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THE STUDY OF EPIDEMIC DISEASES AMONG WILD ANIMALS

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CONTENTS.

I. INTRODUCTION.

FLUCTUATIONS in numbers of wild animals are of importance both in pure and applied ecology. The present paper is a contribution towards co-ordination of the scattered work that has been published on one phase of the main problem. Although a considerable amount of work has been done on epidemic diseases among wild animals, it has mostly consisted of sporadic and independent pieces of research. One of the objects of this paper is to show the value and importance, and in fact, the necessity of widely co-ordinated studies, in obtaining further advances in the subject. I am indebted to the Empire Marketing Board and the Hudson's Bay Company for supporting my research, during the course of which the data presented here were compiled. An appendix to this paper includes several notes upon epidemics observed, which have been communicated to me by naturalists. It was thought best to keep these separate from the summary of published data.

II. THE OCCURRENCE OF DISEASE AMONG WILD ANIMALS.

Up to the present time it has been customary to believe that wild animals possess a high standard of health, which is rigidly maintained by the action of natural selection, and which serves as the general, though unattainable, ideal of bodily health for a highly diseased human civilisation. This belief is partly true and partly false. It is probably true that animals are normally much more healthy than human beings, and that the weaker individuals are weeded out by the enemies of the species and the occasional severity of outer conditions. At the same time, wild animals are not free from *epidemic* diseases, which are of very widespread occurrence among them. The systematic study of disease in wild animals forms one of the latest branches of animal ecology, and it is therefore impossible to make many generalisations which can be of

any permanent value; but it is safe to say that many species of wild animals suffer from outbreaks of disease no less severe than those which attack civilised man. In some cases (*e.g.* the Norwegian lemming) the decimating effects of these epidemics far exceed anything that is witnessed among human beings (Collett, 1911–12, p. 154). An important point to note is that, in a number of instances at any rate, it can be shown that the epidemics are not in any way attributable to human interference with the animals, or indirectly with their environment, nor contracted from domestic animals kept by men in artificial conditions. Disease is, in fact, a perfectly natural phenomenon, and it forms one of the commonest periodic checks upon the numbers of wild animals, especially in the case of mammals, being in this respect no less important than enemies, climate, food supply, and other generally recognised regulating factors. Such epidemic diseases are usually associated with overcrowding in the population, though they may not actually be caused by it directly. In consequence of this, there is usually a rather well-marked fluctuation in the numbers of the population, great density being followed by great scarcity, and this by a period of gradual increase up to another maximum, which is in turn followed by another epidemic. It appears that one of the chief reasons why so little attention has been paid in the past to the pathology of wild animals is the general and widespread belief of biologists that the numbers of wild animals remain constant, whereas in fact they fluctuate more or less violently from year to year. Fluctuations in numbers are also of course commonly caused by other ecological factors such as weather variations.

The importance from a medical point of view of studying epidemics in wild animals is fourfold. First, it is clear that our general attitude towards health in the human population must be affected, if it can be shown that epidemic disease is a normal and frequent phenomenon in nature. Secondly, the existing theories as to the origin of human disease in history may have to be reconsidered; for example, the idea that disease originated through the overcrowding caused by the town life of early civilisations. Thirdly, the widespread existence of a number of unstudied epidemic diseases in wild species with which man and his domestic animals are daily in contact directly or indirectly, offers a possibility of tracing some of the epidemic diseases of man and domestic animals to reservoirs in wild species. Finally, the pathologist is put in possession of a new means of studying epidemics in animals besides man, and thus is able to extend in a different direction the important work upon epidemics in cage-mice initiated in 1919 by Topley (Topley, 1919; Greenwood and Topley, 1925). Owing to the fortunate fact (which we shall discuss more fully in the next section) that these wild animal epidemics can frequently be forecast with some accuracy, it is possible to study the forerunners of an epidemic—a thing which it is very seldom possible to do properly in human outbreaks.

It will be seen, therefore, that there is at least the chance that some light might be thrown upon the mechanism of epidemics in general by a study of

the diseases of some wild animals. We must now consider briefly the distribution of disease among animals, as far as is known at present. Amongst invertebrates such as insects, climate and other factors than disease appear to play the chief part in producing sudden fluctuations in the population. But even here, periodic disease plays its part, as instanced by epidemics in the European crayfish, *Potamobius* (Brocchi, 1896), in the freshwater shrimp, *Gammarus pulex* (Goodrich, 1928), amongst locusts and grasshoppers (Uvarov, 1929) and of septicaemia among cutworms (*Euxoa*) (King and Atkinson,1928). It is when we come to the vertebrates, however, that disease begins to loom importantly in the lives of animals. Natural epidemics have frequently been noted amongst freshwater fish (Fleming, 1871, I, 150, 207, 228, 259; Williamson, 1929) and they occur also in various birds (especially game birds), *e.g.* diphtheria in wood pigeons, *Columba palumbus* (Scone, 1927); coccidiosis in willow grouse, *Lagopus lagopus*, in Norway (Brinkmann, 1926); and gapes (*Syngamus trachea*) in young rooks, *Corvus frugilegus* (Elton and Buckland, 1928); also epidemics among passenger pigeons (*Ectopistes migratorius*) in Ohio (Miner, 1929); house-sparrows and tree-sparrows (*Passer domesticus* and *P. montanus*) in the north of Scotland (Stenhouse, 1928; Inkster, 1929); and "plague" (a virus disease) of blackbirds in northern Italy (Todd, 1930).

Among wild mammals, epidemic disease is remarkably widespread in all parts of the world, occurring generally when the population has become unusually dense. The fluctuations in the population accompanying these epidemics are in some species so remarkable and have such far-reaching effects upon the other animals associated with them, and often also upon the economic, agricultural, and medical problems of man, that there is some very good evidence available on the subject. The lemmings (*Lemmus, Dicrostonyx, Myopus*) and mice (*Microtus, Pitymys, Apodemus*, etc.), in all parts of the world in which they have been studied, undergo violent fluctuations in numbers which are often associated with considerable epidemics. A typical example of a mouse plague followed by an epidemic on a large scale occurred in Nova Scotia (Canada) in 1815, when the early settlements were invaded from the surrounding forest by hordes of field-mice (Patterson, 1886). The increase occurred in a zone about 80 miles long and 50 miles broad (*i.e.* over an area of about 4000 square miles). By midsummer there were enormous swarms: cats, dogs, martens and foxes appeared also in numbers and gorged on the mice, and many cats became feral and multiplied excessively. Most of the hay and corn was destroyed. The mice disappeared rather quickly in the autumn and winter. They were seen crawling about slowly in a languid way, and then began to die in hundreds. Some of them attempted to migrate as well. Next season there was hardly a mouse in the whole district, except in one small locality where the abundance of mice continued for several years.

In Norway, the lemmings living up in the mountains and on the northern tundras increase every three or four years, and migration usually ensues on a large scale (Collett, 1911–12; Elton, 1924). The epidemic disease which kills

off the Norwegian lemming was stated by Horne to be due, in four outbreaks, to a specific bacillus (*B. pestis-lemmi*), said to be allied to the *Pasteurella* which causes avian cholera, and which invades the blood and organs in enormous numbers (Horne, 1912); while Collett states that a high proportion of the lemmings suffer from a skin disease which was found by Johan-Olsen to be associated with a species of bacterium which he named *Streptothrix lemani* (Collett, 1895). Doubtless other organisms also play a part in the tremendous decimation of the population which occurs after each epidemic. The lemming migrations in Norway occur every three or four years. Thus 1922 and 1926 were lemming years, and the next was expected and took place in 1930. Another animal which has been intensively studied of recent years is the South African gerbille (*Tatera lobengula*), a species of desert rodent, which appears to undergo fluctuations similar to those of the lemming, and with probably about the same period of three or four years (Pirie, 1927, p. 138). Its epidemics have attracted considerable attention from local authorities in the Union of South Africa, owing to the spread of the bacillus of bubonic plague (*B. pestis*) in that country during recent years. It seems probable that this rodent normally dies from diseases harmless to mankind (*e.g.* the Tiger River bacillus, *Listerella hepatolytica* (Pirie, 1927, p. 163)), but that bubonic plague, in its spread across the desert region, replaced these harmless organisms during the epidemics of 1923–4, with serious results to the human population, and to a lesser extent again in 1928, when epidemics due to a new species of *Pasteurella* were also recorded (Mitchell and others, 1930).

The Canadian snowshoe rabbit or varying hare (*Lepus americanus*), whose chief home is in the northern forest region of Canada, is subject to astonishing fluctuations in numbers, which are vividly reflected in the fur returns of the Hudson's Bay Company, owing to the fact that certain fur-bearing animals such as the lynx and red fox are mainly dependent upon the snowshoe rabbit for food. The cycle of the rabbit covers about ten years, and although the periodic abundance can be shown to depend partly upon climatic conditions, disease usually plays an important part when the animals become overcrowded (Elton, 1924). Describing the rabbit maximum of 1886 in Manitoba, Seton (1928) said: "The abundance that year was prodigious." During the following winter after the epidemic had broken out "in Manitoba, the country from Whitemouth to Whitesend, 250 miles long by 150 miles wide, was flecked with the bodies of white-furred Hares....The summer and fall of 1892, I spent in the same region, and did not see a single snowshoe." The epidemic just described appears to have been unusually great, but not without parallel, and local outbreaks occur in one part or another of Canada every ten years. It is of some interest to note that the snowshoe rabbit cycle has been going on without a break for at least 100 years, as shown by the fur returns and other records for the rabbits and the lynx.

In the western parts of the United States, similar epidemics occur amongst wild snowshoe rabbits and jack rabbits (Nelson, 1909; Palmer, 1896; Warren,

1910), and whereas we know nothing about the nature of the diseases which attack the Canadian snowshoe rabbit over an area of some millions of square miles, in the United States some light has been thrown on the matter by recent work. In certain cases rabbits have been found dying of tularaemia, a bacterial disease which is highly infectious to man (Green, 1928; Redington, 1928). Outbreaks of tularaemia in several parts of Russia have been recently traced to water-rats (*Arvicola*) (Roubakine, 1930), and the disease has also occurred in Norwegian hares, from which people have been infected (Thiôtta, 1930, 1931). In Eastern Siberia and Mongolia the steppe marmot or tarbagan (*Arctomys bobac*) is one of the most important carriers of plague, and the periodic epidemics to which it is subject have been proved by Wu Lien Teh (1924) to be one of the main foci of bubonic plague in those regions.

It would take too long to enumerate in detail all the cases of natural disease which are known now to occur among wild animals, and we therefore sum up the available information in the table given below. This table is not exhaustive, owing to the extremely scattered nature of the information, but it should be sufficient to indicate the great richness of the field which lies open for research upon the diseases of wild mammals.

Table I. *Fluctuations in numbers of wild animals, associated with outbreaks of disease.*

LEMMINGS (*Lemmus, Dicrostonyx*).

Fluctuations in numbers (four-year cycle, usually) in Norway (Elton, 1924, 1925). Horne (1912) studied the bacteriology of lemming epidemics in 1896, 1903, 1909 and 1910, and found *B. pestis-lemmi*, which was also fatal to guinea-pigs and other animals. Collett states that skin disease is frequently found in old individuals during lemming years. Specimens obtained by Collett in 1891 were examined bacteriologically by Johan-Olsen, who found a micro-organism ("*Streptothrix lemani*") commonly associated with the skin lesions (Collett, 1895). An apparently similar skin affection found by C. Elton in old individuals of migrating lemmings in Finmark, during the summer of 1930. Outbreaks of a kind of gastric fever (known as "lemming fever") in human beings, and apparently also in domestic animals, occur locally during and after lemming years in Norway (Collett, 1895). These outbreaks are attributed to infection of drinking water by dead or dying lemmings, or by their excreta. The subject has never been properly investigated, but the circumstantial evidence is convincing.

Similar fluctuations occur in the populations of lemmings in Sweden, Finland, Russia, Novaya Zemlya, Siberia, Alaska, Arctic Canada, Greenland, Baffin Island, and Labrador (general summary by Elton, 1924, 1925; Novaya Zemlya, Pearson, 1898; Western Siberia, Sedelnikov, 1907; Baffin Island, Kumlien, 1879).

Although epidemics probably play a part in these fluctuations, they have never been investigated.

VOLES OR FIELD-MICE (*Microtus*).

Violent fluctuations in numbers, often associated with epidemics, have been recorded from Great Britain, Norway, Germany, Austria, Czechoslovakia, Russia, Thessaly, Hungary, France, Italy, the Aegaean Islands, Crete and Cyprus, Asia Minor, Palestine, North-eastern Siberia, Kamchatka, Alaska, the whole of Canada, Labrador, and many parts of the U.S.A. The references to the evidence on this subject are so numerous and scattered that they cannot be given here, and will be dealt with in a separate publication. It must be sufficient to refer to the text (pp. 443 *et seq.*) for the mouse cycles in Britain, Norway, and Bavaria. The evidence about epidemics in voles is discussed on p. 453.

FIELD-MICE (*Pitymys*).

Enormous mouse plagues in Italy, notably in 1916, when the mice died from epidemics of bacterial origin (Martelli, 1919).

RED-BACKED VOLES (*Evotomys*)

These mice, which are the typical forest-mice of the more northerly forests of both the Old and the New Worlds, have been shown to undergo important fluctuations in numbers

Table I (*continued*)

in several places, *e.g.* Ontario, Canada (Dymond, Snyder, and Logier, 1928, p. 243); Alaska (Sheldon, 1930, p. 121); and England (Pitt, 1918). It is not known if these fluctuations are accompanied by epidemics. A definite epidemic has been observed in Norway by Dr H. M. Blair (see Appendix, p. 453).

DEER-MICE (*Peromyscus*).

One of the commonest field- and forest-mice of the less northerly parts of North America, and occupying more or less the niche filled by *Apodemus* in the Old World. Fluctuations in numbers are frequent, and there are many records of sudden scarcity following great abundance. Dymond, Snyder, and Logier (1928, p. 244) say: "May it not be that the white-footed mouse (*Peromyscus*) plays a similar rôle in the northern coniferous forest of Canada to that played in the more northerly regions of Europe and America by lemmings and voles?" No direct evidence about epidemics has been recorded.

WOOD-MICE OR LONG-TAILED FIELD-MICE (*Apodemus*).

Took part in a great vole plague in the forest of Dean, England, in 1813 and 1814 (Douglas, 1825); mortality in the New Forest, England, in 1923, associated with similar deaths in voles (Longstaff, cited in Elton, 1924, p. 142). Spontaneous epidemic among voles and *Apodemus* at Charny, France, in February, 1893, from which Danysz obtained cultures for mouse-control work (Danysz, 1893); the numbers fluctuate greatly in Norway (Collett, 1912, p. 172). Frequently forms "plagues" of mice in French agricultural areas (Segnier, 1924) and took part in the vast mouse year that troubled Germany in 1918 (Schwarz, 1918), and Italy in 1916 (Martelli, 1919). On the Volga steppes *Apodemus*, among several other species, has been convicted as a permanent reservoir of bubonic plague, which bursts out in periodic epidemics (Dr I. Ioff, *in litt.*).

WILD MICE (*Mus*).

Australia. Periodic plagues in the wheat belt (both in the fields and in the bush), *e.g.* in 1916–17, when many mice died from a skin disease "somewhat resembling ulcerative syphilis" (Hinton, 1918), while the men who were handling the stacks contracted a skin disease on their arms, necks, and shoulders; the mouse plague was followed by a plague of fleas, which attacked human beings (information through Dr J. R. Baker).

Bubonic plague carriers on Volga steppes (Dr I. Ioff, *in litt.*).

GERBILLES (*Tatera, Desmodillus*) and other rodents.

Great fluctuations in numbers on the steppes and deserts of South Africa, associated with several different epidemic organisms (see text, p. 438).

GERBILLES (*Rhombomys*).

Epidemic of unknown nature in Kara Kum Desert, Turkestan, in 1927 (Kashkarov and Kurbatov, 1930).

HAMSTERS (*Cricetulus*).

Associated with outbreaks of bubonic plague in Mongolia (summary in Elton, 1925); took part in the great mouse year in Germany in 1918 (Schwarz, 1918).

COMMON RATS (*Mus*).

Associated with outbreaks of bubonic plague almost all over the world. In many areas plague periodicity is directly due to periodic epidemics among the rats themselves. A natural epidemic cycle of this sort appears to have existed in English rats in East Anglia between the years 1906 and 1918, and *Bacillus pestis* was proved to be causing deaths both in rats and human beings (literature briefly summarised in Elton, 1925).

WATER-VOLES (*Arvicola*).

Fluctuate in numbers, coincidentally with the lemmings and voles in Norway (Collett, 1912, p. 106; Wolley, 1856).

Proved to be the reservoir of outbreaks of tularaemia in Russia (Roubakine, 1930).

MUSKRATS (*Fiber*).

Marked cycle in numbers in various parts of Canada, partly associated with epidemics, *e.g.* of liver disease (MacFarlane, 1905, p. 737).

BEAVER (*Castor*).

Local epidemics in beaver colonies in the MacKenzie River region of Canada (MacFarlane, 1905, p. 743).

SQUIRRELS (*Sciurus*).

Marked fluctuations in numbers in Great Britain and Ireland (Middleton, 1930 *b*); in Norway (Collett, 1912, p. 223, Wheelwright, 1871, p. 230); in Denmark (Hansen and Schiödte, 1892); in Siberia (Elton, 1925); and in Canada (Seton, 1920).

In the case of British squirrels Middleton (1930 *b*) has obtained convincing evidence that the periods of decline in numbers are very often associated with disease, the nature of which is still uncertain.

Table I (*continued*)

MARMOTS (*Arctomys*).
Proved to be one of the most important reservoirs of bubonic plague in Mongolia (Wu Lien-Teh, 1924). There is a good deal of evidence that the epidemics in marmots are associated with periods of abundance (Elton, 1925).

GROUND SQUIRRELS (*Citellus*).
Convicted as reservoir of bubonic plague and also of tularaemia in California (McCoy, 1910; Francis, 1925).

SUSLIKS (*Citellus*).
Periodic outbreaks of bubonic plague in north Caucasus steppes (Sveridenko, 1929).

VARYING HARES OR SNOWSHOE RABBITS (*Lepus*).
Very marked fluctuations in numbers over nearly the whole of their range, in Canada, Alaska, and also in Montana (Seton, 1920, p. 96; Seton, 1928; Elton, 1924; Sheldon, 1930, p. 329; Howell, 1923). Epidemics of unknown nature occur over many thousands of square miles, but have never been adequately investigated. Recently tularaemia has been found in Canada (Hudson, 1930), but the circumstantial evidence does not favour the idea of the widespread occurrence of this particular disease in Canada, although it is said to have been found several times among varying hares in the U.S.A. (Green, 1928).

JACK RABBITS (*Lepus*).
Subject to occasional epidemics on a large scale in many parts of their range; Western U.S.A. in general (Nelson, 1909), Colorado (Warren, 1910), California, Nevada, Idaho, Utah, and Washington (Palmer, 1896). Proved to be dying of tularaemia in Utah in 1919 (Redington, 1928).

ALPINE HARES (*Lepus*).
Periodic epidemics reported in England and Scotland, mostly of unknown cause, but in one case at least proved to be coccidiosis (Ritchie, 1926). Recently suspected of being reservoir of tularaemia in Norway (Thiötta, 1930). Collett (1912) states that epidemics attack hares in Norway in certain years, causing disease of the lung, and associated with strongylid worms (see also Appendix to this paper, p. 454).

COTTON-TAIL RABBITS (*Sylvilagus*).
Said to take part in epidemics with snowshoe or jack rabbits in U.S.A. (Nelson, 1909).

COMMON RABBITS (*Oryctolagus*).
Frequent epidemics in rabbit populations in the British Isles, little studied. References too numerous to summarise. Apparent causes in some cases are coccidiosis, liver fluke, and rabbit syphilis.

WEASELS (*Mustela*).
Probably subject to outbreaks of disease of unknown cause in Manitoba, Canada (Criddle, 1925).

BADGERS (*Meles*).
Subject to outbreaks of disease of unknown cause in Manitoba, Canada (Criddle, 1925).

RED FOXES (*Vulpes*).
In Britain they appear to suffer from periodic outbreaks of disease called "yellows" and believed to be spirochaetal jaundice (Dunkin, 1926). Certainly subject to outbreaks of nervous disease in various parts of Canada (Elton, through the Hudson's Bay Company, unpublished) and also in Kamchatka (Bergman, 1927).

ARCTIC FOXES (*Vulpes*).
Suffer from periodic outbreaks of a nervous disease resembling encephalitis, which is communicable and fatal to sledge-dogs, in the arctic parts of Canada. These outbreaks probably occur just after the periodic decline in numbers of lemmings (Elton, through the Hudson's Bay Company, unpublished).

MOLES (*Talpa*).
Occasional mortality in England, on a more or less large scale, as in 1926. The causes are unknown (see Appendix, p. 454).

SHREWS (*Sorex*).
Periodic fluctuations in Norway, often at times of lemming abundance (Collett, 1912, p. 9). In Britain they are subject to epidemics which kill them off in large numbers (*e.g.* in Denbighshire in 1925; Moffat, 1926).

DEER (*Cervus*).
Periodic epidemics in early years in Scotland (Fleming, 1871, p. 243).

DEER (*Odocoileus*).
Periodic cycle in numbers on Vancouver Island and in Western British Columbia not investigated for disease (Brooks, 1926).

Table I (*continued*)

ELK OR MOOSE (*Alces*).
Epidemics due to unknown causes in Lithuania (Barnard, 1864) and Canada (Dymond, Snyder, and Logier, 1928, p. 248; Bradshaw, 1916).

CHAMOIS (*Capreolus*).
Epidemic of infectious disease causing blindness in Switzerland (Anon., 1927).

WILD SHEEP (*Ovis*).
Said to suffer from periodic epidemics every twenty years or so in the Pamirs (Cumberland, 1895).

AFRICAN ZEBRAS.
Periodic outbreaks of lungworm in East Africa (Percival, 1924).

HIPPOPOTAMUS.
Epidemics of unknown cause in the Congo (Hilton-Simpson, 1911; see Appendix to this paper, p. 453).

Only a few references have been given to plague (*Bacillus pestis*) in animals, since so many of the records are only isolated cases of infection, and not associated with epidemics on a large scale. A list of animals which have been found dying of bubonic plague is given by Wu Lien-Teh (1925), and criticised and amended by Pirie (1927, p. 122). A brief summary of some of the evidence associating plague epidemics with important fluctuations in the populations of wild animals is given by Elton (1925) and the recent work on this question in South Africa is summed up by Pirie (1927, p. 138), and Pirie and Murray (1927).

It will be observed from Table I that two of the diseases (bubonic plague and tularaemia) which produce epidemics among wild animals also produce epidemics in man. Our biological knowledge of both these diseases is very recent, since it was not until about the beginning of the twentieth century that plague was conclusively proved to arise from rat epidemics, and not until 1923 that it was finally proved to originate in marmots (although the connection was suggested by Clemow in 1900, on a basis of strong circumstantial evidence). Tularaemia is a still more recent discovery, and dates from 1911 and 1912.

It is obvious that the pathology of wild animals is a subject that has hardly passed beyond the observational stage as yet, and that the unknown elements must be incomparably more numerous than the known. The comparative success of medical work in regard to man, both on the bacteriological and pathological sides, has made it possible to forget that our knowledge of disease in wild animals is very meagre. It would hardly be possible, for instance, to sum up our present knowledge of human epidemics in a short table such as is given above! In view of the increasing speed and perfection of modern transport, there are growing up new opportunities for the spread of diseases capable of finding fresh reservoirs in wild animal populations.

III. FORECASTING EPIDEMICS AMONG WILD RODENTS.

The table given in the previous section (p. 439) shows that epidemic diseases are not uncommon amongst wild animals, and that very little is known about their nature or their causes. One reason for this state of affairs has been the difficulty of knowing when to expect the outbreaks. For instance, in the early days of plague research in China, it was for some years almost impossible to prove conclusively that the Mongolian marmot (*Arctomys bobac*) was the actual reservoir of plague for Chinese outbreaks, because scientific expeditions to the districts where the marmots were dying usually arrived after the epidemic had run its course. As a result of this it remained for some years doubtful whether the marmot outbreaks arose from the human plague epidemics or *vice versa* (Wu Lien-Teh, 1922). Finally the establishment of a permanent station to watch for the occurrence of disease, made it possible to demonstrate conclusively the connection between marmot epidemics and human outbreaks of plague. But it proved necessary to watch for about ten years before success was achieved (Wu Lien-Teh, 1924).

The same difficulty is encountered in the study of any other animal epidemics, and is, of course, a prime cause of our ignorance of the mechanism of epidemic diseases in human beings. It is obvious that a reasonably accurate forecasting system for epidemics would enable research to be focussed more efficiently on the problem and would make it possible to go to the right place at the critical moment, and thus avoid the uncertainty, and the waste of time and money which otherwise occur.

Now, the remarkable fluctuations in the population which usually accompany epidemics in certain wild animals, make the building up of such a forecasting system quite feasible, provided enough information can be obtained about the changes in numbers from year to year, and about the course which the cycles have run in the past.

The numbers of mice and lemmings are known to fluctuate in all parts of the world where the subject has been investigated; in Labrador, in California, in the wheat-belt of Australia, in the deserts and steppes of South Africa, on the mountains and also on the lowlands of Scandinavia, on the Volga steppes, over most of central Europe, and in Great Britain. The earlier records that we possess refer simply to "mice," without giving details about the species concerned, and they are mainly descriptions of very large "plagues" of mice, during which crops were eaten up and destroyed. Several such "plagues" are mentioned in the Bible, and they are noticed fairly often in the records of Greek and Roman history. Such large plagues occurred in Alsace in the years 1271, 1278, 1366, 1378, 1451, 1468, 1479, 1501, 1538, 1593, 1617, 1652, 1685, 1719, 1794, 1801–2, 1818, 1822, 1856, 1861 (Gerard, 1871). This list gives an idea of the sort of periodicity shown by really great "mouse plagues," and that there is no especial regularity in their occurrence. (In this case there are 21 in 600 years.) If we examine the records for some country over the

last hundred years we find that there are, in between the great mouse plagues, a good many smaller periods of unusual increase which would not, however, have been sufficiently important to become preserved in the earlier historical records. Thus, it is recorded that the department of the Aisne in France suffered from unusual increase of field-mice in the years 1865, 1870 and 1880 (Chavée-Leroy, 1882), 1909, 1912 and 1919 (Segnier, 1912; Marchal, 1919). It will be seen that the "mouse years" in these two series were three to ten years apart.

This fact leads us to enquire whether there may not be still smaller "mouse years" in which the population reaches a relatively large peak in numbers, which is not absolutely large enough to attract attention through the economic damage which it would cause if the mice were more numerous. In order to discover whether this is so, that is to say whether mouse plagues, in the economic sense, are only unusually big periodic maxima in a natural cycle of numbers, it is necessary to have special means of investigating the periodicity.

It is not easy to carry out direct censuses of wild mice, and such attempts have only been made within the last few years. For obtaining data over a long period of years we have therefore to rely upon indirect means of detecting variations in the numbers. So far, these methods have been applied with success four times; in the study of lemmings and mice in Norway, lemmings in Canada, mice in Great Britain, and mice in Bavaria.

1. Lemmings and mice in Norway. Lemmings (*Lemmus, Dicrostonyx*) are northern representatives of the vole family, whose members are the ecological equivalents of the short-tailed field-mice (*Microtus*, etc.) of the temperate regions. As far as the present problem is concerned, mice and lemmings may be treated as one unit. Owing to the fact that there is a backbone of mountain running from north to south down the centre of Norway, the arctic-alpine zone of vegetation extends far south of the arctic circle, and covers large areas of the Dovre, Jotunheim, and other mountain plateaux of southern Norway, and is inhabited by the Norwegian lemming (*Lemmus lemmus*), which also occurs on the lowland tundras much farther north. The lemming population is subject to the same fluctuations which occur among field-mice, and owing to the peculiar position of the lemmings in southern Norway, and the great amplitude of their fluctuations, they frequently overflow their natural habitat and visit the lowlands. These migrations have long been a subject of wonder and comment, and present many interesting features of their own; but the point with which we are concerned here is that they give an index of the periods of over-population in the lemming habitats. The dates of these migrations have been recorded by Collett (1912) and others, and the periodicity has been studied by Elton (1924). It is found that migrations occur in some part of central or southern Norway at very regular short intervals of three or four years; but they do not necessarily occur over the whole of this region, as we should expect if migration is only an index of unusually great over-population. In the period between 1860 and 1930 this periodicity in migrations (and there-

fore in numbers) has been maintained, and in the course of these seventy years there have been only two occasions (1897 and 1914) when the records do not show a migration at the time expected. These two exceptions were most certainly cases when over-population was not sufficiently great to cause migration, since all degrees of migration have been noted, from enormous waves of lemmings marching in thousands and reaching the sea-coast (*e.g.* 1909 and 1926), to small local migrations such as those recorded for 1880 and 1918.

We conclude from this evidence that there is a regular cycle in numbers of lemmings on the mountains of central and southern Norway, with a periodicity of three or four years. As mentioned on p. 439, epidemics play an important part in the phase of decrease in numbers. A number of parallel records, of a less complete nature, prove that the numbers of wild mice on the mountain slopes and foothills and lowlands usually reach a peak in the same years as the lemming. Furthermore statistics of the government bounties paid for arctic fox and red fox and birds of prey, show peak years at regular intervals, and these correspond with the lemming cycle, and show maxima after 1897 and 1914 when there were no actual migrations of lemmings recorded (Johnsen, 1929). On the basis of this periodicity I forecasted that 1930 would be a lemming year in Norway and ventured a visit on this prediction. It was in fact an important lemming year over the whole of Norway.

2. LEMMINGS IN CANADA. The lemmings are one of the chief foods of the arctic fox in the northern regions of Canada, and it can be shown that the numbers of the latter are probably an index of the numbers of the former. The arctic fox fluctuates in numbers very violently, and these fluctuations are shown in the fur returns of the Hudson's Bay Company (see Elton, 1924, 1925). This curve, which represents mainly the skin collection from Ungava Bay on the northern coast of Labrador, has a periodicity of three or four years. We conclude therefore that the Canadian lemmings have a cycle in numbers similar to that of the Norwegian lemmings—a conclusion which is supported by data about the lemmings themselves. A further striking fact is that the peak years for lemmings in Canada show some correlation with those in Norway, indicating that there may be a common climatic factor influencing lemmings in both countries.

3. FIELD-MICE IN GREAT BRITAIN. Most of the observations which have been made upon fluctuations in numbers of British mice refer to the short-tailed field-mouse (*Microtus*), which occurs all over England, Wales and Scotland, but is absent from Ireland. Long-tailed field-mice (*Apodemus*) may also take part in these fluctuations. An extensive investigation has been carried out by A. D. Middleton under the auspices of the Empire Marketing Board. This work has been published elsewhere (Middleton, 1930 and 1931), and will only be briefly summarised here.

By means of questionnaires and other sources of information from a number of people, it has been ascertained that local mouse plagues or "mouse

years" in Great Britain occur periodically, and that the periodicity is three or four years. Thus 1898, 1902, 1906, 1909–10, 1913–14, 1918, 1922, 1926 and 1929–30 were local mouse years. There is a very strong tendency for these mouse peak years to occur simultaneously in different districts. Thus there were mouse years in 1926 in Somerset, Westmorland, Cumberland, Roxburgh and Argyll. The simultaneous occurrence of these outbursts of numbers, and the comparative scarcity of mice over wide areas in the intervals between them, points to the existence of some controlling factor in climate. This idea is strongly supported by the remarkable correspondence of mouse years in Great Britain with mouse and lemming years in Norway. The tendency for synchronous and parallel fluctuation to appear in the numbers of small rodents on both sides of the North Sea, and the tendency for correlation between lemming years in Canada and Norway, may be taken to indicate some general climatic pulsation acting over the whole of that part of the North Atlantic.

4. Field-mice in Bavaria. A great deal of work has been done upon the incidence and nature of "mouse plagues" in France and Germany, and numerous attempts have been made to control these outbreaks by means of various destructive measures. Much work has been done especially upon the introduction of various "viruses" (mostly cultures of bacteria belonging to the *Salmonella* group; see White, 1929), in order to initiate epidemics among the mice. This work has had a certain local success, but the effects of it have been usually masked and falsified by the fact that the mice have a natural cycle of their own which causes them to disappear after they become abundant. The existence of a regular and natural periodicity in numbers of field-mice was first pointed out by Hiltner (1916), and for a number of years he collected statistics which show conclusively that in Bavaria there is a very definite cycle in numbers, which varies somewhat from one region to another.

The forecasting of the mouse numbers in Great Britain is based therefore at present upon the following knowledge:

(*a*) Knowledge of the widespread and well-authenticated existence of cycles in numbers of small rodents, in many different countries.

(*b*) Knowledge that there is a fairly regular fluctuation in numbers of mice, probably controlled in some way by climate, which occurs more or less simultaneously in England and Norway and even in Canada.

(*c*) Past records showing that the interval between maxima has been usually four years, sometimes three, and rarely more or less.

(*d*) Information as to the date of the last maximum. Thus in 1925 we were aware that 1922 had been a mouse year in Hampshire (New Forest) and Somerset, also that 1922 was the year of a great lemming migration in Norway. The next maximum year would therefore be expected to occur in 1926 or possibly in 1925. An intensive piece of ecological work was started on this prediction, the results of which are being published elsewhere (Elton, Ford, Baker and Gardner, 1931).

(*e*) Information as to the actual state of mouse numbers at the time.

Thus, the comparative scarcity of mice when operations were started in the autumn of 1925, made it seem probable that the peak would not be reached until 1926. Negative information like this, however, is not really trustworthy, since in some peak years the mice may reach only relatively very small density of numbers. For instance it appears probable that the exceptionally severe frosts of the winter of 1928–9 greatly reduced the numbers of wild mice in some parts of England, so that the usual peak is beginning to show itself mainly in areas which had been protected by a snow covering, *e.g.* mountains of Wales and parts of Scotland. It is hoped, as a result of work which is going on at Oxford upon mice, to discover the exact manner in which mouse increase is affected by climate and thus make forecasting more accurate.

We may now turn to the important question of the relation of disease to cycles in numbers of mice, and the evidence that epidemics occur among wild mice at all. The periodic epidemics proved to occur among Norwegian lemmings and South African gerbilles have already been commented upon, while references are given in Table I, pp. 439, 440, to epidemics in many other small rodents such as hamsters, rats, etc. There is an enormous literature devoted to the study of vole plagues in Europe, dealing more especially with the means of controlling outbreaks by using poisons, bacterial diseases, and other measures. The more purely biological aspects of the problem that vole plagues present have not, however, been adequately studied, with the result that much of the work done by bacteriologists upon the control of mouse numbers by disease has been uncritical, based upon erroneous preconceptions, and, above all, lacking in adequate controls in the field experiments. The commonest type of experiment has been to introduce into the mouse population (then at or near its maximum abundance) cultures of *Salmonella enteriditis* or other strains of "mouse typhoid," and watch the effect upon the numbers of the mice. Usually successful results have been claimed. But it has often been noted by independent observers that the voles were disappearing in other areas where no control measures had been applied. In other words the experimenters have seldom reckoned with the fact that the mouse populations into which the epidemic cultures were introduced were probably nearly always at the point of generating some form of epidemic themselves. The recognition of the existence of natural epidemics among wild mice makes it extremely difficult to prove the efficacy of an artificially introduced disease at all, since the time of greatest abundance is the very time when both artificial and natural epidemics might be expected to begin successfully.

The general truth of this situation has been admitted by more than one worker. Thus Rabaté (1914) said, "En effet, les grandes invasions disparaissent subitement, pour des causes encore mal connues, comme en novembre 1905.... La disparition générale semble due à une affection microbienne, épidémique, à propagation rapide, et intéressante à suivre de près, mais pour les campagnols rien encore sur ce point n'a été précisé." His remarks were made in

1914 at the time of a severe vole plague in France, which had been continuing for over a year unabated, in spite of all repressive measures.

A curious point about the work that has been done upon mouse-plague control by means of artificially introduced diseases, is the almost complete absence of published evidence that wild mice and voles ever die of mouse-typhoid epidemics, except when the bacteria have been deliberately introduced. With the exception of the original epidemic in February, 1893, from which Danysz (1893) first obtained cultures of "mouse typhoid," and several epidemics among cage-mice such as that recorded by Loeffler (1892), records on the subject seem to be very rare. At the same time it appears to be widely believed that mouse typhoid is a common source of epidemics among wild mice, and this belief has deflected investigation from the natural epidemics themselves. It would appear, on the contrary, that little is known about the actual organisms responsible for killing off *Microtus* at times of abundance, and that the belief in the widespread nature of mouse-typhoid epidemics has arisen mainly from the propaganda of vested interests concerned in the manufacture of control bacterial cultures on a large scale. It is significant that there is at the present time a general reaction against the use of such cultures, because the epidemics initiated by them are insignificant in extent and duration. The evidence on this point has been conveniently and impartially summed up by White (1929), while it may be noted that the official recommendations of the American Bureau of Biological Survey for mouse destruction do not mention bacterial cultures at all, but confine themselves to poisons and trapping (Silver, 1924). It seems fairly certain that this generally adverse opinion is not due merely to successful propaganda on the part of firms selling poisons for the same purpose! The importance of this point, which has been discussed at some length, is that if the disappearance of field-mice after "mouse plagues" is not caused by mouse typhoid, it must be caused by something else. Moreover, the cause of the mortality must be of a rather obscure nature, to have escaped the attention of the many bacteriologists engaged on a study of the problem. The facts at present known would be explained if we assumed that the voles die off periodically from some virus disease which leaves little trace on the tissues (as might be the case with a rapidly acting disease); and if at the same time we suppose that the epidemic is preceded by a period of lowered resistance to invasion (due for instance to food shortage or to the scarcity of some factor in the food). At such times we might expect the mice to be susceptible to some extent to other organisms, thus accounting for the definite though limited effects of the introduction of mouse typhoid. It should be remembered also that adequate technique for studying filterable viruses is only of recent origin.

Most of the evidence for epidemics in *Microtus* is circumstantial and consists of records of enormous multiplication in numbers, followed by very sudden disappearance, coupled with evidence that the voles have not simply migrated, and often with the discovery of numerous dead bodies which have

no signs of external injury. Innumerable examples of sudden and unaccountable disappearance could be quoted in connection with the European mouse plagues. A good example from Canada has already been described (p. 437). Preble (1908, pp. 186–8) described a mouse plague which overran central Saskatchewan and Alberta (the grain provinces of Canada) in 1900, and he said "immense numbers, many of which were floating down the rivers, were found dead." During the months of April in the following year (1901) he found fairly recent traces almost everywhere he went, between Edmonton and Fort Chipewyan, but practically no signs of any living mice. In 1907 there was a great mouse plague in Nevada, Utah, and north-western California. By November, 1907, the fields were honeycombed with the burrows of the mice, and much damage was done by them. By next August the voles had almost completely disappeared. Piper (1908, p. 302) said: "while the Nevada plague is the most serious recorded in the United States, frequent milder outbreaks in many parts of the country indicate that practically all our species of short-tailed mice periodically tend towards enormous multiplication." At intervals between January and March, 1908, dead and dying mice were noticed (the deaths being found not to be due to poison), but attempts to prove the mortality due to some specific bacterial disease were unsuccessful. Another great outbreak of mice took place in Kern County, California, during 1926–7; this time the main agent in the multiplication was the house-mouse (*Mus musculus*), which overran a newly planted area of land, and subsequently migrated for some distance into surrounding country. This was not, therefore, strictly comparable with the normal type of fluctuation in numbers of field-mice, but it is mentioned here both because *Microtus* also took part in the increase (although in smaller numbers) and also because both kinds of mice were later found to be dying from an epidemic of a disease which was studied by Wayson (1927) and found to be caused by *B. murisepticus*, an organism almost if not quite indistinguishable from that responsible for erysipelas in swine and human beings.

In England, through lack of attention, and also through the lesser economic importance of field-mouse plagues, there is not a very large amount of evidence about epidemics in voles. An epidemic among short-tailed and long-tailed field-mice was noted by Longstaff in the New Forest in 1923 (cited by Elton, 1924), and another by A. Moffat during the great vole plague in Scotland in 1892: "the voles seemed to get into a dormant state as if they were stricken by some disease...they disappeared underground, to be seen no more, where only a few days previously they had been running in thousands" (Moffat, cited by Middleton, 1930, p. 161). Several definite epidemics have been recorded from Scandinavia. Thus Wolley (1856) noted that three species of voles (*Microtus amphibius, agrestis* and *ratticeps*) increased at the same time as the lemmings in 1853 in northern Lapland (Muonuoniska). During the spring of 1854 they nearly all disappeared, and dead and dying bodies were found, with no external injuries, very abundantly in hayricks, etc. An epidemic among

Evotomys in Norway, observed by Dr Blair, is described in the Appendix, p. 453.

It will be noted that most of these descriptions agree in one respect: the absence of any obvious symptoms of the type one has been led to expect in bacterial diseases. Plague and erysipelas are exceptions, but it does not appear that *Microtus* is a common carrier of these diseases; on the other hand the records of epidemics in other kinds of voles and mice are more specific. These facts tend to confirm the hypothesis that *Microtus* usually dies from some disease or diseases that will require a special technique for their examination.

The account which has just been given of mouse cycles and the research which is being developed in order to forecast their periods of abundance and scarcity, and their epidemics, illustrates a technique which can be applied to a number of other species about which at present we have much less knowledge. It can undoubtedly be applied to the ten-year cycle in Canadian wild animals such as the varying hare (snowshoe rabbit) and the ruffed grouse, and probably the muskrat. A full account of investigations which I have been carrying out in co-operation with the Hudson's Bay Company upon cycles in Canadian wild life, will be published in the early future. It is clear that ecology, and particularly that branch of it which deals with animal numbers, has a definite contribution to make towards the epidemiology of mammals, and also towards the solution of certain important problems in human disease.

Summary.

1. Outbreaks of epidemic disease are common in populations of wild animals, including species little influenced by contact with the diseases of human beings or domestic animals.

2. Such epidemics form one of the commonest factors responsible for fluctuations in numbers of wild mammals.

3. An attempt is made to summarise the available published records of such epidemics (Section II), while certain unpublished records (communicated to me by naturalists) are contained in an Appendix.

4. Little is known of the causes of these epidemics except in the cases of plague and tularaemia.

5. The fluctuations in numbers of some wild mammal populations are sufficiently regular to make the forecasting of epidemics possible. This method is already applicable to wild mice.

6. Mouse periodicities are discussed in detail, with special reference to epidemics and their causes, which are mainly obscure (Section III).

7. Development of the forecasting methods described will make possible the prediction of many other wild mammal epidemics, and render intensive pathological and epidemiological studies more practicable than they have hitherto been.

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APPENDIX.

SOME UNPUBLISHED DATA ON ANIMAL EPIDEMICS.

The following data have been communicated to me by naturalists during the course of my enquiries. I wish to thank them for permission to publish their first-hand observations here.

1. EPIDEMIC AMONG HIPPOPOTAMI IN THE CONGO. In 1911 Captain F. W. Hilton-Simpson published a note on this subject in his book *Land and Peoples of the Kasai* (p. 29), in which he stated: "During our journey up the Kasai, the captain of the *Velde* told us that about the year 1904 or 1905 a very deadly epidemic had broken out among the hippopotami of that river and the Sankuru. So great had been the mortality among the animals (which even now exist in the middle Kasai in almost as great numbers as in Wissman Pool) that the factories on the bank had been obliged to employ men with canoes to push out into the current the carcases which had lodged on the shore close at hand, the stench from which, as they began to decay, had been appalling. I could gather no information as to the nature of this disease."

Captain Hilton-Simpson wrote to me as follows: "It is, however, interesting to note that whereas hippos were very common in the Kasai and Sankuru Rivers in 1907–9, my friend, Captain Douglas Fraser, who was out there last year (1927), could scarcely believe me when I told him that they were no extreme rarities. Neither he nor I believe that any human agency has exterminated them, and, on my mentioning the epidemic I refer to, he agreed with me that there have probably been one or two recurrences of it."

2. EPIDEMIC AMONG RED-BACKED VOLES (*EVOTOMYS RUFOCANUS*) IN NORWAY. During the years 1924–8 Dr H. M. Blair collected a great deal of valuable information about the fluctuations in numbers of wild mice in the north of Norway (Varanger Fjord). He has kindly allowed me to record here an epidemic observed among red-backed voles, *Evotomys rufocanus*, during the early summer of 1927. Voles had increased progressively during the years 1924–6 and "1926 was noteworthy as a mouse year; both species (*Microtus ratticeps* and *E. rufocanus*) literally swarmed around Svanvik in the Pasvik Valley, and in Nord-Varanger they were very abundant. The winter of 1926–7 was long and severe, the spring of the latter year very cold and backward, yet voles were as common as in 1926....In the early part of June 1926 many dead voles (*E. rufocanus*) were lying amongst the short grass of a little meadow surrounding my lodgings at Svanvik on the Pasvik River. In many cases no obvious cause of death could be discovered, but quite a number of the bodies showed a rather remarkable condition. In these large abscesses had formed either in the abdominal wall, or in connection with the external genitals. These collections of pus did not appear to be due to an actual injury with septic infection, but rather to a pyaemia. In the surrounding marsh no dead voles were found, but they would be easily concealed from sight by the thick growth of dwarf birch, Ledum, etc."

This record is important in three ways particularly. It was very fortunate that the epidemic should have been observed by a trained medical man; it is the only record of a definite epidemic in this genus *Evotomys* that I have heard of; and it can have had no connection with lemming epidemics since no lemmings visited Varanger Fjord in these years. Dr Blair adds: "In 1928, Dr Stewart of Elleray Bank tells me he did not see any signs of mice around Svanvik."

3. Epidemics among hares (*Lepus timidus*) in the north of Norway. It has long been known that hares in Norway are subject to outbreaks of disease (Collett, 1911–12, p. 64). In 1930 Thiötta drew attention to the existence of tularaemia in human beings in Norway derived from hares, and this has focussed interest on the natural epidemics occurring among these animals, which have not been properly investigated. While working on rodents, during the Oxford University Lapland Expedition in 1930, I obtained three pieces of information about hare epidemics which seem worth putting on record.

(*a*) Mr Tharald Frette, Norwegian forestry officer at Karasjok (a Lapp settlement in the middle of Finmark, north Norway), informed me of the great fluctuations in numbers of hares noticed by him during the period he had been in the district. This information was obtained through the help of Mr Einar Mathisen, who was the Norwegian member of the expedition. Between 1920 and 1926 there were numerous hares every year. In 1926 there were still a great many, but fewer than in 1925. In 1927 the numbers began to drop heavily, and there were outbreaks of epidemic among all the older hares. Mr Frette believed it to be the lung disease described by Collett. In 1928 hares were scarce. By 1929 they had begun to increase again, and in 1930 they were numerous once more.

(*b*) Mr Claus Anderson of Tromso informed me that outbreaks of epidemic disease take place among the hares on some of the islands off the coast, near Tromso. These outbreaks apparently occur when the hares become too abundant.

(*c*) Mr Mathisen informed me that on an island in Porsanger Fjord (north Finmark), people were killing a great many hares every day in the autumn of 1930.

4. Epidemics among moles (*Talpa europaea*) in the south of England. During the early summer of 1927 evidence was obtained of widespread mortality among moles in the country for about five miles round Oxford. A striking epidemic was observed by Mr F. J. Prewett on his farm at Tubney, Berks., during June. Moles had been abundant here for some time, and they suddenly began to come to the surface and die, with the result that after about a week very few were left on the farm. I found a recently dead mole at Cothill, near the last area mentioned, at the same time. Three specimens were examined bacteriologically by Dr A. D. Gardner, who has kindly communicated the following notes to me:

1. Mole found in dying condition by Mr Prewett on his farm, 24. vi. 1927.

Examined shortly after death. The lungs showed white-grey spots visible to the naked eye. A smear of the lungs showed no bacteria, cultures from heart blood, lungs and spleen were negative, nor was any parasite seen in blood smears. No special symptoms were noted.

2. Found dead by Mr Prewett at the Tubney farm, 24. vi. 1927. The lungs were in the same condition as in No. 1. All cultures were negative, as also were blood smears.

3. A lung from a specimen found dead at Cothill was sectioned and submitted to Dr Gardner.

In the lungs of all three specimens Dr Gardner observed small darkly staining bodies about the size of red-blood corpuscles. The nature of these was not determined.

A number of other dead moles were reported independently by various other observers in districts round Oxford. A summary of these records is given below: in no case were signs of external injury seen, as might have been expected had the moles been killed by human beings or animals or birds of prey.

Shotover, 15. vi. 1927. Dead mole on Johnston's Piece, Shotover Hill (Mr E. M. Nicholson).

Baldon, end of May, 1927. Moles were extremely abundant at the end of May, and then all disappeared within about a week, all being gone before the middle of June. In October, 1927, they had only just begun to return again, and one dead one was found lying on a mole hill at the beginning of this month (Mr D. Skilbeck).

Boar's Hill, beginning of June, 1927. Dr Gardner found a dead mole.

Eynsham, 30. vi. 1927. Three dead moles seen between Oxford and Eynsham, in the fields (Mr G. Tickner).

Bagley Wood, May and June, 1927. Dead moles were seen by me in various parts of the wood, in early May, June 7th, between June 20th and July 11th, and on July 11th (six altogether). In September, 1927, I noted that moles were scarce in the wood, where they had been abundant in the previous spring, and on October 6th I found another dead one. These observations are supported by those of other people. Prof. J. W. Munro saw three dead moles in the wood in mid-June, while Mr Cross, the gamekeeper, reported several in another part of the wood during the end of June and beginning of July.

Farther afield, there was also some slight evidence of reduction in numbers and deaths. Thus, I found a dead mole, apparently uninjured, near Kirtlington on 4. vi. 1927. Mr O. W. Richards found two dead moles near Cholsey (near Wallingford) on 25. v. 1927. In the New Forest, Dr T. G. Longstaff informed me that moles were very numerous in his garden at Picket Hill, Ringwood, in the early spring of 1927. When he returned in July, after an absence of three months, there were practically none left. Finally, a remarkable mortality was witnessed a year later (July, 1928) by Mr T. Bird, on a farm near Colchester, when moles were observed to come out in numbers and die on the surface. We are justified, then, in concluding that in May and June,

1927, there was considerable mortality among moles round Oxford, and apparently also in places farther away. There was also mortality a year later at Colchester. The examinations made by Dr Gardner point to some epidemic disease being partly responsible. It should be noted, however, that the summer of 1927 was very dry, and that the unusual conditions may possibly have had something to do with the mortality, if only by predisposing the moles to attack by disease. I am informed by Mr Gee, who has a farm near Oxford, that during the drought of 1921 moles came out and died and that when he gave water to one of them it lapped eagerly. It is of course well known that moles make runs to water. He believed that the dry conditions of 1929 were in some areas having a similar effect as he had seen two dead ones (2. vii. 1929). There is no evidence that drought in 1927 was anything like as severe as 1921, and I suggest provisionally that lack of water (which indirectly causes lack of food, through the ground being dry) may be a determining factor in starting epidemics among moles.

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