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# Epigenetic polymorphism in wild populations of *Mus musculus*

By R. J. BERRY

*Medical Research Council Experimental Genetics Research Unit, University College, Gower Street, London, W.C.*1*

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The difficulty in working with small mammals in nature lies in the lack of distinguishing features in most populations. One way of overcoming this problem in practice is by marking animals on toes or ears, releasing them, and estimating such parameters as 'home-range', population size, age structure, etc., on the basis of those recaptured. This procedure is very time- and labour-consuming, and a method of scoring differences between populations would clearly be of value, both for genetical and ecological studies.

Grüneberg and his collaborators (1950 and later papers) have described over fifty discontinuous (or, as Grüneberg called them, *quasi-continuous*) variants occurring in highly inbred strains of mice. Most of the variants so far recognized are skeletal; the original ones studied were imperfectly formed transverse foramina of the cervical vertebrae, dystopia of the tuberculum anterius of the sixth cervical vertebra, fusions of varying degree between the atlas and axis, and the size of the processus spinosus of the second thoracic vertebra. Weber (1950) showed that the same type of variation can be found in wild caught mice, and Berry & Searle (1963) recognized many morphologically similar variants in the skeletons of nine more species of rodents. Since the incidences of almost all the variants which have been studied are uncorrelated (Truslove, 1961), and changes in incidence can be taken as mutational changes (Deol, Grüneberg, Searle & Truslove, 1957), the sensitivity of detection of differences between strains increases with the number of variants.

This paper is an attempt to extend these findings to the situation in the wild, and particularly to test whether a population sharing a common gene-pool in nature possesses a characteristic pattern of incidences. This situation would be analogous to the polymorphisms existing in human populations with regard to the blood groups, and in *Drosophila* with different chromosome types. Berry & Searle suggested the concept of *epigenetic polymorphism* as being of potential value in the study of the type of variation revealed by the skeletal studies.

The method employed was to collect samples of house mice (*Mus musculus* Linn.) from different localities and compare the incidences of variants found in each sample. The house mouse was chosen because it is technically easier to obtain large numbers

* Present address: Royal Free Hospital School of Medicine, 8, Hunter Street, London, W.C.1.

of this species than of other British rodents. It is, in fact, the third commonest small mammal of English arable land (Southern & Laurie, 1946). The skeletons of over 1200 mice have been classified.

## 1. MATERIAL

Attention was restricted to skeletal variants; since all parts of the body are interdependent, variation in a bone will almost invariably involve other anatomical structures and may, indeed, be secondary to variation in the latter. The skeletons classified in this investigation were prepared by a modification (Searle, 1954*a*) of Luther's (1949) technique for the maceration of skinned, eviscerated and boiled carcasses with the enzyme papain. The bones collected were the skull, vertebrae, girdles (including the clavicles), sternum and long bones. Every skeleton was classified by myself with the exception of those from America which were classified by Dr M. S. Deol. Dr Deol kindly allowed me to classify some of his material and I am satisfied that my classification is in reasonable accord with his. The animals used in this study fall into three groups:

1. Mice from fifteen different corn ricks on a large farm at Odiham, Hampshire.
2. Mice from a further ten localities in the British Isles.
3. Populations from North and South America, Singapore, India and Israel.

The original intention was to collect fifty mice from each locality to be studied. In practice this could not always be realized; if many more were caught only about fifty of them have been utilized. Whenever possible, mice were collected from corn ricks. Ricks are built in the late summer immediately following the harvest, and are threshed during the winter—normally between Christmas and the end of March, which tends to be a slack period on farms. Mice invade the ricks from the hedgerows during the autumn and increase in numbers at a rapid rate. When the ricks are broken down for threshing the mice can fairly easily be caught by hand. By this means a 'population' can be obtained in one day, which by trapping might take several weeks.

### (i) *Roke Farm, Odiham*

This is a large farm (*c.* 2000 acres) on which a large amount of corn is grown. In the winter of 1959–60 there were forty-two ricks scattered over the whole area of the farm, singly or in groups of two ('paired') or three (Fig. 1). The infestation Control Department of the Ministry of Agriculture, Fisheries and Food had chosen this farm for a comparison of different poisoning methods (involving the rodenticide, warfarin) for controlling the rat and mouse population of ricks. Mr F. P. Rowe very kindly let me have the mice that were collected from each rick. Unfortunately (for me), the control methods practised by Mr Rowe and his colleagues were rather efficient. There were fifteen ricks with more than nineteen adult mice in each (Table 1); all other ricks (with the exception of four which were part of another Ministry of Agriculture experiment) had too few mice to be of practical value for the

present purpose. These fifteen ricks included two composed of oats, five of barley and eight of wheat. There were no obvious physical barriers to mice; the road past Readon Farm runs along a shallow valley, the land rising to a height of about 100–150 ft on both sides. Stapely Down Farm is in a side valley (Fig. 1).

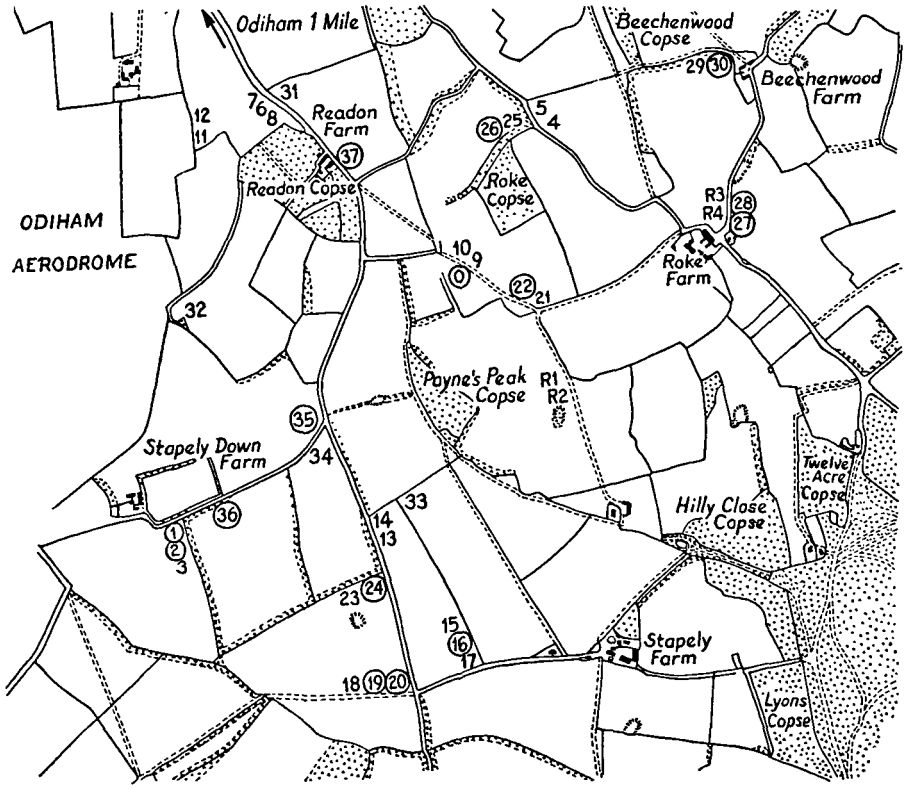


Fig. 1. Map of Roke Farm, Odiham, showing the position of corn ricks (0, 1–37, R1–4) in 1959–60. Ricks with a circle around indicate those from which mice were used. Scale 3 in. to 1 mile. Based upon the Ordnance Survey Map with the sanction of the Controller of H.M. Stationery Office. Crown Copyright reserved.

### (ii) *Other British populations*

These mice came from various sources (Fig. 2) and details of the origin of some of them are obscure. They were all caught in the spring or summer of 1960. Some of these populations are very small, but they have been included for comparison. The Odiham mice have been included as a single population.

a. *Northallerton:* 6 mice from a colony of 100–150 in a two-year-old bay of unthreshed oats on a farm at Low Moor, Northallerton, Yorkshire.
b. *Langtoft:* 12 mice from the village of Langtoft, 5 miles north of Driffield, Yorkshire.

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Table 1. *Percentage incidence of skeletal variants in fifteen corn ricks at Roke Farm, Odiham*

| Rick number | 0 | 1 | 2 | 16 | 19 | 20 | 22 | 24 | 26 | 27 | 28 | 30 | 35 | 36 | 37 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Standing time of rick (weeks) | 80–90 | 25 | 18 | 38 | 40 | 43 | 40 | 39 | 32 | 27 | 27 | 37 | 39 | 28 | 41 |
| Type of corn* | B | O | O | W | W | W | W | W | B | B | B | B | W | W | W |
| Total number of live mice | c. 300 | 33 | 22 | 221 | 128 | 220 | 84 | 54 | 22 | 45 | 32 | 68 | 92 | 47 | 53 |
| Number of ♂♂ classified | 25 | 14 | 8 | 26 | 26 | 26 | 26 | 20 | 12 | 19 | 8 | 24 | 24 | 15 | 15 |
| Number of ♀♀ classified | 25 | 19 | 11 | 26 | 26 | 27 | 26 | 13 | 8 | 13 | 15 | 28 | 27 | 14 | 19 |
| Type of poison treatment† | — | B | A | A | A | A | A | A | C | C | D | C | C | C | C |
| Characters | | | | | | | | | | | | | | | |
| 1. Preorbital foramen double | 3·0 | 5·0 | 13·9 | 4·9 | 13·5 | 8·7 | 7·8 | 7·8 | 12·8 | 8·1 | 9·1 | 9·8 | 8·1 | 1·7 | 7·4 |
| 2. Interfrontal present | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 3. Parted frontals | 58·0 | 69·7 | 84·2 | 46·2 | 59·6 | 43·4 | 40·4 | 42·4 | 60·0 | 71·9 | 52·2 | 51·9 | 74·5 | 65·5 | 61·8 |
| 4. Fused frontals | 2·0 | 3·0 | 0 | 7·7 | 0 | 5·7 | 0 | 0 | 0 | 0 | 0 | 1·9 | 2·0 | 0 | 0 |
| 5. Frontal fontanelle present | 4·0 | 12·1 | 5·3 | 3·8 | 3·8 | 5·7 | 1·9 | 6·1 | 0 | 6·2 | 4·3 | 3·8 | 2·0 | 6·9 | 2·9 |
| 6. Frontal foramen double | 3·0 | 10·8 | 7·9 | 9·6 | 3·8 | 1·9 | 1·9 | 6·1 | 25·0 | 3·2 | 2·2 | 2·9 | 2·9 | 1·7 | 4.4 |
| 7. Maxillary foramen I absent | 10·0 | 10·6 | 28·9 | 15·4 | 8·7 | 22·9 | 22·1 | 9·1 | 17·5 | 15·6 | 21·7 | 21·2 | 8·8 | 6·9 | 17·6 |
| 8. Maxillary foramen I double | 8·0 | 18·2 | 18·4 | 8·7 | 2·9 | 9·5 | 14·4 | 9·1 | 15·0 | 31·2 | 17·4 | 14·4 | 8·8 | 22·4 | 14·7 |
| 9. Maxillary foramen II absent | 45·5 | 43·9 | 60·5 | 68·3 | 60·6 | 41·5 | 49·0 | 50·0 | 25·0 | 60·9 | 71·7 | 63·5 | 73·5 | 72·4 | 57·4 |
| 10. For. pal. maj. double | 1·0 | 3·4 | 5·7 | 0 | 3·8 | 5·9 | 0 | 0 | 0 | 7·9 | 2·3 | 1·0 | 2·9 | 6·9 | 1·5 |
| 11. For. pal. min. ant. absent | 24·0 | 27·9 | 29·4 | 12·5 | 21·2 | 21·6 | 27·9 | 10·9 | 17·5 | 17·7 | 25·0 | 15·4 | 31·0 | 27·6 | 7·4 |
| 12. For. pal. min. post. absent | 31·0 | 25·4 | 29·4 | 25·0 | 33·7 | 33·3 | 48·1 | 23·4 | 22·5 | 49·2 | 38·6 | 26·9 | 40·0 | 41·4 | 42·6 |
| 13. Alae palatinae | 0 | 3·3 | 0 | 0 | 1·9 | 6·0 | 16·5 | 3·1 | 0 | 3·4 | 9·1 | 13·5 | 5·2 | 13·8 | 2·9 |
| 14. For. sphen. med. present | 74·0 | 42·4 | 47·4 | 53·8 | 48·1 | 45·3 | 34·6 | 21·2 | 21·1 | 56·2 | 26·1 | 32·7 | 37·3 | 51·7 | 44·1 |
| 15. For. sphen. lat. ventr. present | 0 | 7·6 | 7·9 | 4·8 | 2·9 | 10·4 | 1·9 | 19·7 | 5·0 | 9·4 | 0 | 18·3 | 4·9 | 0 | 8·8 |
| 16. Processus pterygoideus absent | 15·0 | 4·6 | 0 | 8·7 | 4·9 | 15·3 | 24·5 | 15·9 | 0 | 9·5 | 21·7 | 4·8 | 16·7 | 12·0 | 2·9 |
| 17. Foramen ovale single | 47·0 | 68·9 | 52·9 | 66·3 | 51·0 | 78·4 | 46·1 | 23·4 | 76·9 | 56·5 | 75·6 | 59·6 | 44·6 | 67·2 | 54·4 |
| 18. Foramen infra-ovale double | 6·0 | 13·6 | 7·9 | 9·6 | 2·9 | 5·7 | 5·8 | 1·5 | 18·9 | 4·7 | 0 | 12·5 | 11·8 | 3·4 | 7·4 |
| 19. Foramen pterygoideum double | 1·0 | 4·5 | 2·6 | 3·8 | 1·9 | 1·9 | 0 | 1·5 | 16·2 | 3·2 | 2·2 | 8·7 | 0 | 3·4 | 5·9 |
| 20. Preoptic sutures present | 43·0 | 81·4 | 79·4 | 85·6 | 90·3 | 79·4 | 82·7 | 68·7 | 75·0 | 68·3 | 84·1 | 92·3 | 81·2 | 79·3 | 88·2 |
| 21. Metoptic roots abnormal | 61·0 | 77·8 | 82·4 | 53·8 | 80·6 | 61·0 | 63·7 | 45·3 | 58·3 | 56·7 | 59·1 | 86·5 | 54·9 | 50·0 | 82·4 |
| 22. Foramen hypoglossi single | 72·0 | 73·4 | 75·7 | 85·6 | 80·8 | 81·1 | 76·0 | 75·8 | 65·0 | 67·2 | 84·8 | 81·4 | 65·7 | 87·9 | 80·9 |
| 23. Accessory mental foramen | 37·0 | 40·9 | 57·9 | 26·9 | 26·0 | 28·3 | 26·9 | 22·7 | 35·0 | 26·6 | 23·9 | 28·8 | 20·6 | 22·4 | 38·2 |
| 24. Mandibular foramen double | 22·0 | 18·2 | 13·2 | 14·4 | 6·7 | 3·8 | 12·5 | 4·5 | 15·0 | 17·2 | 10·9 | 23·1 | 7·8 | 3·4 | 1·5 |
| 25. Accessory scapular foramen | 17·0 | 33·3 | 31·6 | 46·2 | 37·5 | 33·0 | 24·0 | 24·2 | 50·0 | 23·4 | 15·2 | 30·8 | 31·4 | 31·0 | 29·4 |
| 26. Fossa olecrani perforata | 36·0 | 66·7 | 65·8 | 45·2 | 54·8 | 71·7 | 38·5 | 68·2 | 82·5 | 43·7 | 56·5 | 59·6 | 67·6 | 62·1 | 58·8 |
| 27. F.t.i. of C. VI | 0 | 3·0 | 5·3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 28. Proc. spin. of Th. I absent | 20·0 | 6·1 | 5·3 | 7·7 | 7·7 | 3·8 | 1·9 | 6·1 | 20·0 | 12·9 | 21·7 | 1·9 | 5·9 | 3·4 | 11·8 |
| 29. Proc. spin. of Th. II absent | 4·0 | 3·0 | 0 | 3·8 | 1·9 | 3·8 | 1·9 | 3·0 | 0 | 6·7 | 0 | 9·6 | 0 | 0 | 0 |
| 30. Dyssymphysis of Th. I | 0 | 0 | 0 | 0 | 3·8 | 0 | 0 | 0 | 15·0 | 0 | 4·3 | 1·9 | 0 | 0 | 0 |
| 31. Dyssymphysis of Th. II | 2·0 | 6·1 | 0 | 0 | 1·9 | 3·8 | 0 | 0 | 0 | 0 | 4·3 | 7·7 | 0 | 0 | 0 |
| 32. Dyssymphysis of Th. X | 2·0 | 0 | 0 | 1·9 | 0 | 3·8 | 0 | 3·0 | 5·0 | 0 | 4·3 | 11·5 | 7·8 | 0 | 5·9 |
| 33. For. acetabuli non-perf. present | 36·7 | 63·1 | 56·8 | 68·9 | 54·8 | 56·2 | 57·7 | 59·4 | 42·5 | 51·6 | 43·5 | 49·0 | 45·1 | 50·0 | 61·8 |
| 34. Third molar missing | 2·0 | 0 | 0 | 0 | 0 | 1·4 | 6·7 | 0·8 | 0 | 0 | 0 | 0 | 0 | 0 | 2·2 |
| 35. Presence of 26 presacral vert. | 82·0 | 75·0 | 84·2 | 98·1 | 90·4 | 86·8 | 92·3 | 87·9 | 100·0 | 81·2 | 91·3 | 96·2 | 96·1 | 93·1 | 76·5 |

* O = oats; B = barley; W = wheat. †A = baited externally at the base only; B = baited externally at the base and internally with metal or cardboard containers; C = hole-baited for rats only; *D = hole-baited for rats, and internally with paper baits.*

c. *Shetland:* 10 mice trapped in corn fields in the Shetland Islands which lie 100 miles north of the Scottish mainland.

d. *Skokholm:* 26 mice trapped mainly on the cliffs and around dry stone walls on the small island of Skokholm which lies two miles west of the Pembrokeshire (South Wales) coast.

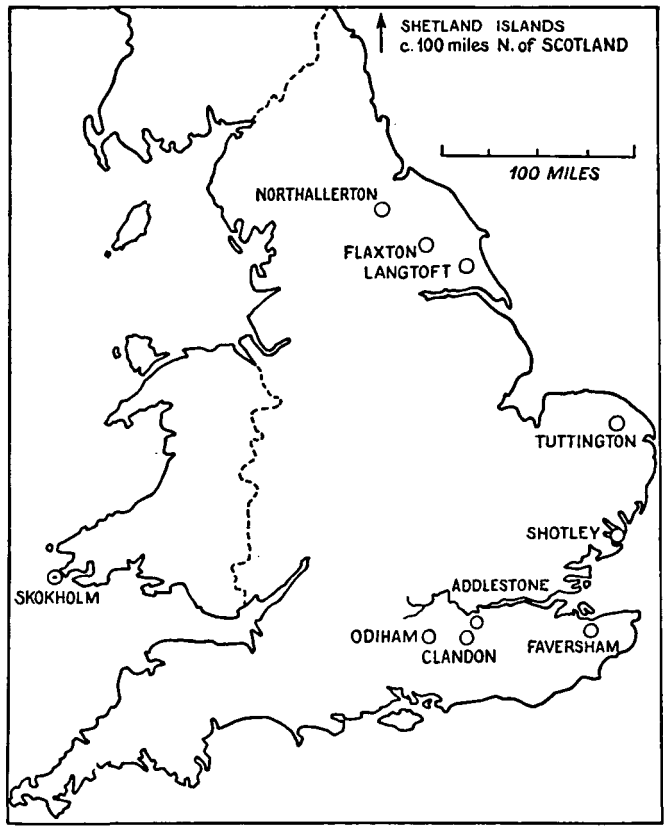


Fig. 2. England and Wales showing the localities from which mice were obtained.

The rest of the populations were all collected at threshings:

e. *Tuttington:* 27 mice from a wheat rick on a farm near Tuttington, 12 miles north of Norwich.

f. *Shotley:* 45 mice from a wheat rick on a farm at Shotley, on the Suffolk coast near Harwich.

g. *Flaxton:* 52 mice from 145 collected from an oat rick at Wigginton, Flaxton, York.

h. *Faversham:* 52 mice from 136 collected from a wheat rick at Gosmere, near Faversham in Kent.

i. *Clandon:* 50 mice from 137 collected from a wheat rick, the second of a row of four outside the village of West Clandon, near Guildford in Surrey.

j. *Addlestone:* 51 mice from a wheat rick, the middle one of a row of five on a farm close to the suburbs of London at Addlestone, near Egham in Surrey.

### (iii) *World-wide populations*

a, b. *South America:* Harland (1958) described mice from Peru. These mice were from five localities: 21 from the low altitude villages of Ancon and Nana, and 27 from villages at around 15,000 ft. in the Andes (Morococha, Huaron and Ticlio). As the numbers from individual villages are small, I have grouped the animals into a 'low-altitude' and a 'high-altitude' population. The sole justification for this is Harland's statement (referring to the Huaron mice), they 'were of the same type as those from Morococha and Ticlio'.

c, d, e, f. *North America:* These four populations have been described by Deol (1958). They include 30 mice from Great Gull Island, New York, 36 from Norwich, Vermont, 51 from Storrs, Connecticut and 76 laboratory bred mice from a cross between New York City and Philadelphia stocks (R-3).

g. *India:* 24 mice trapped in grain shops in Delhi in the autumn of 1959.

h. *Singapore:* 48 mice caught in 1957–8, 'mainly in the Kandang Kuban and Tan Tock Seng Hospitals and in the coffee shops in the Bukit Tinah Road area. The range of bodily dimensions fit fairly well with the description of *Mus musculus castaneus*, a commensal form of *M. m. wagneri*' (Searle, 1962). They have been partially described by Searle (1960).

i. *Israel:* 8 specimens of *M. m. praetextus* bred in the Genetics Dept., Cambridge, from mice obtained by Dr D. Michie.

Three British populations (Odiham, Skokholm and Faversham) were used for comparison.

## 2. VARIANTS CLASSIFIED

Berry & Searle (1963) have reviewed and briefly described fifty-five minor variants of the skeleton, most of them previously reported by Grüneberg or one of his co-workers. In the present study, it was desired to make use of as many characters as feasible to provide as sensitive an estimate as possible of inter-population differences. However, many of the described characters were felt to be unsuitable for wild animals because of the heterogeneity that such populations must exhibit in respect of age. For this reason all fusions between bones have been omitted, except fusion between frontals. Many characters which occur at a fairly high frequency in some inbred strains were rare or absent in my material. For example, inframaxillary crest is present in *c.* 90% of C57BL mice (Deol, 1955) but it was only found in one out of the 585 mice from Odiham, and hence was of little value for comparative purposes. On the other hand several other variants were noted which had sufficiently varying incidences in different populations to be valuable. Although all the previously described variants were scored, only thirty-five have been used

in this study (Figs. 3–12; for references see Berry & Searle). A short description is given for those not previously described.

1. Preorbital foramen double.
2. Interfrontal present.
3. Parted frontals.

FIGS. 3–12. DRAWINGS OF ELEMENTS OF THE MOUSE SKELETON TO SHOW THE VARIANTS SCORED. VARIANT NO. 26 (*fossa olecrani perforata*) IS ILLUSTRATED BY BERRY & SEARLE (1963)

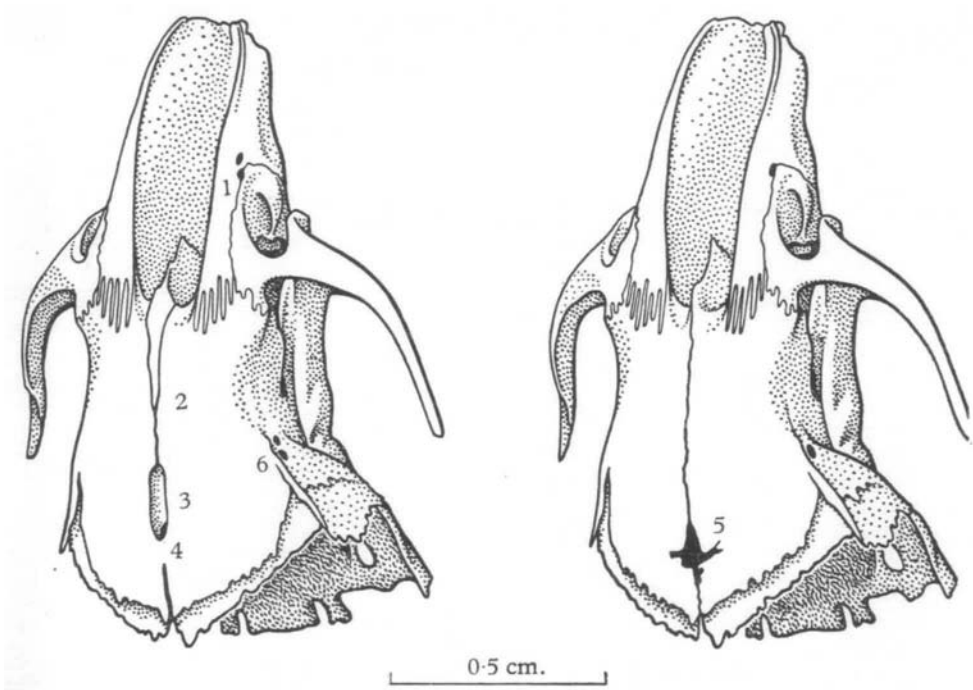


Fig. 3. Dorsal and right lateral surfaces of the skull:

1. Preorbital foramen double
2. Interfrontal
3. Parted frontals
4. Fused frontals
5. Frontal fontanelle
6. Frontal foramen double

4. Fused frontals.
5. Frontal fontanelle present.
6. Frontal foramen double.
7. Maxillary foramen I absent.
8. Maxillary foramen I double.
9. Maxillary foramen II absent. In the house mouse there are frequently two pairs of maxillary foramina on the ventral surface of the maxilla, one on the inter-alveolar margin anterior to the tooth row (maxillary foramen I) and



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one more variable in position but situated laterally on the zygomatic arch (maxillary foramen II). Maxillary foramen I may be single, double or absent; maxillary foramen II may be present or absent, or, very rarely, double.

10. Foramen palatinum majus double.
11. Foramen palatinum minus anterius absent.
12. Foramen palatinum minus posterius absent. The two pairs of minor palatine foramina have been called anterior and posterior, and may be present or absent.

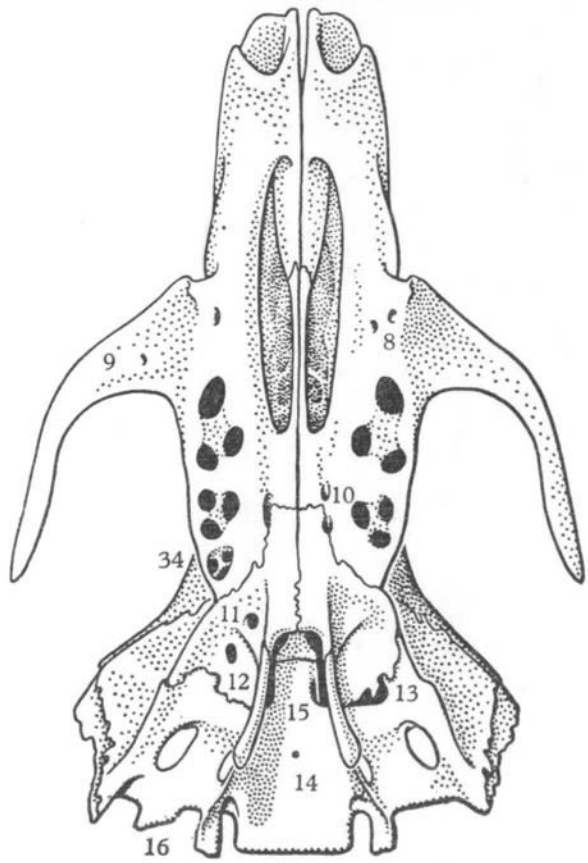


Fig. 4. Ventral surface of the skull:

8. Maxillary foramen I double
9. Maxillary foramen II (absent on the other side)
10. Foramen palatinum majus double
11. Foramen palatinum minus anterius (absent on the other side)
12. Foramen palatinum minus posterius (absent on the other side)
13. Alae palatinae
14. Foramen sphenoidale medium
15. Foramen sphenoidale laterale ventrale
16. Processus pterygoideus (absent on the other side)
34. Socket of third molar (missing on the other side)

13. Alae palatinae.
14. Foramen sphenoidale medium present.
15. Foramen sphenoidale laterale ventrale present. On the lateral side of the sphenoid bone there are occasionally large foramina, usually anterior to the foramen sphenoidale medium. These have been classified as present if visible from the ventral surface of the skull.

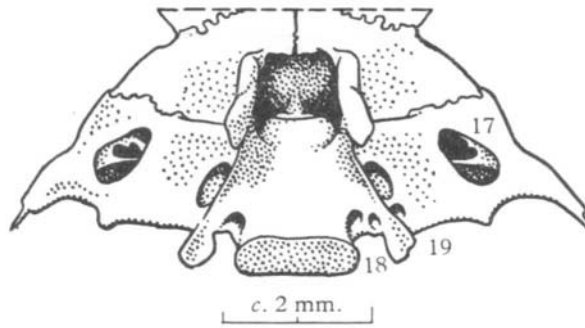


Fig. 5. Ventral view of the sphenoidal region of the skull:

17. Foramen ovale single
18. Foramen infra-ovale double
19. Foramen pterygoideum double

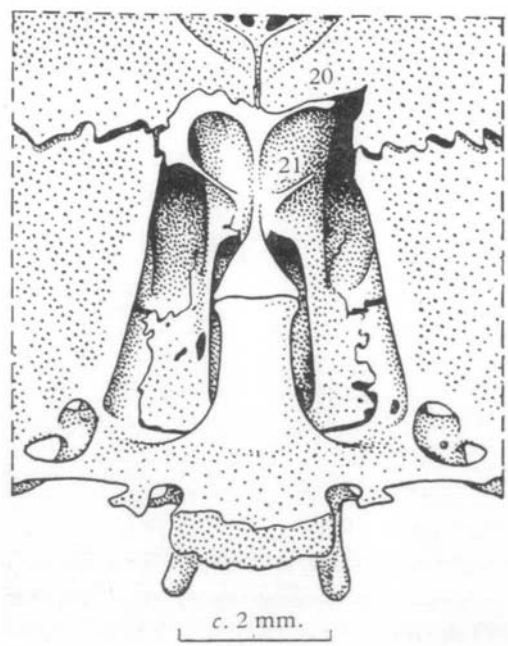


Fig. 6. Dorso-cranial view of the internal bones of the skull:

20. Preoptic sutures
21. Metoptic roots abnormal

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16. Processus pterygoideus absent. This has been scored as complete absence versus presence.
17. Foramen ovale single.
18. Foramen infra-ovale double.

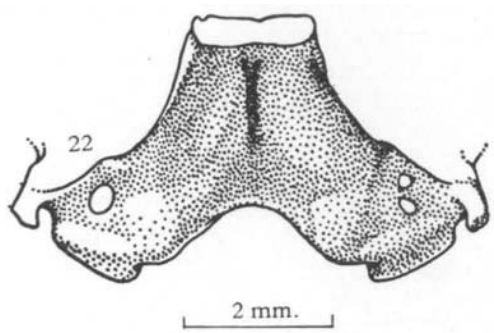


Fig. 7. Obliquely ventral view of the occiput:
22. Foramen hypoglossi single

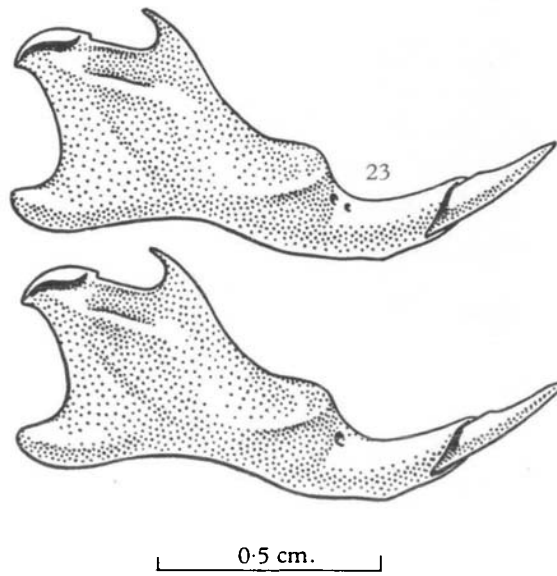


Fig. 8. Lateral view of the right mandible:
23. Accessory mental foramen

19. Foramen pterygoideum double. Medial to the foramen ovale are two foramina running into the sphenoid visible from the ventral surface. The one lateral to the petrosal process has been named the foramen pterygoideum, that on the inside the foramen infra-ovale. The normal state of these foramina seems to be single, but they are sometimes divided into two.
20. Preoptic sutures present.
21. Metoptic roots abnormal.

22. Foramen hypoglossi single.
23. Accessory mental foramen.
24. Mandibular foramen double. The mandibular foramen is occasionally double.

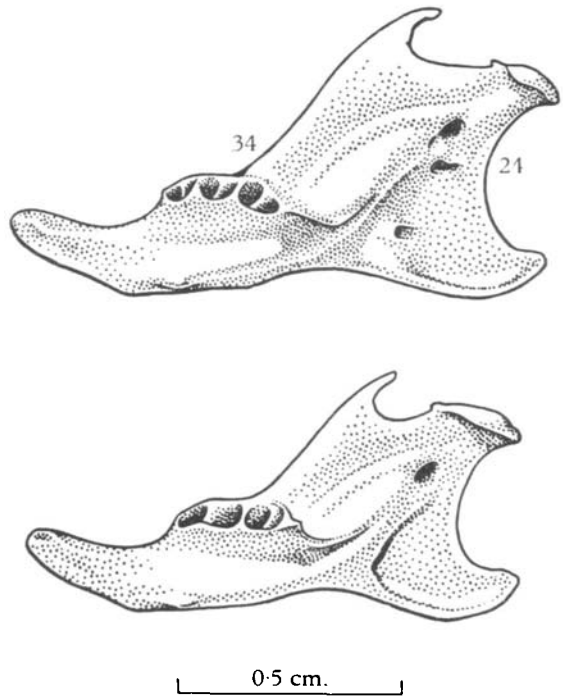


Fig. 9. Medial view of right mandible:
24. Mandibular foramen double
34. Socket of third molar (missing in the lower illustration)

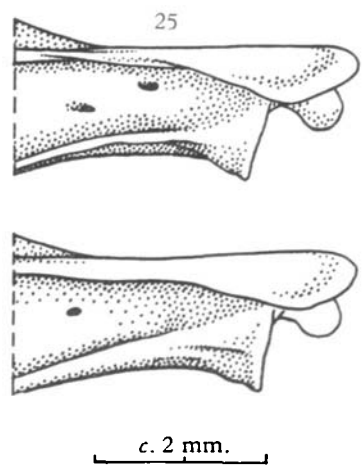


Fig. 10. Lateral surface of the coracoid end of right scapula:
25. Accessory scapular foramen

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25. Accessory scapular foramen.
26. Fossa olecrani perforata.
27. Foramina transversaria imperfecta of the sixth cervical vertebra.
28. Processus spinosus of the 1st thoracic vertebra absent (Grüneberg, 1950).
29. Processus spinosus of the second thoracic vertebra absent. This has been scored as complete absence versus presence.
30. Dyssymphysis of Th. I.

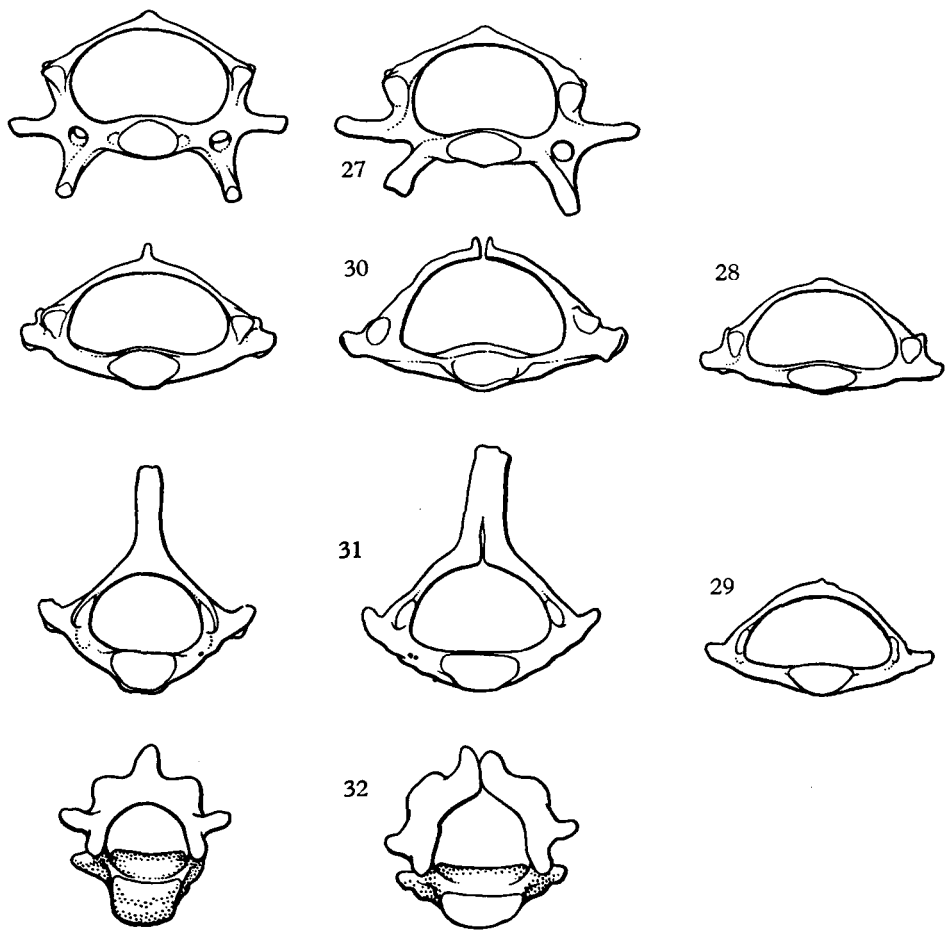


Fig. 11. Vertebrae C. VI, Th. I, II, X:
27. F.t.i. of C. VI
28. Processus spinosus of Th. I absent
29. Processus spinosus of Th. II absent
30. Dyssymphysis of Th. I
31. Dyssymphysis of Th. II
32. Dyssymphysis of Th. X

31. Dyssymphysis of Th. II.
32. Dyssymphysis of Th. X.
33. Foramen acetabuli non-perforans present. This is not the same variant as foramen acetabuli perforans. Frequently there are foramina in the fossa acetabuli leading into the bone. The presence of one or more of such foramina has been named 'foramen acetabuli non-perforans present'.

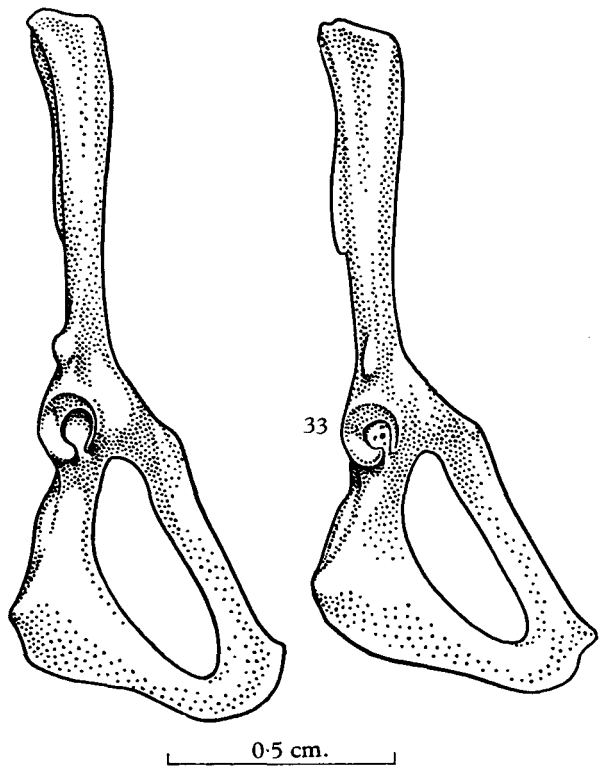


Fig. 12. Lateral view of right half of pelvic girdle:
33. Foramen acetabuli non-perforans present

34. Third molar missing.
35. Presence of twenty-six presacral vertebrae. Variation may occur at both the thoracico-lumbar and the lumbo-sacral borders. The variant chosen is insensitive in that it conceals a good deal of the variation. However, detection of variation at the thoracico-lumbar border requires much more careful examination than was in fact given, and hence the choice. As most of the skeletons were prepared by me, I am reasonably confident that vertebrae were not lost.

Deol (1958) and Harland (1958) scored foramen transversaria imperfecta of the fifth cervical vertebra and inflexum and absence of the tuberculum anterius of the sixth cervical. These characters have been included in populations from different parts of the globe classified by me.

Table 2. *Percentage incidence of skeletal variants in eleven populations from the British Isles*

| | Northallerton | Langtoft | Shetland | Skokholm | Tuttington | Shotley | Flaxton | Faversham | Clandon | Odiham | Addlestone |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Number of ♂♂ classified | 5 | 6 | 6 | 16 | 20 | 19 | 26 | 26 | 25 | 288 | 27 |
| Number of ♀♀ classified | 1 | 6 | 4 | 10 | 7 | 26 | 26 | 26 | 25 | 297 | 24 |
| Characters | | | | | | | | | | | |
| 1. Preorbital foramen double | 0 | 4·5 | 5·0 | 7·7 | 4·3 | 11·1 | 14·4 | 2·9 | 6·3 | 7·9 | 2·9 |
| 2. Interfrontal present | 0 | 0 | 10·0 | 46·2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 3. Parted frontals | 50·0 | 33·3 | 30·0 | 61·5 | 55·6 | 77·8 | 32·7 | 73·1 | 58·0 | 56·9 | 23·5 |
| 4. Fused frontals | 16·7 | 0 | 30·0 | 0 | 14·8 | 8·9 | 3·8 | 3·8 | 2·0 | 1·9 | 7·8 |
| 5. Frontal fontanelle present | 0 | 0 | 0 | 0 | 0 | 2·2 | 0 | 0 | 0 | 4·4 | 0 |
| 6. Frontal foramen double | 0 | 26·1 | 70·0 | 3·8 | 5·7 | 4·4 | 1·0 | 5·8 | 4·0 | 5·0 | 8·8 |
| 7. Maxillary foramen I absent | 0 | 20·8 | 35·0 | 38·5 | 15·1 | 15·6 | 17·5 | 23·1 | 15·2 | 15·4 | 14·7 |
| 8. Maxillary foramen I double | 16·7 | 0 | 5·0 | 21·2 | 18·9 | 33·3 | 16·5 | 7·7 | 22·2 | 12·9 | 20·6 |
| 9. Maxillary foramen II absent | 50·0 | 47·8 | 15·0 | 25·0 | 38·5 | 37·8 | 46·6 | 51·9 | 61·6 | 56·7 | 47·1 |
| 10. For. pal. maj. double | 0 | 0 | 10·0 | 7·7 | 8·2 | 3·4 | 2·1 | 0 | 2·2 | 2·6 | 14·7 |
| 11. For. pal. min. ant. absent | 25·0 | 22·2 | 20·0 | 7·7 | 24·4 | 33·3 | 21·6 | 22·1 | 12·1 | 21·0 | 21·6 |
| 12. For. pal. min. post. absent | 36·4 | 23·5 | 30·0 | 26·9 | 52·8 | 32·6 | 39·8 | 35·6 | 31·8 | 34·3 | 48·0 |
| 13. Alae palatinae | 0 | 21·1 | 55·0 | 1·9 | 3·7 | 4·5 | 1·3 | 1·0 | 3·8 | 5·6 | 1·0 |
| 14. For. sphen. med. present | 33·3 | 41·7 | 90·0 | 73·1 | 42·3 | 93·3 | 39·2 | 65·4 | 62·0 | 43·8 | 68·6 |
| 15. For. sphen. lat. ventr. present | 16·7 | 37·5 | 5·0 | 32·7 | 17·3 | 17·8 | 34·7 | 33·7 | 17·0 | 6·8 | 35·3 |
| 16. Processus pterygoideus absent | 0 | 9·1 | 0 | 1·9 | 2·1 | 1·1 | 2·1 | 7·7 | 7·6 | 11·2 | 3·9 |
| 17. Foramen ovale single | 63·6 | 15·0 | 45·0 | 32·7 | 24·3 | 24·4 | 29·2 | 46·2 | 41·4 | 56·8 | 23·5 |
| 18. Foramen infra-ovale double | 33·3 | 13·0 | 15·0 | 23·1 | 17·3 | 10·0 | 16·7 | 14·4 | 17·2 | 7·4 | 18·6 |
| 19. Foramen pterygoideum double | 8·3 | 8·7 | 0 | 3·8 | 12·0 | 2·2 | 13·4 | 2·9 | 9·7 | 3·3 | 2·0 |
| 20. Preoptic sutures present | 58·3 | 72·2 | 75·0 | 96·2 | 61·5 | 81·4 | 48·6 | 74·0 | 75·6 | 78·9 | 52·0 |
| 21. Metoptic roots abnormal | 66·7 | 50·0 | 65·0 | 84·6 | 54·2 | 67·9 | 73·2 | 70·2 | 60·5 | 65·0 | 52·9 |
| 22. Foramen hypoglossi single | 70·0 | 85·0 | 50·0 | 73·1 | 58·8 | 68·9 | 76·8 | 76·0 | 54·7 | 76·9 | 57·8 |
| 23. Accessory mental foramen | 33·3 | 41·7 | 20·0 | 25·0 | 31·5 | 35·6 | 22·1 | 9·7 | 45·0 | 29·6 | 17·6 |
| 24. Mandibular foramen double | 0 | 4·3 | 0 | 21·2 | 0 | 25·8 | 1·9 | 4·9 | 8·1 | 11·8 | 11·8 |
| 25. Accessory mental foramen | 50·0 | 66·7 | 26·3 | 63·5 | 84·9 | 76·4 | 71·0 | 62·5 | 46·9 | 30·6 | 70·6 |
| 26. Fossa olecrani perforata | 33·3 | 41·7 | 90·0 | 76·9 | 83·3 | 58·9 | 44·2 | 65·4 | 32·3 | 56·8 | 49·0 |
| 27. F.t.i. of C. VI | 0 | 0 | 0 | 9·6 | 0 | 0 | 1·0 | 0 | 0 | 0·3 | 0 |
| 28. Proc. spin. of Th. I absent | 0 | 33·3 | 40·0 | 0 | 14·8 | 13·3 | 21·6 | 11·5 | 8·2 | 8·2 | 10·0 |
| 29. Proc. spin. of Th. II absent | 0 | 8·3 | 0 | 0 | 0 | 0 | 0 | 0 | 4·1 | 2·9 | 0 |
| 30. Dyssymphysis of Th. I | 0 | 8·3 | 0 | 76·9 | 7·4 | 0 | 0 | 1·9 | 4·0 | 1·4 | 2·0 |
| 31. Dyssymphysis of Th. II | 0 | 0 | 0 | 19·2 | 3·7 | 0 | 2·0 | 5·8 | 2·0 | 1·9 | 3·9 |
| 32. Dyssymphysis of Th. X | 0 | 8·3 | 0 | 57·7 | 0 | 6·7 | 5·9 | 5·8 | 2·0 | 3·2 | 0 |
| 33. For. acetabuli non-perf. present | 41·7 | 95·5 | 50·0 | 46·9 | 56·0 | 37·1 | 56·9 | 43·3 | 44·0 | 53·4 | 60·4 |
| 34. Third molar missing | 0 | 4·2 | 0 | 0 | 1·9 | 0 | 0 | 0 | 1·0 | 1·1 | 0 |
| 35. Presence of 26 presacral vert. | 83·3 | 50·0 | 100·0 | 100·0 | 88·9 | 95·6 | 90·4 | 76·9 | 78·0 | 89·2 | 90·0 |

## 3. METHODS OF ANALYSIS

The percentage incidences of the thirty-five variants in the various British populations are given in Tables 1 and 2. Deol (1958) and Harland (1958) used a smaller selection of characters in classifying their American material and Table 3 only includes eighteen variants. The incidence of bilateral variants is based on the number of *sides* on which the character occurs; this use of the total incidence (rather than unilateral and bilateral figures) has been made to reduce the data to manageable proportions. In a few cases damage to a specimen means that the percentage incidence is based on a lower total than that recorded for the population.

The data were subjected to two methods of analysis. The distribution of ricks at Odiham made it possible to investigate the effect of the relative position of the ricks, and also the effect of maternal diet on the variation.

The whole data from Odiham can be put in the form of a contingency table for each character:

| | Character | |
|---|---|---|
| Rick No. | Present | Absent |
| 1 | ... | ... |
| ... | ... | ... |
| ... | ... | ... |
| ... | ... | ... |
| 37 | ... | ... |

This analysed according to the usual method would give a heterogeneity $\chi^2$ with 14 degrees of freedom. However, this $\chi^2$ can be subdivided into three components, 'between paired ricks (within diets)', 'between non-paired ricks within diets' and 'between diets', which yield $\chi^2$ with respectively 3, 9, and 2 degrees of freedom. The numerical work is made much simpler by the use of Woolf's (1957) formula, instead of the conventional formula for $\chi^2$, and this has the additional advantage that $\chi^2$ calculated by Woolf's method is exactly additive, the three components totalling to the $\chi^2$ with 14 d.f. for the whole table. For example, the $\chi^2$ for 'between non-paired ricks within diets' is obtained by writing down a contingency table for each diet. The $\chi^2$ for these tables is then found by Woolf's procedure, and the values added together give a $\chi^2$ with 9 d.f. 'between non-paired ricks within diets'. Similarly for the $\chi^2$ 'between diets' we take the total numbers in all ricks within each diet including the two ricks in each pair added together and treated as a single rick, getting a single table:

| Diet | Present | Absent |
|---|---|---|
| Oats | ... | ... |
| Wheat | ... | ... |
| Barley | ... | ... |

and this gives, as usual, a $\chi^2$ with 2 d.f.

Table 3. *Percentage incidence of skeletal variants in twelve populations from scattered places on the globe*

| | South America Low altitude | South America High Altitude | North America Great Gull Island | North America Norwich, Vermont | North America Storrs, Ct. | North America R.3 | United Kingdom Odiham | United Kingdom Skokholm | United Kingdom Faversham | India Delhi | Singapore | Israel |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Number of animals classified | 21 | 27 | 30 | 36 | 51 | 76 | 585 | 26 | 52 | 24 | 48 | 8 |
| | | | | | | Characters | | | | | | |
| 1. Parted frontals | 9·5 | 11·1 | 100·0 | 61·1 | 43·1 | 42·1 | 56·9 | 61·5 | 73·1 | 12·5 | 12·5 | 0 |
| 2. Fused frontals | 14·3 | 11·1 | 20·0 | 13·9 | 0 | 13·2 | 1·9 | 0 | 3·8 | 4·2 | 2·1 | 0 |
| 3. Interfrontal present | 14·3 | 25·9 | 26·7 | 22·2 | 5·9 | 1·3 | 0 | 46·2 | 0 | 8·3 | 4·2 | 0 |
| 4. Alae palatinae | 2·4 | 1·9 | 0 | 26·4 | 14·7 | 42·8 | 5·6 | 1·9 | 1·0 | 0 | 8·4 | 0 |
| 5. For. sphen. med. present | 28·6 | 44·4 | 66·7 | 33·3 | 27·5 | 57·9 | 43·8 | 73·1 | 65·4 | 58·3 | 52·1 | 62·5 |
| 6. Processus pterygoideus absent | 0 | 5·6 | 3·3 | 16·6 | 3·9 | 1·9 | 1·9 | 11·2 | 7·7 | 14·6 | 5·2 | 43·7 |
| 7. Foramen ovale single | 38·1 | 53·7 | 40·0 | 26·4 | 28·4 | 14·5 | 56·8 | 32·7 | 46·2 | 70·8 | 64·6 | 37·5 |
| 8. Preoptic sutures present | 21·4 | 13·0 | 8·6 | 27·8 | 31·3 | 41·5 | 78·9 | 96·2 | 74·0 | 16·7 | 71·3 | 87·5 |
| 9. Metoptic roots abnormal | 21·4 | 5·6 | 6·9 | 11·1 | 4·9 | 25·0 | 65·0 | 84·6 | 70·2 | 16·7 | 57·4 | 100·0 |
| 10. Foramen hypoglossi single | 77·5 | 24·1 | 48·3 | 62·5 | 56·9 | 62·5 | 73·1 | 76·9 | 76·0 | 58·3 | 56·5 | 85·7 |
| 11. Accessory mental foramen | 0 | 0 | 1·7 | 0 | 1·0 | 2·6 | 25·0 | 29·6 | 9·7 | 12·5 | 15·8 | 0 |
| 12. F.t.i. of C. V | 0 | 0 | 1·7 | 0 | 0 | 19·1 | 0·1 | 1·9 | 0 | 0 | 2·1 | 0 |
| 13. F.t.i. of C. VI | 0 | 0 | 0 | 0 | 0 | 0 | 0·3 | 9·6 | 0 | 2·1 | 9·8 | 12·5 |
| 14. Tuberculum anterius inflexum | 0 | 0 | 3·3 | 0 | 0 | 0·7 | 0 | 0 | 0 | 2·1 | 5·4 | 0 |
| 15. Tuberculum anterius absent | 0 | 1·9 | 0 | 0 | 0 | 0·7 | 0·3 | 1·9 | 1·0 | 0 | 0 | 0 |
| 16. Dyssymphysis of Th. II | 0 | 0 | 3·3 | 0 | 0 | 0 | 1·9 | 19·2 | 0 | 2·1 | 4·2 | 0 |
| 17. Dyssymphysis of Th. X | 0 | 0 | 3·3 | 8·3 | 0 | 0 | 3·2 | 57·7 | 5·8 | 0 | 0 | 50·0 |
| 18. Upper third molar missing | 0 | 18·5 | 0 | 0 | 0 | 0 | 1·9 | 0 | 0 | 0 | 9·4 | 0 |

An inspection of the results obtained shows that in many cases the 'between ricks within diets' $\chi^2$ is highly significant, indicating that the numbers are subject to a 'geographical' variability rather greater than would be expected if the numbers were subject to mere random fluctuation on the basis of the binomial distribution. The question therefore arises as to whether the significantly high values obtained for 'between pairs' and 'between diets' may not also be adequately explained as being due to this 'geographical' variation, and not really due to any appreciable effect of the diets themselves. To test this, the $\chi^2$ in this decomposition have been considered as analogous to the sums of squares in an analysis of variance. Taking 'between pairs' as analogous to residual, $F = (\chi^2_9/9)/(\chi^2_3/3)$ with (9, 3) d.f. This was never significant at the 5% probability level, suggesting that there was more variability between non-paired than between paired ricks. To test for differences between diets, it seems reasonable to compare $\chi^2_2/2$ with $(\chi^2_3+\chi^2_9)/(3+9)$ in an F-test, and this was significant in the case of five characters, three of these five being due to the single pair of oat ricks. This suggests that the apparent differences between diets may be almost entirely due to whatever is causing the 'geographical' variation, and not to the effect of diets themselves. The exact mathematical validity of using a $\chi^2$/d.f. as a 'mean square' in the analysis of variance will depend in a complicated way on exactly how the factors causing the variation are distributed, but it seems plausible that it will give a reasonably reliable guide to general conclusions.

Further calculations were concerned with obtaining measures of distinctiveness or divergence between populations. The method was devised by C. A. B. Smith and is set out by Grewal (1962*a*).

The percentage incidence of each character was transformed into angular values, thus making the part of the variance due to errors of sampling independent of the incidence of the character. The angular value $\theta$ corresponding to the percentage incidence $p$ was defined by:

$$\theta = \sin^{-1}(1-2p)$$

measured in radians. This has the advantage over the more usual angular transformation ($\theta = \sin^{-1}p$ in degrees) that the variance of $\theta$ in a sample of $n$ is nearly $1/n$ independently of the value of $\theta$, instead of $820{\cdot}7/n$. Suppose that in two large populations the actual incidences of a character are $P_1$ and $P_2$, and the corresponding angular transformations are $\theta_1$ and $\theta_2$, then we can take $(\theta_1-\theta_2)^2$ as a convenient (but arbitrary) 'measure of divergence' between the populations. In practice we shall have samples from these populations of size $n_1$ and $n_2$, say, with observed character incidences $p_1$ and $p_2$, and corresponding angular transformations $\theta_1$ and $\theta_2$. In that case we take as the measure of divergence (or distinctiveness) in the samples

$$(\theta_1-\theta_2)^2-(1/n_1+1/n_2)$$

This is an estimate of $(\theta_1-\theta_2)^2$ in the populations; the term $(1/n_1+1/n_2)$ which is subtracted represents the additional variance ($V$) due to random sampling fluctuations which will go to swell the value of $(\theta_1-\theta_2)^2$. This computation has the additional property that, since $\theta_1$ has variance $1/n_1$ and $\theta_2$ has variance $1/n_2$, then

$\theta_1 - \theta_2$ has variance $1/n_1 + 1/n_2 = V$, and where there is no real difference between the large populations from which the two samples are drawn, the observed $\theta_1 - \theta_2 = D$ will be a nearly normal variate with mean zero and variance $V$. Thus

$$(\theta_1 - \theta_2)^2/V$$

will be approximately distributed as $\chi^2$ with one degree of freedom. The 'measure of distinctiveness' is accordingly distributed as $V(\chi_1^2 - 1)$; and hence it will be significant at (e.g.) the 0·05 level if it is greater than $3V$, and at the 0·01 level if it is greater than $6V$. The variance of $D^2$ will be approximately $4D^2 \times$ variance of $D = 4D^2(1/n_1 + 1/n_2)$ for each character, and $= 4(1/n_1 + 1/n_2) \sum D^2$ for populations. A rough estimate of this variance will be given by

$$4(1/n_1 + 1/n_2) \sum [(\theta_1 - \theta_2)^2 - (1/n_1 + 1/n_2)]$$
$$= 4V \times \text{summed measures of divergence for each character between two populations.}$$

Thus the mean 'measure of distinctiveness' between two populations is a quantitative expression of the separation, and an individual population can be further characterized by calculating its mean distinctiveness from those populations with which it is compared.

This method assumes that all variants have an equal effect on fitness, and therefore can be summed legitimately. This assumption is almost certainly incorrect, but since one gene difference may affect a number of characters (Grüneberg, 1955), it is hoped that a summation of all differences is a reasonable estimate of genetic divergence between the populations concerned.

## 4. RESULTS

### (i) *Odiham*

It was important to separate the extrinsic (or ecological) components of variation from the intrinsic (or developmental) ones. The most obvious ecological variables in the Odiham situation were the food of the mice (i.e. the type of corn of the rick from which they came); the geographical location of the ricks—their position relative to other ricks and to buildings, open country, etc.; the number of animals that invade each rick; and the effects of different intensities of selection for resistance to warfarin. The age composition of different ricks was unknown; sex was scored, but was assumed to have no effect and ignored.

The results of the analysis are set out in Table 4. As previously stated, in three out of the five characters (nos. 23, 27 and 33) which show significant differences between diets at the 5% probability level, this difference is the effect of the single pair of oat ricks (there are only four occurrences of f.t.i. of C. VI, two in each oat rick). Although a small 'diet effect' cannot be excluded, it is certainly true to say that the main source of variability between the ricks is a 'geographical' one.

Table 4. *Heterogeneity $\chi^2$ values between groupings of Odiham ricks*

| | Diets | | Between paired ricks | | Between non-paired ricks | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | $\chi^2_4$ | $P$ | $\chi^2_3$ | $P$ | $\chi^2_9$ | $P$ | $F_{3,9}$ | $P$ | $F_{9,12}$ | $P$ |
| 1. Preorbital foramen double | 0 | | 3·5 | | 10·3 | | 1·04 | | 0 | |
| 2. Interfrontal present | 0 | | 0 | | 0 | | 0 | | 0 | |
| 3. Parted frontals | 9·2 | ++ | 6·4 | | 19·2 | + | 1·00 | | 2·16 | |
| 4. Fused frontals | 0·8 | | 5·0 | | 11·8 | | 1·27 | | 0·29 | |
| 5. Frontal fontanelle present | 2·8 | | 1·1 | | 3·9 | | 0·86 | | 3·40 | |
| 6. Frontal foramen double | 5·3 | | 0·6 | | 28·6 | ++++ | 0·06 | | 1·10 | |
| 7. Maxillary foramen I absent | 1·0 | | 14·4 | +++ | 17·2 | + | 2·51 | | 0·20 | |
| 8. Maxillary foramen I double | 9·2 | ++ | 7·2 | | 11·4 | | 1·89 | | 2·97 | |
| 9. Maxillary foramen II absent | 3·7 | | 11·8 | +++ | 41·5 | ++++ | 0·85 | | 0·42 | |
| 10. For. pal. maj. double | 1·1 | | 2·6 | | 16·2 | | 0·48 | | 0·35 | |
| 11. For. pal. min. ant. absent | 3·5 | | 0·8 | | 31·3 | ++++ | 0·08 | | 0·66 | |
| 12. For. pal. min. post. absent | 3·5 | | 1·5 | | 20·5 | ++ | 0·22 | | 0·95 | |
| 13. Alae palatinae | 3·6 | | 5·8 | | 58·8 | ++++ | 0·30 | | 0·33 | |
| 14. For. sphen. med. present | 0·7 | | 5·4 | | 36·4 | ++++ | 0·45 | | 0·10 | |
| 15. For. sphen. lat. ventr. present | 0·8 | | 12·0 | +++ | 55·5 | ++++ | 0·65 | | 0·07 | |
| 16. Proc. pterygoideus absent | 12·1 | +++ | 12·2 | +++ | 35·1 | ++++ | 1·04 | | 1·54 | |
| 17. Foramen ovale single | 4·0 | | 23·6 | ++++ | 60·7 | ++++ | 1·17 | | 0·28 | |
| 18. For. infra-ovale double | 3·8 | | 4·0 | | 15·1 | | 0·80 | | 1·19 | |
| 19. For. pterygoideum double | 7·9 | +++ | 0·4 | | 26·1 | +++ | 0·04 | | 1·79 | |
| 20. Preoptic sutures present | 18·0 | ++++ | 8·4 | + | 72·9 | ++++ | 0·35 | | 1·33 | |
| 21. Metoptic roots abnormal | 12·5 | +++ | 9·9 | ++ | 47·9 | ++++ | 0·62 | | 1·30 | |
| 22. For. hypoglossi single | 3·5 | | 2·0 | | 22·5 | +++ | 0·27 | | 0·49 | |
| 23. Accessory mental foramen | 17·8 | ++++ | 3·3 | | 9·1 | | 1·09 | | 8·64 | +++ |
| 24. Mandibular for. double | 32·8 | ++++ | 2·4 | | 17·5 | + | 0·37 | | 9·88 | +++ |
| 25. Access. scapular foramen | 12·3 | +++ | 1·9 | | 30·3 | ++++ | 0·19 | | 2·29 | |
| 26. Fossa olecrani perforata | 7·0 | + | 8·5 | + | 57·6 | ++++ | 0.44 | | 0·64 | |
| 27. F.t.i. of C. VI | 19·5 | ++++ | 0·3 | | 0 | | | | 325·0 | ++++ |
| 28. Proc. spin. of Th. I absent | 9·1 | ++ | 1·5 | | 15·9 | | 0·28 | | 3·14 | |
| 29. Proc. spin. of Th. II absent | 4·4 | | 3·5 | | 9·3 | | 1·14 | | 2·06 | |
| 30. Dyssymphysis of Th. I | 4·3 | | 4·5 | | 10·9 | | 1·24 | | 1·68 | |
| 31. Dyssymphysis of Th. II | 5·2 | | 3·8 | | 3·8 | | 3·02 | | 4·13 | + |
| 32. Dyssymphysis of Th. X | 5·3 | | 4·4 | | 12·9 | | 1·03 | | 1·84 | |
| 33. For. acetabuli non-perf. present | 16·5 | ++++ | 0·9 | | 17·0 | + | 0·16 | | 5·54 | ++ |
| 34. Third molar missing | 8·7 | ++ | 4·1 | | 44·5 | ++++ | 0·28 | | 1·07 | |
| 35. Pres. of 26 presacral vert. | 5·8 | | 2·0 | | 23·6 | | 0·26 | | 1·36 | |

+ represents $P$=2–5%; ++ represents $P$ = 1–2%; +++ represents $P$ = 0·1–1%; ++++ represents $P$ = less than 0·1%.

There were three pairs of ricks which were separated by only a few yards and which yielded sufficient mice to enable comparisons to be made (1, 2: 19, 20: 27, 28). Eight of the thirty-five characters showed significant levels of heterogeneity between these paired ricks, but in every case these were characters which showed a greater degree of heterogeneity over the farm as a whole. In ricks as close together as these 'paired' ones, it would be surprising if there was not some movement between ricks serving to keep populations mixed (even though Southern & Laurie (1946) state that 'once mice have entered a rick, they rarely move out until the rick is threshed') and hence the similarity between the pairs would be expected. However, the finding does establish the point that rick populations can be compared by the method used.

The differences between the incidences of characters over the whole area of Roke Farm are much greater. In the past (Weber, 1950; Deol, 1958; Grüneberg, 1961) comparison between populations has been based entirely on the number of significant differences between the incidences of the characters in the populations concerned. C. A. B. Smith's method (v.s.) has made the characterization of population distinctiveness much more precise. Table 5 shows the measures of distinctiveness between all combinations of two ricks (paired ricks treated as single populations). An examination of this table and of the incidences of individual characters (Table 1) and comparison with Fig. 1, does not show any recognizable pattern in the measures of separation between different ricks.

The last column in Table 5 is the mean distinctiveness, or uniqueness, of any rick from those with which it is compared. The two highest values are those for ricks 0 and 26. Rick 0 was a year older than all the other ricks. It was a barley rick built in the autumn of 1958, and thus stood for 80–90 weeks. It contained over 300 mice and 100 rats, and was not included by the Ministry scientists in their poisoning trials. Rick 26 was less obviously peculiar. It was situated on the edge of the farm in a group of four ricks which yielded only forty-one live mice, twenty-two of these coming from rick 26.

Three other extrinsic causes could conceivably affect the variation of mice in different ricks:

(*a*) Every rick (except 0) was given one of four different poisoning treatments (Table 1), varying in intensity but all involving the poison warfarin (Rowe, Taylor & Chudley, 1961). These treatments differed significantly in their effects and were certainly responsible for reducing the infestation. Since there is evidence of genetical resistance to warfarin poisoning, it was conceivable that some of the differences between ricks might have been due to the results of different intensities of selection for warfarin resistance. However the pairs of ricks 27 and 28, and 1 and 2 received different intensities of treatment and, as we have seen, there was little difference between members of either pair (the measures of divergence were 0·018 and −0·029 respectively). Although the effects of warfarin selection cannot be ruled out, they do not appear to have been great.

Table 5. *Measures of distinctiveness between Odiham rick populations.*
(*The italicized figures are estimates of the standard deviation*)

| Rick no. | 1/2 | 16 | 19/20 | 22 | 24 | 26 | 27/28 | 30 | 35 | 36 | 37 | Measure of uniqueness |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 0 | 0·072 | 0·073 | 0·066 | 0·073 | 0·084 | 0·165 | 0·039 | 0·137 | 0·079 | 0·069 | 0·078 | 0·935 |
| | *0·018* | *0·018* | *0·015* | *0·018* | *0·022* | *0·036* | *0·013* | *0·025* | *0·019* | *0·021* | *0·021* | |
| 1/2 | | 0·049 | 0·023 | 0·074 | 0·071 | 0·072 | 0·022 | 0·037 | 0·053 | 0·040 | 0·011 | 0·523 |
| | | *0·015* | *0·009* | *0·018* | *0·020* | *0·024* | *0·010* | *0·013* | *0·015* | *0·016* | *0·008* | |
| 16 | | | 0·024 | 0·060 | 0·050 | 0·084 | 0·039 | 0·050 | 0·046 | 0·038 | 0·035 | 0·547 |
| | | | *0·009* | *0·016* | *0·017* | *0·026* | *0·013* | *0·015* | *0·014* | *0·015* | *0·014* | |
| 19/20 | | | | 0·030 | 0·032 | 0·098 | 0·015 | 0·029 | 0·021 | 0·018 | 0·010 | 0·365 |
| | | | | *0·010* | *0·012* | *0·026* | *0·007* | *0·010* | *0·008* | *0·009* | *0·007* | |
| 22 | | | | | 0·033 | 0·173 | 0·044 | 0·070 | 0·042 | 0·026 | 0·044 | 0·669 |
| | | | | | *0·014* | *0·037* | *0·014* | *0·017* | *0·014* | *0·013* | *0·016* | |
| 24 | | | | | | 0·107 | 0·034 | 0·070 | 0·032 | 0·056 | 0·028 | 0·598 |
| | | | | | | *0·031* | *0·014* | *0·020* | *0·013* | *0·020* | *0·014* | |
| 26 | | | | | | | 0·097 | 0·099 | 0·104 | 0·124 | 0·090 | 1·214 |
| | | | | | | | *0·028* | *0·028* | *0·029* | *0·035* | *0·028* | |
| 27/28 | | | | | | | | 0·041 | 0·027 | 0·010 | 0·027 | 0·394 |
| | | | | | | | | *0·013* | *0·011* | *0·008* | *0·012* | |
| 30 | | | | | | | | | 0·058 | 0·079 | 0·034 | 0·704 |
| | | | | | | | | | *0·016* | *0·022* | *0·014* | |
| 35 | | | | | | | | | | 0·007 | 0·035 | 0·504 |
| | | | | | | | | | | *0·007* | *0·014* | |
| 36 | | | | | | | | | | | 0·024 | 0·491 |
| | | | | | | | | | | | *0·013* | |
| 37 | | | | | | | | | | | | 0·415 |

(*b*) Threshing was spread over a considerable period, such that the standing time of ricks varied from 18 to 43 weeks (80–90 weeks in the case of rick 0). Mice can, of course, enter or leave a rick at any time, but the evidence (Rowe, 1962) is that the main ingress of animals takes place with the onset of cooler weather in the autumn. Breeding takes place throughout the year in ricks, the 'annual productivity . . . approaching the theoretical maximum' (Laurie, 1946). Hence it can be assumed that the great majority of animals have either been born in the rick, or have lived there for at least 3 to 6 months. The spring rick populations are therefore isolates from an essentially continuous population that was fairly widespread in the fields (and farm buildings) in the late summer of the previous year, and in which isolates sampling changes have occurred during the winter. The absence of any pattern in the difference between rick populations suggests that the previous year's field population was reasonably uniform.

(*c*) Most of the ricks contained rats (*Rattus norvegicus*); some had one or two weasels (*Mustela nivalis*) or a few *Apodemus*, or a number of harvest mice (*Micromys minutus*). Ricks with many rats have proportionately fewer mice; weasels, of course, kill any rodents they encounter. It seems unlikely that such biological competition would have any effect on the variation.

### (ii) *Other British populations*

The populations from Northallerton, Langtoft and the Shetland Isles are so small that they can be neglected. They have been included to show that the pattern of variation is broadly similar to places from which a larger sample was obtained. The mean measures of divergence were calculated as for the populations from Odiham (Table 6). The most obvious feature is the striking divergence of the Skokholm mice from populations from any part of the British Isles. They are in fact very different from all other populations examined (except the small Northallerton sample). The reasons for this distinctiveness will be discussed elsewhere. Besides this island, there are three pairs of populations which differ markedly: Odiham from Shotley, from Flaxton and from Addlestone. Again omitting Skokholm, the general impression, however, is of the lack of distinctiveness or divergence of populations from different parts. This is borne out by the measures of uniqueness of the different populations (last column in Table 6).

### (iii) *World-wide populations*

The populations from parts of the world outside the British Isles were only classified for eighteen characters. Table 7 shows the measures of divergence between these populations. Three British populations: Odiham, Skokholm and Faversham are included for comparison. The divergences between these three based on eighteen characters do not differ greatly from the previous analysis based on thirty-five. Not very surprisingly there are many significant divergences between populations. The environments from which many of them come are so grossly different that it is quite impossible to draw any conclusions about the operation of selection

Table 6. *Measures of distinctiveness between British mouse populations*

| | Langtoft | Shetland | Skokholm | Tuttington | Shotley | Flaxton | Faversham | Clandon | Odiham | Addlestone | Measures of uniqueness |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Northallerton | 0·123 | 0·239 | 0·371 | −0·038 | 0·053 | −0·051 | −0·030 | −0·062 | −0·018 | −0·006 | 0·058 |
| Langtoft | | 0·260 | 0·430 | 0·108 | 0·215 | 0·081 | 0·100 | 0·061 | 0·122 | 0·115 | 0·161 |
| Shetland | | | 0·489 | 0·178 | 0·212 | 0·272 | 0·235 | 0·260 | 0·230 | 0·201 | 0·257 |
| Skokholm | | | | 0·339 | 0·312 | 0·277 | 0·285 | 0·319 | 0·341 | 0·361 | 0·352 |
| Tuttington | | | | | 0·093 | 0·030 | 0·049 | 0·061 | 0·098 | 0·023 | 0·094 |
| Shotley | | | | | | 0·104 | 0·066 | 0·072 | 0·107 | 0·088 | 0·132 |
| Flaxton | | | | | | | 0·045 | 0·052 | 0·085 | 0·029 | 0·092 |
| Faversham | | | | | | | | 0·038 | 0·057 | 0·062 | 0·091 |
| Clandon | | | | | | | | | 0·027 | 0·053 | 0·088 |
| Odiham | | | | | | | | | | 0·107 | 0·115 |
| Addlestone | | | | | | | | | | | 0·103 |

Table 7. *Measures of distinctiveness between populations from scattered places on the globe*

| | South America High Altitude | North America Great Gull Island | North America Norwich, Vt. | North America Storrs, Ct. | North America R-3 | United Kingdom Odiham | United Kingdom Skokholm | United Kingdom Faversham | India Delhi | Singapore | Israel | Measures of uniqueness |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| South America, Low Altitude | 0·079 | 0·393 | 0·109 | 0·061 | 0·156 | 0·295 | 0·721 | 0·306 | 0·065 | 0·210 | 0·615 | 0·273 |
| South America, High Altitude | | 0·385 | 0·181 | 0·144 | 0·311 | 0·480 | 0·899 | 0·500 | 0·062 | 0·279 | 0·887 | 0·382 |
| Great Gull Island | | | 0·195 | 0·299 | 0·406 | 0·503 | 0·755 | 0·387 | 0·363 | 0·618 | 1·302 | 0·510 |
| Norwich, Vt. | | | | 0·048 | 0·115 | 0·324 | 0·582 | 0·275 | 0·201 | 0·342 | 0·751 | 0·284 |
| Storrs, Ct. | | | | | 0·110 | 0·269 | 0·707 | 0·277 | 0·117 | 0·260 | 0·789 | 0·280 |
| R-3 | | | | | | 0·247 | 0·705 | 0·274 | 0·290 | 0·256 | 0·759 | 0·330 |
| Odiham | | | | | | | 0·313 | 0·034 | 0·275 | 0·103 | 0·419 | 0·297 |
| Skokholm | | | | | | | | 0·286 | 0·671 | 0·426 | 0·352 | 0·583 |
| Faversham | | | | | | | | | 0·303 | 0·190 | 0·349 | 0·289 |
| Delhi | | | | | | | | | | 0·122 | 0·637 | 0·282 |
| Singapore | | | | | | | | | | | 0·359 | 0·288 |
| Israel | | | | | | | | | | | | 0·656 |

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in producing the divergences. For example, it is interesting that the two populations from hot climates, Singapore and Delhi, differ but little, but the significance of this is removed by the fact that their measure of divergence is greater than that between Singapore and Odiham! One comment can be made about the North American populations: Great Gull Island differs from the others much more than they do from each other. This island is in Long Island Sound and has been unoccupied since World War II (Dunn, Beasley & Tinker, 1960). It may be that we have here a parallel situation to Skokholm.

## 4. DISCUSSION

Searle (1954*a*, *b*) and Deol & Truslove (1957) have analysed the causes of epigenetic polymorphism in inbred strains of mice. They found that the occurrence of any particular variant is determined by the attainment of a critical size at the relevant stage of development, the incidence of variants being altered by deficient diets fed to the mother. Grewal (1962*b*) further demonstrated that this critical size is a function of the genotype.

The conclusion from comparing ricks of different compositions was that there is little ground for asserting that the diet plays any part in determining the incidences of any of the variants studied in the wild. It may be that a large proportion of the mice caught were not born to parents which had lived for long in the rick. This would be unlikely in view of the normal rate of increase of populations in ricks (Southern & Laurie (1946) estimated that rick populations double themselves every two months). However, all but one of the Odiham ricks were poisoned and it might be that young mice were killed preferentially and the large proportion of older mice surviving would swamp any 'diet effect'. The corollary of this would be an undue proportion of older mice surviving in some populations. There was no heterogeneity between heavily and lightly poisoned populations in the frequency of sacral fusions which give some measure of the age structure (for unpaired ricks, v.s., $\chi^2_9 = 15{\cdot}9$, $p = 5$–$10\%$), nor between Odiham mice and those from non-poisoned ricks elsewhere.

The most likely explanation for the absence of a 'diet effect' is that there is elimination of the smaller members of litters. This would in fact be selection against any effect of diet. Indeed it could represent powerful selection against extreme forms of any sort. Deol & Truslove (1957) postulated just such a selective effect. They found that 'while the oats diet as such is least deleterious to the mice, its effect on the skeleton tends to be strongest; conversely, barley is by far the poorest diet, yet its effect on the skeleton seems to be comparatively slight . . . only the bigger and hence presumably more normal young tend to survive, while on the less deleterious oats and wheat diets smaller and hence presumably less normal (mice) also stand a chance'.

Several workers (e.g. Southwick, 1955; Christian, 1956) have found heavy mortality among the young of mice as the result of over-crowding in confined colonies. Brown (1953) discovered the 'most important factor directly related to

survival of the young was the condition of the nest at and shortly after parturition. . . . By the time the population is composed of several adults, the nest defence has been thoroughly broken down and nest disturbance is at a maximum.' Shared nests are a common finding in ricks. Southwick (1958) detected a progressive deficiency of male mice weighing over 12·5 g (i.e. weanling and adult) with increasing density in ricks which may reflect this infant mortality. However, some of this deficiency must be the result of the emigration of mature males (Rowe (1962) found that 80% of the animals leaving a rick are mature males). Southwick also found that the incidence of foetal resorption increased from 14% in females living in a rick under low density conditions, to 27% in those living at a high density (although Laurie (1946) reported only 3% of embryos underwent resorption in her rick study, and found no evidence of mortality among nestlings).

Further circumstantial evidence for the action of selection comes from the fact that there is so little divergence between British populations (Table 6), although considerably different patterns of variation can exist (Table 7). This is at least suggestive of the action of stabilizing selection.

There is also indication of genetical drift between the Odiham ricks. The populations of these ricks are different; I have suggested that the original population in the autumn of 1959 (the founder populations) was fairly uniform. If this is correct, the observed divergence must have taken place after the ricks were built. The poisoning operation makes impossible any quantitative estimation of the extent of the drift. All that can be said is that the effective population number must have been small (Rowe (1962) found that 100 mice entering an unpoisoned rick in the autumn multiplied to form a colony of 1500 the following spring). Grüneberg (1961) found heterogeneity in the pattern of skeletal variation in five populations of rats (*Rattus rattus*) caught in grain shops in Delhi. He mooted that the rat population of the city is split up into numerous small isolates which 'differ from each other genetically due to genetic drift rather than to the forces of natural selection'.

The study described here was intended to test the value of epigenetic polymorphism for population studies. Workers in the past have always emphasized the importance of the environment in the manifestation of epigenetic characters (e.g. Searle, 1960). The results obtained show clearly that, in conditions of nature, the epigenetic skeletal patterns of different populations can be used to characterize those populations genetically. This means that objective comparison can be carried out between two otherwise uniform populations, and hence the method should be a valuable complement to ecological methods. For example, it could be used to investigate the genetical factor suspected by Chitty (1957) in the fluctuations in number of populations of *Microtus agrestis*.

Biological systems are not 'perfect' in the physical sense, but show a degree of unpredictability sufficient to arouse the suspicions of 'exact' scientists. Epigenetic polymorphism is one aspect of this unpredictability; the aim is to turn something which normally has nothing but nuisance value into an asset. It has already proved its worth in 'pure' genetics (Deol, Grüneberg, Searle & Truslove, 1960); I suggest that this usefulness can be extended into the study of micro-evolution.

6. SUMMARY

It has been suggested (Berry & Searle, 1963) that the discontinuous ('quasi-continuous') variants studied by Grüneberg *et al.* in the skeleton of rodents can be regarded as constituting *epigenetic polymorphism* in different populations. Comparisons have been made between the incidences of skeletal variants in house mouse populations collected from: corn ricks on a single farm in Hampshire; eleven separated localities in different parts of the British Isles; and nine other places throughout the world. These showed that the method could profitably be used for genetically characterizing and hence comparing populations. There was evidence suggestive of genetical drift between local populations and stabilizing selection over a larger area.

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