periodicals published anywhere in the world. Volume I (1936) of the proceedings has just appeared. Any communications and copies of proceedings for exchange should be sent to Dr. J. H. Kusner, secretary, Florida Academy of Sciences, University of Florida, Gainesville, Florida.

DISCUSSION

SHOCK DISEASE AND THE SNOWSHOE HARE CYCLE

THE periodic decimation of the snowshoe hare in North America, which approximately every ten years reduces the numbers of these animals to scarcity, seems again to have run its course in Minnesota. For several years past hares have decreased in numbers throughout the state, and in most areas appear to be near or at the extreme low point. On the Lake Alexander Area in Morrison County, where extensive field and laboratory studies have been carried on since 1930, a peak population of hares was reached in 1933, when the trapping census showed 478 hares per square mile. The decline has been continuous since that time, but the greatest drop occurred during the fall and winter of 1935, so that by the spring of 1936 the population was reduced to 164 hares per square mile. Now in the fall of 1937 the numbers are so low that any enumeration will be difficult.

It has been the finding of the Minnesota Wildlife Disease Investigation,[1] from intensive studies on the Lake Alexander hare population and from supporting investigations on hare samplings throughout the state of Minnesota, that the die-off of snowshoe hares is due to a new disease entity in that species which we have called "shock disease."[2]

Hares suffering from shock disease appear perfectly normal until they are suddenly stricken with convulsions and die in the seizures or abruptly sink into a fatal coma. We first used the term "shock disease" when we recognized that some hares trapped and held in capitivity would die from the shock of change in environment. Later we learned that the convulsive seizures in these animals were hypoglycemic in character and that death was usually due to an abnormally low blood sugar.

So far as we have been able to determine, the basis of the disease is a degeneration of the liver. Usually in advanced cases the liver is a dark mahogany in color and is atrophic and definitely smaller than normal, so that the capsule is separated from the parenchyma and lies as a wrinkled membrane over the surface of the organ. Microscopically, the liver cords are highly atrophic. Thus far we have recognized consistent changes only in the liver; pathological findings in other organs are variable.

The liver degeneration is associated with a failure to store glycogen. When the carbohydrate reserve in the liver reaches a low value, the hare leads a precarious existence. As excitement or exertion depletes the small glycogen reserve, the blood sugar drops below the normal range, unconsciousness supervenes, and the hare dies. Routine technical procedures have shown that normal hares have an average liver glycogen value of 5.5 per cent., while hares in shock have values from 0.02 per cent. to 0.18 per cent.

Our first investigations of this disease in live hares were confined to animals found sick in box traps and dying, as a rule, while being taken to the laboratory. When it was recognized that the condition was so widespread that large numbers of hares died when subjected to the mild but continuous strain of captivity, intensive investigations were carried out on captive groups. During March, 1936, just at the end of the period of sharpest decline, we held 204 hares under observation. The animals survived an average of only 4.2 days, and not a single hare survived the 20th day. The conditions of captivity were favorable for extended survival as the food and surroundings were identical with those provided for groups of hares held under observation indefinitely, previous to the widespread occurrence of shock disease in the hare population. The hares appeared at ease, hopped around interestedly, and ate a variety of foods. However, a hare appearing normal would suddenly spring into the air in convulsions or sink to the floor in coma. In either case, death usually followed from a few minutes to an hour after the onset of symptoms.

This type of sudden death of hares was observed in the woods under entirely natural conditions by our staff at the same time, and similar occurrences were reported to us by others. During the summer of 1936, with the assistance of Deane Mather, of the U. S. Forest Service, shock disease was demonstrated to occur in hares 6 weeks old while living in a wild state in fenced natural ranges in which they were born. It has become evident that the death of hares which we have observed in captivity is a process which has been accelerated, but which is otherwise identical with that involving the animals undisturbed in the wild.

The large groups of hares which we have held in captivity while observing deaths from shock disease represented samples from central Minnesota, northeastern Minnesota and the northwest area lying south

[1] Executive Reports of the "Minnesota Wildlife Disease Investigation," Vol. 1 (July, 1933, to December, 1934), and Vol. 2 (January, 1935, to June, 1936). Mimeographed by U. S. Department of Agriculture.

[2] R. G. Green and C. L. Larson, *Am. Jour. Physiol.*, 119: 319–320. 1937.

of Lake of the Woods. The hare populations in these regions were afflicted with shock disease to varying degrees. Since animals from these districts rapidly succumbed to shock disease in captivity, and hares were dying from the same disease in the wild, there would seem to be no doubt that the hare population throughout Minnesota was in a process of destruction from shock disease during the winter and spring of 1936. The evidence that shock disease is almost wholly responsible for the decimation of hares is greatly augmented by our extensive attempts to demonstrate a significant mortality from other diseases which might be a factor in the area of the die-off. Such infectious diseases as tularemia we have found to play but a minor role in the mortality of hares. Moreover, the general picture of the decline, continuing until populations are extremely low, is not one typical of the course of an epizootic disease. In the sphere of our investigations both positive and negative findings point to shock disease as the primary cause of the periodic decimation of the snowshoe hare.

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PRODUCTION OF WHEALS IN THE HUMAN SKIN[1]

UNPUBLISHED data of Abramson, Sookne and Moyer have indicated that there is in ragweed extract an active amphoteric constituent of high molecular weight which is negatively charged at pH 7.4. We have therefore attempted to elicit allergic wheals by the electrophoresis of ragweed extracts from absorbent cotton into the skin of individuals hypersensitive to aqueous extracts of ragweed pollen. It was discovered that wheals were obtained after five to ten minutes with current densities of 0.3 to 0.5 milliampere not only with the negative pole but also with the positive pole used in the same fashion. Large wheals with pseudopods were obtained following electrophoresis of dialyzed extracts. The same technique employed on subjects not hypersensitive to ragweed did not result in whealing. To account for the fact that the positive pole gave as large if not larger wheals than the negative pole, we may hypothesize either the presence of positively charged active constituents, or invoke the notion that the positively charged water molecules which set up an electro-osmotic stream through the pores of the skin may be responsible. Similar results have been obtained using extracts from the pollen of timothy grass. In unpublished experiments with Lubkin, it has been shown that positively charged histamine ions can be electrically transported from the skin from wheals produced by histamine by application of the negative electrode. Prior to the formation of an allergic wheal by means of the electrophoresis of pollen extract using the negative pole, the formation of histamine is the generally accepted theory. It should then be possible to remove the histamine from an allergic wheal by applying the negative pole. A recovery of histamine in this fashion has not as yet been achieved.

Similar experiments on the electrophoresis of insulin through the skin are in progress.

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SAUROPOD DINOSAUR REMAINS IN THE UPPER CRETACEOUS

AN expedition from the Smithsonian Institution working in the Wasatch Plateau region of central Utah during the summer of 1937 made a discovery of more than ordinary interest. In the type section of the North Horn formation, in Emery County, a considerable portion of a very large sauropod dinosaur was found in beds carrying horned (Ceratopsian) and duck-billed (Hadrosaurian) dinosaur remains. These animals fully indicate the Upper Cretaceous age of the lower eight hundred feet of the North Horn formation.

The Sauropod specimen is of special interest not only because of its unexpected appearance here, but also because it pertains to the family Titanosauridae, the first recognized occurrence of this family in North America. At this time only a scapula and a section from the mid-caudal region has been prepared, but the latter are sufficient to show the caudal vertebrae to be procoelus with small neural arches confined to the anterior half of the centra, both typical features of the family as known at the present time.

The genus *Titanosaurus* was established by Lydekker on caudal vertebrae from the Upper Cretaceous (Lamenta Beds) of India, and the same authority was the first to report the genus from the Upper Cretaceous of Patagonia.

The specimen under discussion has not been sufficiently prepared as yet for proper comparison with the type of *Alamosaurus sanjuanensis,* from the Upper Cretaceous (Ojo Alamo) of New Mexico, but such comparisons as have been possible appear to show its affinities to be near, if not belonging to that genus.

It should be recalled that *Alamosaurus* was the first sauropod dinosaur to be recognized from the Upper Cretaceous of North America, and the skepticism with which that announcement was received may now be dissipated by this second discovery under circumstances that are even more convincing than the first, if that is necessary.