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

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[9] Weitnauer, E., *Orn. Beobachter*, **44**, 146 (1947).
[10] Lack, D., *Swifts in a Tower*, 190 (Oxford, 1956).
[11] Jenkins, D., Watson, A., and Miller, G. R., *J. Anim. Ecol.*, **32**, 347 (1963).

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.

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[1] Pouchet, G., *J. Anat. Physiol.*, **12**, 1, 113 (1876).
[2] von Frisch, K., *Pflüg. Arch. ges. Physiol.*, **138**, 319 (1911).
[3] von Frisch, K., and Stetter, H., *Z. vergl. Physiol.*, **17**, 686 (1932).
[4] Healey, E. G., in *Symp. Small Animal Anæsthesia* (Pergamon Press). (in the press).
[5] Kappers, C. U. A., Huber, G. C., and Crosby, E., *The Comparative Anatomy of the Nervous System of Vertebrates* (New York, 1936).

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