homografts in the standard manner. The operative techniques used were satisfactory since the autografting of skin was consistently successful.

In contrast to the observations of Werder and Hardin[1,2], we were unable to demonstrate any beneficial effect on the survival of homografted skin after repeated grafting from either specific or random donors to either splenectomized or non-splenectomized recipients.

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ROY G. SHORTER
JACK L. TITUS
DONALD M. LEVY

Section of Experimental and
Anatomic Pathology,
Mayo Clinic and Mayo Foundation,
Rochester,
Minnesota.

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Group Selection and Kin Selection

WYNNE-EDWARDS[1,2] has argued persuasively for the importance of behaviour in regulating the density of animal populations, and has suggested that since such behaviour favours the survival of the group and not of the individual it must have evolved by a process of group selection. It is the purpose of this communication to consider how far this is likely to be true.

The strongest arguments for believing that conventional behaviour is the immediate cause regulating population density concern cases of territorial behaviour, particularly in birds. But it does not follow that such behaviour has evolved by group selection, because territorial behaviour capable of adjusting the population density to the available food supply could evolve by selection acting at the level of the individual rather than of the group. The appropriate degree of aggression would evolve if: (1) individuals which are too aggressive raise fewer offspring, either because they suffer physical damage or because they waste in display time and energy which should be spent in raising their young; (2) individuals which are too timid either fail to establish a territory or establish one too small to contain an adequate food supply for the young. Further, the degree of 'choosiness'—that is, the readiness to fight for a territory in one kind of area rather than put up with one in a less favourable area—will evolve by individual selection in such a way as to lead to an efficient distribution in space. This will happen because if, on one hand, individuals are too 'choosy', territories in the favoured areas will become too small in relation to the food supply, so that less choosy individuals breeding in the less favoured but more sparsely inhabited areas will leave more offspring, whereas if individuals are too little choosy, selection will act in the reverse direction.

Thus there is no need to invoke group selection to explain the evolution of individual breeding territories, or the adjustment of territory size to food supply or to variations in the habitat. But there are other characteristics of animals which are more difficult to explain by individual selection; sex is an obvious and important example, but difficulties also arise in explaining the evolution of 'altruistic' characters, such as alarm notes or injury-feigning in birds.

It is possible to distinguish two rather different processes, both of which could cause the evolution of characteristics which favour the survival, not of the individual, but of other members of the species. These processes I will call kin selection and group selection, respectively. Kin selection has been discussed by Haldane[3] and by Hamilton[4].

By kin selection I mean the evolution of characteristics which favour the survival of close relatives of the affected individual, by processes which do not require any discontinuities in population breeding structure. In this sense, the evolution of placentæ and of parental care (including 'self-sacrificing' behaviour such as injury-feigning) are due to kin selection, the favoured relatives being the children of the affected individual. But kin selection can also be effective by favouring the siblings of the affected individuals (for example, sterility in social insects, inviability of cotton hybrids due to the 'corky' syndrome[5]) and presumably by favouring more distant relatives. There will be more opportunities for kin selection to be effective if relatives live together in family groups, particularly if the population is divided into partially isolated groups. But such partial isolation is not essential. In kin selection, improbable events are involved only to the extent that they are in all evolutionary change—in the origin of genetic differences by mutation.

If groups of relatives stay together, wholly or partially isolated from other members of the species, then the process of group selection can occur. If all members of a group acquire some characteristic which, although individually disadvantageous, increases the fitness of the group, then that group is more likely to split into two, and in this way bring about an increase in the proportion of individuals in the whole population with the characteristic in question. The unit on which selection is operating is the group and not the individual. The only difficulty is to explain how it comes about that all members of a group come to have the characteristic in the first place. If genetically determined, it presumably arose in a single individual. It cannot be pictured as spreading to all members of a group by natural selection, because if it could do that, it could equally well spread in a large population—either by individual selection or kin selection—and there is no need to invoke a special mechanism of group selection to explain it. Hence the only way in which such a characteristic could spread to all members of a group would be by genetic drift. (There is also the possibility that it might spread through a group by cultural transmission, but this is unlikely to be important in animals other than man.) If this were to happen at all often, then the groups must be small (or else commonly re-established by single fertilized females or single pairs), the disadvantage of the characteristic to the individual slight, and the gene flow between groups small, because every time a group possessing the socially desirable characteristic is 'infected' by a gene for anti-social behaviour, that gene is likely to spread through the group. These conditions are severe, although they may sometimes be satisfied.

The distinction between kin selection and group selection as here defined is that for kin selection the division of the population into partially isolated breeding groups is a favourable but not an essential condition, whereas it is an essential condition for group selection, which depends on the spread of a characteristic to all members of a group by genetic drift.

Wynne-Edwards[2] points out that birds may return after migration to the precise spot where they were raised, and argues that this would favour the operation of group selection. This is not so. What is required for group selection is that the species should be divided into a large number of local populations, within which there is free interbreeding, but between which there is little gene flow. The mere fact that many birds breed near where they were born does not bring about this situation; it would favour the operation of kin selection, but it is difficult to see how kin selection could bring about the evolution of many of the types of population-regulating behaviour which Wynne-Edwards believes he has discovered.

Wynne-Edwards also argues that the behavioural mechanisms he hypothesizes would be proof against the occurrence by mutation and subsequent spread of anti-

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social behaviour patterns because of genetic homœostasis. This is a piece of special pleading: it also shows a misunderstanding of the situations in which homœostasis of this kind is to be expected. Both genetical theory and the experimental evidence suggest that if natural selection has been pushing a character in a given direction for a long time, it will be difficult for selection to produce further change in the same direction, but comparatively easy to produce a change in the reverse direction. Thus it would only be plausible to suggest that there are genetic reasons why anti-social behaviour should not increase if it were also suggested that selection had already produced an extreme degree of anti-social behaviour, and this is precisely what Wynne-Edwards denies. In fact, 'anti-social' mutations will occur, and any plausible model of group selection must explain why they do not spread.

There is one special form of group selection which is worth considering in more detail, because it can, perhaps, explain the evolution of 'self-sterilizing' behaviour; that is, behaviour which leads an individual not to breed in circumstances in which other members of the species are breeding successfully. (This is quite different from behaviour which leads individuals not to breed when other members of the species are attempting unsuccessfully to breed, or to produce fewer offspring when conditions are such that they would be unable to raise a larger number; such behaviour, although of great interest, presents no special difficulty to a selectionist.) The difficulty is that if the difference between breeders and non-breeders is genetically determined, then it is the breeders whose genotype is perpetuated.

A possible explanation is that what is inherited is the level of responsiveness to the presence of other breeding individuals. Thus suppose that there are aggressive A individuals which continue to breed or to attempt to breed at high densities, and timid a individuals, which are discouraged from breeding when the density of breeding individuals reaches a certain level, the difference between A and a being genetically determined. In a mixed group of A and a individuals, if the density is high, only A will breed, and a will be lost from the group. In a group of A individuals at high density all will attempt to breed, with the consequence that the food supply may be exhausted and the group produce few progeny. In a group consisting entirely of a individuals, at high densities some will breed and some will not, the difference between breeding and non-breeding individuals being due to age, to previous environmental history, or even to chance. Consequently an a group is less likely to outstrip its food supply, and so will leave more progeny. The difference between A and a groups at high densities is an example of the difference between a scramble (A) and a contest (a) (ref. 6).

Given such a behavioural difference, the following conditions seem necessary if a is to increase under natural selection:

(1) Groups must for a time be reproductively isolated, because a is eliminated from mixed groups.

(2) Groups must be started by one or a few founders, since otherwise groups consisting entirely of a individuals would never come into existence.

(3) When a group of A individuals outstrips its food supply, it must not immediately encroach on the food supply of neighbouring a groups, for if it did so, the advantage of a groups would disappear. This is a difficult condition to meet, and appears to rule out this mechanism in cases in which the population is divided into herds, flocks, troops or colonies, each group having a joint feeding territory which borders that of neighbouring groups. The condition is most likely to be met when the food supply is discontinuous in space, each patch of food supporting its own group.

A greatly oversimplified model of this type of selection will now be given. To fix ideas, suppose that there exists a species of mouse which lives entirely in haystacks. A single haystack is colonized by a single fertilized female, whose offspring form a colony which lives in the haystack until next year, when new haystacks are available for colonization. At this time, mice migrate, and may mate with members of other colonies before establishing a new colony. The population consists of aggressive A and timid a individuals, timidity being due to a single Mendelian recessive; a/a are timid, and A/a and A/A aggressive.

Only when a colony is started by an a/a female fertilized by an a/a male will it consist finally of a individuals; all other colonies will lose the a gene by selection, and come to consist entirely of A individuals. Thus at the time when colonies are about to break up, there are only two kinds of colony, A and a. It is assumed that an a colony contributes $1 + K$ times as many mice to the migrating population as does an A colony, and has a proportionately greater chance of having a daughter colony.

In one summer, let the frequency of a colonies be P_0. Then, in the migrating population, the proportion of a/a individuals is:

$$\frac{P_0(1 + K)}{P_0(1 + K) + 1 - P_0} = \frac{P_0(1 + K)}{1 + KP_0} = p \text{ say}$$

It is assumed that a proportion r of all migrating female mice mate with males from their own colony, the remaining $(1 - r)$ mating at random. Hence the frequency of $a/a \times a/a$ mating as a fraction of all matings is

$$rp + (1 - r)p^2 = P_1$$

where P_1 is the frequency of a colonies in the next summer.

Hence the condition for the evolutionary spread of 'timid' behaviour—that is, of the a gene—is:

$$rp + (1 - r)p^2 > P_0, \text{ where } p = \frac{P_0(1 + K)}{1 + KP_0}$$

This reduces to

$$r(1 + K) - (1 - P_0K^2) > 0$$

Thus when P_0 is large ($P_0 \simeq 1$),

$$r + K > 1$$

and when P_0 is small ($P_0 \simeq 0$),

$$r(1 + K) > 1$$

Thus, if there is little or no interbreeding between colonies even at migration ($r \simeq 1$), timid behaviour will evolve provided it is an advantage to the group; this corresponds to the case in which the population is divided into more or less permanently isolated groups, which are periodically reduced to very small numbers, and which may either become extinct or split to give rise to two groups. However, the conclusion that timid or altruistic behaviour can readily evolve if there is no interbreeding between groups means little, since it is unlikely that species are often divided into a large number of small and completely isolated groups.

If there is fairly free interbreeding between colonies at regular intervals (that is, if r is small), selection could maintain the gene for timidity once it had become the common allele in the population. For example, if there were random mating, $r = 0$, between members of different colonies at the time of migration, then selection could maintain a as the common allele if a colonies had a two-fold advantage. But, with random mating, selection could not cause a to increase if it were initially rare: if $r = 0$, the condition $r(1 + K) > 1$ cannot be satisfied.

With an intermediate amount of gene flow between colonies, selection could both establish and maintain timid or altruistic behaviour, provided that colonies with altruistic behaviour have a large selective advantage, and that colonies are founded by very few individuals.

The model is too artificial to be worth pursuing further. It is concluded that if the admittedly severe conditions listed here are satisfied, then it is possible that behaviour

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patterns should evolve leading individuals not to reproduce at times and in circumstances in which other members of the species are reproducing successfully. Whether this is regarded as an argument for or against the evolution of altruistic behaviour by group selection will depend on a judgment of how often the necessary conditions are likely to be satisfied.

J. MAYNARD SMITH

Department of Zoology,
University College,
London.

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DR. MAYNARD SMITH'S communication raises a good many more questions than I can attempt to answer here. The major obstacle to constructive discussion between us really arises from the understandable (though regrettable) differences in outlook and experience between a laboratory geneticist and a field ecologist. To me his picture of territorial systems and other aspects of conventional behaviour appears scarcely true or comprehensive enough to provide a basis for valid deduction; my own grasp of the genetical theory of natural selection, on the other hand, no doubt looks still more halting and inept to him. We ought to enlarge the area of common ground, but that is too big a task to discharge effectively here.

It is not permissible to isolate 'territory' in Dr. Smith's sense from the other overlapping forms of real property won and defended by animals, such as nest-sites of colonial birds, basking or resting places of, say, seals or crocodiles, roosting perches of starlings or domestic fowls, display arenas of manakins or bower-birds, burrows of foxes or beach crabs, and so on. Many of these have no direct connexion with food or with rearing families; but all are indissociably bound up with the status of their possessor in the social system to which he belongs, and the rights which this status confers. What we have to explain is how social systems can evolve and their conventional machinery be perfected. What appears to be inevitably required is a process of selection discriminating between one social system and another.

Social systems are collective entities, in the higher animals frequently involving an element of tradition as well as genetic transmission as they pass down from generation to generation. They entail codes of behaviour with which the individual members instinctively comply, even when compliance demands the resignation of rights to vital resources or to reproduction. The hereditary compulsion to comply, for example, in lemmings doomed to emigrate or sticklebacks inhibited from maturing by the inescapable domination of an α male, is the real keystone of social adaptation. Individuals submitting to total deprivation are eliminated altogether, most often before they have produced any offspring; yet the tendency to comply is renewed in every subsequent generation and is not bred out. One is bound to conclude that it is very securely buffered from 'ordinary' selection acting against submissive individuals and at the same time promoting their dominant sibs; and from the effects of simple Mendelian situations of the A/a type in Dr. Smith's model. I stand corrected if it is technically wrong to think of this as genetic homœostasis; the apparent result is the same. The situation I describe here is real and not, I think, controversial; it is the explanation which presents difficulties.

Most ecologists would agree that the prerequisite of group selection that calls for a subdivided population structure is commonly and indeed normally found in animals. Dr. Smith says that the *Ortstreue* or return of migrant birds to their native locality would not bring it about; perhaps it is easier to see then in the case of the salmon or trout spawning in its natal tributary stream, where it more obviously becomes a member of a partially isolated breeding group.

The model of the mice in the haystacks is not, perhaps, a sufficiently close approximation to any natural situation to help us far towards a solution. A realistic counterpart might be, for example, the woodlice (*Porcellio scaber*) that fed on the green alga *Protococcus* living on tree-trunks, studied by Brereton[1]; marked woodlice confined their feeding to their own particular tree, and the population appeared to be subdivided thus into breeding units. Had any of the latter increased too freely they could have exterminated their stock of this particular food plant, which does not regenerate easily. Supposing in Dr. Smith's model that all the A colonies grow so fast that they finish the food and die of starvation before "migration time" arrives; then $K = \infty$, $p = 1$, $r = 1$, and $P_1 = 1$, and group selection wins the trick!

V. C. WYNNE-EDWARDS

Department of Natural History,
University of Aberdeen.

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Survival of Young Swifts in Relation to Brood-size

IN a recent article in *Nature*, Wynne-Edwards[1] said "the assumption, still rather widely made, [is] that under natural selection there can be no alternative to promoting the fecundity of the individual, providing this results in his leaving a larger contribution of progeny to posterity. This assumption is the chief obstacle to accepting the principle of intergroup selection".

Wynne-Edwards implies that this assumption is wrong, but omits to note that there are data which show that at least some species are producing as many surviving young as possible, for example, the starling, *Sturnus vulgaris*[2], the great tit, *Parus major*[3], and the laysan albatross, *Diomedea immutabilis*[4]. The swift (*Apus apus*) is a convenient species for a study of this kind since, like the albatross but unlike the passerine species studied, considerable mortality, due to starvation, occurs in the nest.

In England the swift normally lays a clutch of two or three eggs, clutches of four being very rare indeed (less than 0·25 per cent)[5,6], and it is interesting to consider what would happen if a larger clutch were laid. One cannot hope to observe a natural change (presumably a genetical mutation) which results in enough swifts laying clutches of four eggs instead of three to provide significant samples. However, by transferring young at hatching it is possible to compare the survival of young from broods of four with that from broods of two and three.

Swifts feed exclusively on airborne arthropods[7], the availability of which is greatly affected by the weather. In cold, wet summers the arthropods are less active and therefore less available to the swifts than in fine weather. At Oxford, Lack[5] showed that in fine summers the average number of young produced per brood was highest from broods of three whereas in cold, wet summers it was highest from broods of two.

In the summers of 1958–61 inclusive I increased some broods of swift to four young by adding a newly hatched chick at the time that a fourth egg would have been expected to hatch. Subsequent survival is summarized in Table 1. In the summers of 1958, 1960, and 1961, the weather, and therefore the feeding conditions for swifts, were fairly good, and in 1959 they were exceptionally so. (Following the method used by Lack[5], the mean maximum temperatures during the nestling period were 68, 70, 72 and 70° F for the four years, respectively.)

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Table 1

Year	Brood size	No. of broods	No. of young	No. lost	% lost	No. fledged/ brood
1958	1	7	7	2	28·6	0·71
	2	21	42	2	4·7	1·95
	3	4	12	1	8·3	2·75
	4	2	8	4*	50·0	2·00
1959	1	10	10	0	0	1·00
	2	15	30	0	0	2·00
	3	4	12	0	0	3·00
	4	4	16	5	31·2	2·75
1960	1	6	6	0	0	1·00
	2	18	36	2	5·6	1·89
	3	6	18	4	22·2	2·33
	4	5	20	14	70·0	1·20
1961	1	7	7	0	0	1·00
	2	18	36	1	2·8	1·95
	3	6	18	4	22·2	2·33
	4	5	20	13	65·0	1·40

* One more apparently left the nest prematurely.

It will be seen that, in all four years, the nestling mortality was markedly higher in larger broods, while in none of them did broods of four produce, on average, more surviving young per brood than broods of three. Even in 1959 when no nestling died in broods of 1, 2 or 3, 5 died out of 16 in broods of 4. (In three supplemented broods of four studied earlier by Lack (*in litt.*), eight young flew, but four of the six in the exceptionally fine summer of 1957 apparently fledged prematurely and there is doubt as to whether any of these survived.)

Hence, even in an unusually fine summer, swifts starting with 4 young raised less per brood than those with broods of three, so that natural selection must operate against any laying of clutches of four. As mentioned earlier, in some summers at Oxford broods of three have been the most productive and, in others, broods of two. Thus the present situation (of some individuals laying three eggs and others two, but virtually none four) is precisely what one would expect on the basis of natural selection. This conclusion is in agreement with the results for the other species cited, in all of which broods of larger than average size gave rise to fewer surviving young per brood than those of average size. This conclusion contrasts with the theory of Wynne-Edwards[1,8] that animals might maintain their reproductive rate below the maximum possible in order to prevent over-exploitation of the food supply, a theory which has yet to be demonstrated for any animal.

Christopher Perrins

Edward Grey Institute of Field Ornithology,
Oxford.

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I doubt if it is possible from Dr. Perrins's results to convince the sceptic that swifts are "producing as many surviving young as possible", and to demonstrate in this way that natural selection has, as he believes, forced their fecundity up to the potential limit of efficiency. A second look at the figures soon puts the conclusion in doubt. I agree that in England clutches of four tend to be inefficient; they constitute a relatively infrequent group, although they seem to have been commoner in 1958–61 than they were in the 1946–56 period[5,6]. Setting them aside we cannot be far wrong in assuming (having combined Perrins's samples for 1958–61 with those of Lack and Lack[6] for 1946–51) that the remaining swifts breeding in the Oxford neighbourhood in recent years have produced clutches of one, two or three chicks in a percentage ratio of about 24 : 61 : 15. Two-chick broods have evidently been by far the most common.

The average productivity of each of these brood-sizes, in terms of flying young produced, is shown in Table 2.

It is clear that the most productive brood-size over the 15 years in question has been three; by comparison with these, the broods of two have averaged fewer recruits and broods of one less than half as many. Dr. Perrins directs attention to the fact that in summers with poor weather the broods of three have no advantage over those of two: in fact, in the worst years like 1946–8 and 1953 they were fractionally (10 per cent) less productive[5,6]. But in average and good years they yield a favourable margin, and, as Table 1 shows, overall they have averaged not far short of 20 per cent more flying young than the broods of two. Why, one is entitled to ask, has natural selection not stabilized three as the optimum clutch, instead of just less than two, which is the observed average figure? Unless some additional hypothetical factor is postulated, like a differential survival of juvenile birds between leaving the nest and becoming adult breeders, Dr. Perrins's conclusion seems difficult to defend.

Table 2

Brood size	Young raised per brood: Lack and Lack (ref. 6)	Lack (ref. 5)	Perrins (preceding communication)
1	0·83	0·94	0·93
2	1·66	1·64	1·93
3	1·68	2·16	2·55

In Switzerland it is known that broods of three are still more productive (more than 90 per cent of the chicks so hatched grow into flying young); yet still only 67 per cent of the swifts there lay three eggs[9]. On the hypothesis that selection at the individual level necessarily promotes maximum fecundity it appears *a fortiori* "curious that all the pairs there do not lay three eggs"[10].

It is instructive to compare the swift with a bird like the red grouse (*Lagopus lagopus scoticus*), in which clutch-size is much more variable, and production of flying young progressively increases with brood-size throughout the whole size range[11] (Fig. 1). Notwithstanding the fact that female grouse in suitable circumstances lay ten or more eggs and the more they lay the more young they produce, the mean clutch-size in north-east Scotland has been found to be only 7·5 eggs. (Reproductive performance is subject to annual fluctuations and, in fact, the average clutch has varied between 5·9 and 8·2 in different years since 1957.) Dr. Perrins's hypothesis would not fit this species any better than it does the swift; indeed, the situation here seems further still from being "precisely what would be expected on the basis of natural [inter-individual] selection".

On my alternative hypothesis that inter-group selection has fixed the fecundity range around the optimum level

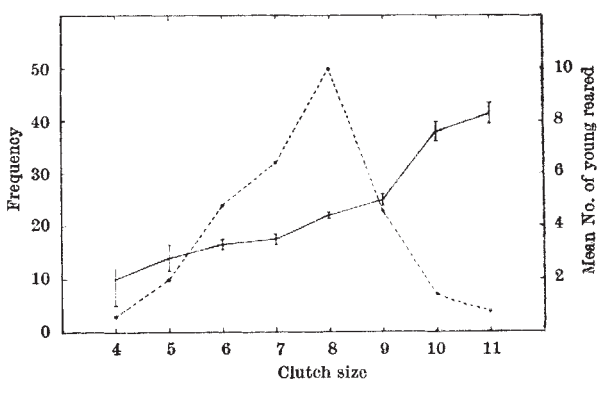

Fig. 1. Continuous line: Relation between clutch-size and mean number of young reared, in 153 broods of red grouse followed until August 12 (vertical bars show standard errors of means above and below). Broken line: Frequency distribution of clutch-sizes in same sample. (Drawn from data in ref. 11)

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for recruitment to the population as a whole, the facts for both species seem reasonably intelligible.

V. C. Wynne-Edwards

Department of Natural History,
University of Aberdeen.

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Spinal Pigmentomotor Tract of the Minnow (*Phoxinus phoxinus* L.)

Teleosts possess both neural and humoral pigmentomotor control. The neural control of teleost melanophores is known to be autonomic[1]. A melanophore-aggregating centre exists in the medulla of the minnow (*Phoxinus phoxinus* L.). A spinal pigmentomotor tract passes caudally from the medulla and has a spinal outflow localized around vertebra 15. The pigmentomotor nerves then pass cephalically and caudally along the sympathetic chains and innervate the melanophores through the spinal and trigeminal nerves[2]. The investigation recorded here was concerned with the location of the spinal pigmentomotor tract.

Mallory's trichrome, Mallory's eosin and methylene blue and Weigert Pal preparations were used to examine the anatomy of the intact spinal cord. Lesions were placed in the spinal cord with the aid of microdissection instruments, ground from fine steel needles by a high-speed 'Carborundum' wheel. Operations were performed under urethane anæsthesia on an operating table modified from that used by earlier workers in this field[3,4].

Ninety fish were examined. Lesions were placed at vertebræ 3, 4 and 10 and also around the spinal outflow. After recovery from anæsthesia the background reversal colour responses were tested and compared with the pre-operational background reversal colour responses. Finally, the extent of each spinal lesion was ascertained histologically from serial sections stained with Mallory's trichrome.

At vertebræ 3, 4 and 10, ventral spinal lesions, which destroyed up to 70 per cent of the spinal cross-sectional area, left the colour responses unaffected. At the same spinal-levels, dorsal lesions, which destroyed as little as 25 per cent of the spinal cross-sectional area, resulted in the elimination of the nervously controlled colour responses. Lateral lesions, which destroyed up to 30 per cent of the dorsal spinal cord on either side, had no effect on the colour responses. However, lateral lesions, which damaged or destroyed the dorsomedial region, did affect the colour responses. Lesions around the spinal outflow were varied in effect. The results indicate a spinal outflow between vertebræ 12 and 14, somewhat anterior to that recorded by von Frisch in his mid-European specimens[2]. Several dorsal lesions in this region left the colour responses unaffected, whereas a number of ventral lesions did affect the colour responses.

It is concluded that the spinal pigmentomotor tract lies within the dorsomedial area. Anterior to the region of spinal outflow the pigmentomotor nerves disperse ventralwards. The dorsomedial area of the spinal cord of the minnow includes the dorsal horns of the grey matter, surrounded by fine myelinated fibres, and the dorsal part of the corpus commune posterius. The dorsal horns of the minnow, and other teleosts, show little separation and consist largely of substantia gelatinosa Rolandi[5]. It has been suggested that the cells of origin of teleost preganglionic sympathetic fibres probably lie near the base of the dorsal horns and, also, that the substantia gelatinosa Rolandi might have a sympathetic function[5]. These experimental results indicate that the pigmentomotor tract of the minnow is located within the tissues around the base of the dorsal horns. This conclusion substantiates an autonomic function for the dorsal part of the corpus commune posterius and the substantia gelatinosa Rolandi.

This investigation was conducted, under the supervision of Dr. E. G. Healey, during the tenure of a Department of Scientific and Industrial Research studentship.

D. Burton

Department of Zoology,
Bedford College,
Regent's Park,
London, N.W.1.

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GENETICS

Mutagenic Activity of 1-Methyl-3-nitro-1-nitrosoguanidine on *Arabidopsis*

The efficiency of mutagenic agents is a complex value. It is not only dependent on the reactivity of the agent with the genetic material and on its applicability to a certain biological system, but also on the degree to which chromosome aberrations and other toxic effects are induced in addition to mutations. In order to examine the relationship between these forms of activity and to compare the efficiency of different mutagens, we have started investigations in which mutagenic agents are being tested for several biological effects under various treatment conditions in the same test system.

This communication describes experiments with 1-methyl-3-nitro-1-nitrosoguanidine (NG). NG has already been proved to be mutagenic in *E. coli*[3], it has cancerostatic activity in mice[2] and induces chromosome aberrations in *Vicia faba*[1], although the frequency of aberrations is relatively very low, even with sub-lethal doses.

Seeds of *Arabidopsis thaliana* were soaked for 18 h at 24° C in 0·06–10 mM NG (dissolved in distilled water, *p*H 4·2). The somatic effects were estimated by evaluating the percentage of germination, the speed of germination, the rate of growth of the primary root and the shoot, as well as the rate of survival. As a measure of mutagenic activity, the frequency of recessive lethals was determined by the embryo test[5,6]. In every M_1 plant 150 embryos in five successive pods of the main inflorescence were scored. (For the calculation of the values given in Table 1, compare with ref. 7.) The degree of semi-sterility may be used as an approximate measure of the frequency of chromosome aberrations.

With lower concentrations no significant somatic effects could be determined. At 1 mM the germination was approximately slowed down by 12 h (at normal rate of germination) and the root and shoot growth was markedly inhibited. The degree of survival was about 70 per cent (under aseptic conditions). At 2 mM 10 per cent of plants survived, at higher concentrations all plants died in the seedling stage. Since the decrease in the rate of survival only occurs at almost complete sterility (Table 1), NG can be classified as a mutagen with an extremely low relative toxicity (similar to ethyl methane-sulphonate[8] and to nitrosomethyl-urea[7]) and clearly differs in this respect from nitrogen mustard and also from X-rays[8]. The somatic effects can probably be traced back almost entirely to chromosome aberrations.

The frequency of mutations increases with increasing concentration, although only very slowly at the beginning; at higher concentrations, however, there is a marked linear rise. This form of the dose response curve is also typical for ethyl methane-sulphonate (EMS). At 1 mM

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