, the similiarity of their manifestation from species to species, and, in particular, the extent to which they often recall the GOETHEAN concept of the flower as the equivalent of a vegetative shoot.

What more, then, can be said about this claim beyond what was said by the pre-eminent students of plant morphology of half a century ago? Clearly, if any less equivocal assessment is to be made than theirs, it must be based upon sources of evidence not then available, and of such sources the most important are to be found in genetics and those branches of physiology concerned with growth and development.

Few writers on teratology of the post-Darwinian period have considered the phenomena they observed without in some way speculating as to their phylogenetical implications. Many have been concerned frankly to prove that they are largely atavistic — in WORSDELLS 1915 words, ". . . reversions, or harkings back to ancestral conditions." But it is, of course, possible to view morphological problems without any evolutionary associations whatever, as indeed did GOETHE, DE CANDOLLE and others who laid the foundations of classical morphology. It seems appropriate first to consider floral teratology thus, divested of its atavistic imputations.

MOQUIN-TANDON 1841, with a view uninfluenced by evolutionary thought, stated (quoted by WORSDELL) ". . . it would be an error to regard vegetable anomalies as freaks of nature, as strange or blind irregularities, resulting from fortuitous causes and leaving on the mind a confused memory of inexplicable deformities. Anomalies are particular modifications which can be brought under common, simple, and exact principles which are themselves but corollaries of the most general laws of organisation." A little more than a century later we possess a somewhat better understanding of these laws of organisation. SACHS' hypothesis that growth and organogenesis might in some way be governed by chemical substances has received conclusive proof, and we know that organisation is often imposed by the movements of determining substances in both plant and animal kingdoms. The plant growth substances, as stressed above, are well known to have important functions in the co-ordination and orientation of growth, largely through their control of cell elongation. They have in addition powers of provoking cell divisions when supplied in high concentrations to tissue no longer meristematic, and of promoting the formation of certain organs, apparently d e n o v o. This last capacity, expressed most unequivocally in the production of roots, is significant in its morphogenetic implications. Auxin treatment does not only accelerate the production of roots

by stem and other cuttings; it may actually lead to the appearance of roots where none would otherwise have been. Whether it acts directly as an evocator, or through the intermediary of a "rhizocaline" (WENT 1938), it is a link in the chain of an organ- forming process. In the animal kingdom, understanding of developmental morphogenesis has progressed to a point somewhat beyond that so far attained for the higher plants, in that more is known about the cytoplasmic gradients which determine the early stages of embryonic development, and also about the specific substances — evocators — which promote particular histological changes and organ-forming processes. But, as emphasised by ANDERSON 1937, one feature of the growth pattern of higher plants differentiates it in a highly important way from that characteristic of animals — namely the possession of an open rather than a closed system of development. Organogenesis is essentially an embryonic phase of animal development; in contrast, plant growth involves the serial production of new organs throughout life, so that some parts of the plant are perpetually in an embryonic state. The organs formed at any one growing point, while not all alike, are generally regarded as conforming to a limited number of basic types, and the vegetative part of the life cycle of the higher plant consists simply in the growth in thickness and length of stems and the continuous production of homologous appendages at more or less regular intervals. The co-ordination and correlation of this monotonous pattern of growth is in part at least a function of the plant auxins, but the rôle these substances play in development is still a matter for speculation.

The transition to the reproductive phase is marked by a suspension in some or all of the branches of this regular pattern of vegetative growth, and the formation of flower primordia. The conditions under which this happens differ widely from species to species, but the following points seem clear: (a) that in the majority of cases the passage to the reproductive state is governed by certain environmental controls, of which temperature and day-length are the most important; (b) that within broad or narrow limits, depending upon the species, the time of transition from the production of new leaves and vegetative shoots to the production of flowers can be altered by modifying these controls and (c) that the change from the vegetative to the reproductive state is not abrupt, but is heralded often by physiological changes in the plant (sometimes accompanied by the appearance of leaves of different pattern) which indicate "ripeness to flower".

In conformity with the modern tendency to attribute growth phenomena to hormonal stimuli, it has been suggested that the transmission of flower-forming substances from the vegetative parts of plants (where they are formed under the appropriate environmental conditions) to the

growing points may be responsible first for the vegetative changes recognised as indicating ripeness to flower, and subsequently for the diversion of development from vegetative to reproductive organs. While no such substances have yet been isolated, repeated demonstration that the flowering stimulus can pass through a graft union (evidence reviewed by CROFTS 1951) provides cogent reason for believing it to be chemical in nature.

The difference between the plant and animal patterns of development is here very notable: the reproductive organs are amongst the first to be differentiated in animal ontogeny, and exert a controlling influence upon certain aspects of somatic development throughout life; in plants, conditions are reversed, in that vegetative tissues apparently produce the stimuli which actually initiate the formation of the reproductive organs. Furthermore, it is apparent from the above that the flower in effect replaces a vegetative shoot in the usual developmental progression, with the inflorescence as an intermediate term. Whether or not we are prepared to accept the flower as the equivalent of a vegetative shoot on morphological grounds, that is what it is physiologically speaking. The issue cannot be escaped by a suggestion such as that of GRÉGOIRE 1938 that the flower rudiment does not arise from a vegetative apex but from a new lateral outgrowth; the morphological and ontogenetic evidence refuting this has been reviewed by PHILIPSON 1947, who himself concludes that the organisation of apical meristems cannot be regarded as essentially different in the vegetative and reproductive states (see also the discussion by WEBER 1951).

The changes which occur at the apex involve a cessation of the indefinite production in serial succession of primordia which develop into leaves, and the formation there instead of those which form the parts of flowers. Added complications follow: the growth of the shoot becomes determinate in that it ceases after the formation of a limited number of primordia; the order of outgrowth of the primordia is no longer necessarily strictly acropetal; the pattern of "phyllotaxis" may alter, and it may be that the growth fields formed round the apex of the stem may ultimately become toroidal. Irrespective of these secondary occurrences, in the physiological sense, leaves and the parts of flowers are equivalent organs, representing alternative developments at the growing point of the shoot. According to the hypothesis of GREGORY 1949, which is formed there depends on which of two organ-forming substances is produced from a common precursor in the prevailing environmental conditions. There is here no immediate phylogenetical implication, nor for that matter any obligation to regard floral parts as "transformed" leaves — one could as well view the matter in the reverse sense. The physiological approach thus results in a view of the process strikingly similar to

what seems to have been that of GOETHE (discussed by ARBER 1937) to whom the idea of e q u i v a l e n c e held no particular implication of ontogenetic or phylogenetic t r a n s f o r m a t i o n.

The work of the last two decades on the physiology of flowering bears on the subject of floral teratology in a number of important respects. Whereas the transition from the vegetative to the reproductive state is usually a fairly definite process, it seems that the flowering stimulus does not necessarily behave as simple trigger mechanism which at a certain threshold level sets in train a succession of irreversible changes. In certain cases it has been shown that there is a form of quantitative relationship between the inducing procedure (control of temperature, photoperiod) and the number of flowers produced or their structural perfection as such (BIDDULPH 1935, NIELSEN 1942, MADSON 1947 and others). That a constant stimulus, presumably hormonal in nature, is required for the initiation of the normal process of sporogenesis even after flower-set is demonstrated by the results of GREGORY 1949, who found that while excised anthers cultivated i n v i t r o grew actively, even to a size greater than normal, if they had been removed before the inception of reduction, the archesporium continued mitotic divisions without undergoing meiosis.

When we apply these ideas to the interpretation of floral teratism, the force of MOQUIN-TANDONS words quoted above becomes apparent. The vast literature of photoperiodism provides many examples of the results of modification of the orderly succession of external influences, which govern the normal transition of the plant from the vegetative to the reproductive state, in such a way that the growing point is not committed positively to one or the other course of development. The organ-forming processes are dislocated, and the result is frequently the production of morphological anomalies. Often these are identical with "teratisms" reported in nature: foliaceous bracts and sepals, suppressed corollas, contabescent stamens, or even the complete transformation of the inflorescence into "vegetative flowers" (*Rudbeckia,* MURNEEK 1940; *Glycine,* NIELSEN 1942; *Kalanchoë,* HARDER and BODE 1943; *Cosmos,* MADSON 1947, etc.) Yet the production of these abnormalities is indeed, as believed by MOQUIN-TANDON, but a corollary of a general law of organisation.

A physiological explanation for many types of teratism, vegetative and floral, can thus be supplied in terms of anomalous distributions of the hormones (auxins) concerned with growth processes and those (so far putative) governing development. Both groups of hormones are morphogenetic agents, and the auxins at least are extremely unspecific in their action, producing their characteristic responses in a wide range of plants. The rooting stimulus, for example, is a general one, effective

in monocotyledons and dicotyledons alike. But the stimulus is simply one link in a complex chain: auxin treatment is effective in promoting root formation only in tissue competent to react in that manner to its presence, and the pattern of root produced is a function of the reacting tissue. Equivalent situations must prevail whenever histological changes or organ-forming processes are governed by substances which are effective in a wide range of organisms — as for example with the evocators responsible for promoting organ and tissue differentiation in animal embryos (NEEDHAM 1942).

If we accept the postulate that somatic segregation of nuclear material does not occur, then we must attribute totipotency to all the nuclei of the organism. That cells are not all equally capable of reacting to stimuli must then be the result of diversity of nuclear environment. The differential may result from the position of the particular tissue in the organism or from differences in cell history: in any case it must be expressed through the cytoplasm and be chemical in nature unless we take account of the important possibility of electrical polarisation. Differences between tissues in their capacity to react to stimuli arise thus during the life-time of the individual, and in part at least are functions of their "commitment" to special purposes — although such commitment can occasionally be over-ruled, and the tissue restored to the juvenile state (LAIBACH 1935). It may be that the history of a cell in the life of an individual may not only affect its capacity to react to a stimulus but also to some extent the form of its reaction, but largely this will be governed by its genetical make-up. And this brings us to a point where we can no longer ignore the genetical (and thus evolutionary) aspect of morphological abnormality.

Since the points at issue are more readily comprehended from a concrete case, let us continue with the consideration of floral teratology. We have seen that from the physiological point of view vegetative and reproductive development can be regarded as alternative processes, and that which shall prevail at a growing apex is governed by a physicochemical system. If this functions normally, the growing point is committed positively one way or the other; should it be modified, structures of an intermediate character may be produced. It is indeed difficult to imagine how it could be otherwise. Whatever course of development is pursued, the cells involved are genetically equivalent (if any proof is required, it is supplied by the fact that complete plants can be regenerated from leaves, while of course all developmental possibilities must exist in the spores), and a physiological mechanism which could commit them with 100% certainty to one or the other path under all environmental conditions without ever producing an indeterminate result is well nigh inconceivable. This exposition of the flowering process and of the

circumstances under which abnormalities arise has no phylogenetical implications in itself, and would have equal validity in the absence of any concept whatever of organic evolution.

It takes on phylogenetical significance almost, as it were, by accident, when attempts are made to deduce ancestory from the structure and ontogenetic behaviour of living organisms. The possession of positively differentiated vegetative and floral structures is seen as "advanced"; therefore intermediate structures do not only indicate the homology of leaves and floral parts in the GOETHEAN sense, they must be taken to represent a reversion to an ancestral condition, albeit "modified according to the idiosyncrasy of the present types which exhibit them" (WORSDELL 1916).

Now this claim contains not only a postulation as to the path evolution has taken in the flowering plants, but also one at to h o w it has progressed. It implies that the flower has arisen through increasing specialisation of function, but that the genetical developments involved have been superimposed one upon the other. Thus nothing has ever been lost in the angiosperm lineage, and the germ-plasm of modern plants retains all of the capacities of ancestral forms, and these, with or without external provocation, reappear from time to time as "atavisms". This proposition, although its full significance does not ever seem to have been recognised by any of those suggesting atavistic explanations for teratological phenomena, is perhaps not without its germs of truth.

Strictly, the evolutionary problem is not how leafy shoots became transformed into flowers, since reproductive organs must have been present at all stages of evolution of the sporophytes of land plants, but how a basic structural unit supplying both functions became specialised for one or the other [1]). If this was indeed the primitive state, advance from it must have been in two directions: progressive sterilisation of one group of organs with specialisation for carbon assimilation and other vegetative functions, and progressive reduction of the vegetative capacity of the other, with specialisation for spore production, and later for seed formation and associated functions such as pollination. The evolutionary process must therefore have involved a form of neoteny — progressive delay of maturation, with more and more cell generations of the diplophase devoted to nutrition before the onset of reproduction; in other words, continued operation of NÄGELIS fundamental "law" of organic development (see discussion by BOWER 1935). A genetical mechanism by which this might be achieved would involve genes having the function simply of slowing down the rate of developmental processes leading to meiosis in relation to the rate of vegetative cell

[1]) The telome theory (ZIMMERMANN 1930) provides a picture of what this unit may have been like.

multiplication. Viewed thus, the physiological problem of reproduction in plant sporophytes, pteridophytan and spermatophytan, is not how sporogenesis is brought on, but how it is held off during vegetative growth. It may be in this direction that the significance of recent discoveries relating to the inhibiting action of auxins — substances promoting vegetative growth — upon flower production actually lies.

The acquisition of the mechanism for delaying spore-production must have been an early event in the evolution of the sporophytes of the vascular plants, and organ specialisation — notably absent in *Rhynia*, for example — a later development. The genes governing the developmental process must have been supplemented by others effective in promoting the efficiency of the vegetative organs in their various nutritional roles but without effect in later reproductive stages, and by those similarly increasing the efficiency of the reproductive structures but not effective earlier. These different rôles of genes in the life of the individual, and their capacity for exerting different effects upon homologous organs according to the time of production (and thus position) of those organs in the plant, have been studied in modern species, for example in *Primula sinensis* (de WINTON and HALDANE 1933; ANDERSON and de WINTON 1935). It is interesting to note that these time relations between vegetative and reproductive development are parallelled in animals which pass through larval stages; here genes may have their major effects in either the larval or adult stages, not being equally effective in both (cf. HALDANE 1932).

This line of argument leads to the conclusion that in one sense at least evolutionary developments in spore-producing plants have indeed been cumulative. Specialisation has been attained through the limitation of once generalised functions to particular periods of the life-cycle of the individual, and through the acquisition of new capacities concerned with these functions and operative during the same periods. Thus genes controlling specific features of the specialised organs (in flowering plants one might quote palisade characters in the leaf, pigmentation and scent in the flower) acquired later in evolution would also come into action late in ontogeny, after tissues had become committed to one or the other course of development.

The whole system is thus likely to be of considerable complexity, and when we consider what would be implied by a complete reversion to the presumed primitive state in which foliar and floral structures were undifferentiated, then the force of the principle of evolutionary irreversibility (usually referred to as DOLLOS Law) becomes clear. Not only would such a change have to affect the initial commitment of the tissues, but it would also have in some manner to prevent the operation of all the subsequent processes of tissue differentiation

which would normally be expressed in the specialised modern foliar and floral organs but which cannot have had any counterpart in the primitive unspecialised form. Complete reversion to this form we may take, then, as highly improbable; what must happen when we so modify the environment as to upset the positive commitment of the growing point is that we release the potentialities of both forms of development together, and the organ produced is intermediate in character so far as these potentialities are capable of being simultaneously realised in the same tissue.

This must also be true of flower teratisms in nature, and that such must be the case was to some extent recognised by WORSDELL 1915, even while he claimed their atavistic nature, as shown by the following words. — "... a vegetatively proliferated rose ... tells us ... that the flower has been derived in the past by congestion and abbreviation of an axis, and by extreme reduction and modification of leafy sporophylls. But it would be absurd to suppose that the leafy shoots from which our flowers originally sprang in any sense resembled, save in the matter of possessing an elongated axis and leafy sporophylls, those into which our modern flowers so frequently proliferate."

Reversion to extremely remote ancestral conditions is thus highly improbable in that not one but many correlated changes would be involved. Are the possibilities of less extreme reversions any greater? Here it would seem that we are dealing with a case of statistical probability; the fewer the steps involved, the more likely that they can be traced in the correct order. Where one or two gene changes are involved, there can be no doubt that ancestral conditions can be regained, either through reverse mutation (MULLER 1939), through reconstitution of ancestral gene patterns through hybridisation, or through the overruling of gene action by environmental stimuli. Instances of this are to be found in the re-appearance of floral organs, the development of which presumably has been suppressed in relatively recent evolutionary time. An example is the reappearance of the fifth stamen in *Digitalis* (and many other four-stamened *Scrophulariaceae*) usually to be found in some flowers in any large collection. This stamen may be even functionally perfect, and its presence must affect the normal pollination mechanism but slightly if at all. Presumably the genetic mechanism responsible for its suppression in normal individuals is relatively simple. Innumerable parallel cases can be found in the literature on so-called sex-reversal in flowering plants; the commonest form is the reversion of unisexual flowers to the bisexual condition through the appearance of the suppressed androecium or gynoecium. This may arise through the functional failure of the genetical mechanism responsible for the suppression (WESTERGAARD 1948) or through external treatment

designed to overcome the internal genetical controls (LÖVE and LÖVE 1945). All of these instances are of the nature of reversions, and all are in the biological meaning of the word, teratological — although there is nothing basically monstrous in the appearance of bisexual flowers in *Melandrium* of the type found in *Silene*, or of the fifth stamen in *Digitalis* so that the flower is similar to that of *Verbascum*.

Pelory, a phenomenon common enough in many species possessing zygomorphic flowers, is doubtless of the same order. The change from radially symmetrical growth of primordia which produces an actinomorphic corolla to the slightly asymmetric pattern which produces zygomorphy is relatively small; presumably the genetical change which produces it is also not of a very basic type (e. g., *Antirrhinum*, BAUR 1930). Genetical reversion and developmental reversion over-ruling gene control are probably both effective in producing the peloric flowers so often observed in populations of normally zygomorphic species. Here there is some evidence that the peloric forms are not always as well adapted for pollination as the zygomorphic flowers of the same species or as actinomorphic flowers of other species, and it may be that other changes have been superimposed on the change in growth pattern so that mere reversion to symmetrical growth does not wholly reproduce the ancestral type.

While all developmental abnormalities provoked by external causes must follow courses governed by the genetical potentialities of the tissues concerned, there is no reason to suppose that changes may not arise in the germ-plasm itself — as they must have in the past — which might provoke morphological aberrations of a type "new" to the species — new in the sense that the potentialities for their production had never existed before. Sufficiently gross aberrations of this nature would, initially at least, be considered teratological. The evolutionary importance of such changes would naturally depend upon their survival value; successful large mutations (the "hopeful monsters" of GOLDSCHMIDT 1940) must be extremely rare, if for no other reason that the chance of a single change involving a large number of functions being of such a kind as not to destroy their integration is likely to be small.

If the above discussion seems to have proved everything of teratisms: that they may be meaningless and meaningful, atavistic and not atavistic, progressive and retrogressive, provoked from within as well as from without, then it has served its purpose in drawing attention again to the potentialities of the field for further study. Evidently, each separate case must be considered on its own merits, and to do so is not illogical, as charged by ARBER 1931, but is rather a recognition of the multitude of causational factors. All developmental abnormalities are physiologically significant since through their study lies the hope

of comprehending the "normal": the provocation of abnormality is the main experimental method of physiology. To morphology some teratisms are significant, for as ARBER 1937 has said elsewhere " ... it is an undeniable truth — indeed a truism — that aberrant forms, since they show what an organ c a n do, may sometimes throw light on what it i s." To the evolutionary geneticist, teratological phenomena have special significance if he is prepared to apply caution in interpretation, for they can both suggest where evolution has come from and where it may be going to — if the directions can be disentangled —, as well as supplying a form of evidence as to the mechanisms involved.

Summary

The implications of recent work on the physiology of plant growth and development are discussed in relation to the causes of teratological phenomena, and it is concluded that under this heading a wide range of different anomalies is grouped. These include:

(a) major abnormalities of g r o w t h, which are sub-pathological in nature and have little morphological significance except to the extent to which they shed light on the origin of normal patterns of branching, phyllotaxis etc.;

(b) abnormalities of d e v e l o p m e n t, resulting probably from failure of the hormonal systems governing flowering, and producing, i n t e r a l i a, structures intermediate between leaves and floral parts. These abnormalities may have significance in interpreting homologies of organs, but cannot be regarded as atavistic;

(c) minor abnormalities of organogenesis which may arise from internal (genetical) cause or external (environmental) causes, some of which may in the true sense of the word be considered reversionary in that they may reproduce exactly ancestral conditions, and others of which may be progressive in that they represent developmental paths not previously followed in the evolutionary lineage.

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