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Factors in the Mass Mortality of a Herd of Sika Deer, *Cervus nippon*[1, 2]

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ABSTRACT

A six year population study on sika deer, *Cervus nippon*, introduced in 1916 on James Island in Chesapeake Bay, Maryland, provided unique results because of the unusual completeness of the data due to an islandic situation. A density of one deer per acre was reached in 1955. In 1958, 60 percent of the population, mainly young and females, died during January and February. Gross and microscopic studies were made on 18 deer, shot and autopsied in 1955, 1957–60, plus one recently dead at the time of the die-off.

Adrenal weight increased, especially in the young, from 1955 to 1958 and then dropped 50 percent following the die-off. Inhibition of growth observed before and during the die-off vanished afterwards. Changes in the adrenal *zona glomerulosa* and medulla suggested overstimulation and a severe imbalance of fluid-electrolyte metabolism as the cause of the die-off. These changes may have been secondary to prolonged hyper-stimulation of the cortex as a result of excessive population density and its resultant social pressures. An inclusion hepatitis and glomerulonephritis are described which involved all deer, especially after 1958, but not in 1955. These diseases were ruled out as causal factors in the die-off, as were malnutrition and poisoning. The deer were apparently in good nutritive status throughout.

It was concluded that physiological derangements resulting from high population density produced the observed effects.

Introduction

For many years the physical deterioration and increased mortality of deer herds which often occur in winter have been attributed, with few exceptions, to malnutrition or starvation (Latham, 1950; Leopold *et al.*, 1951; Longhurst *et al.*, 1952; Dasmann, 1956; Swank, 1956). However, competent observers occasionally have suggested that malnutrition may not be the common critical factor (Cheatum, 1952) but that other and more subtle agents may be more important than food. In fact, malnutrition often is an *ex post facto* diagnosis, with critical studies lacking (Longhurst *et al.*, 1952). The deer are dead and appear to be in poor condition—therefore they must have starved. Actually, the diagnosis of malnutrition usually is based on macroscopic changes in the carcasses, changes that are known to be non-specific and that can be induced by a variety of factors which impose unusual demands on physiological adaptive processes. Evidence will be presented here to show that at least in sika deer, *Cervus nippon*, population density, not food, can be the major factor that limits both physical condition and numbers.

Material and Methods

This study concerns the herd of sika deer on James Island, Maryland, a tract of 280 acres located off the Eastern Shore of Chesa-

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Fig. 1.—Part of a herd of sika deer, *Cervus nippon*, on James Island, Maryland, in May, 1958, to illustrate the well nourished and healthy appearance that characterized the animals throughout the period of study. Photograph by Ted Kell, courtesy of the *New York Herald Tribune*.

peake Bay. Fig. 1 shows the species under typical conditions on James Island. The history of this herd has been reviewed recently (Flyger and Warren, 1958; Flyger, 1960). The present phase of observations on the herd began in 1955 when a census indicated a population of 280–300 deer, a density of one per acre, at which time a die-off seemed inevitable.

Accordingly, deer were shot for study in April 1955, April 1957, March 1958, February 1959, and March 1960. Autopsies usually were begun within a half-hour after death, and representative blocks of tissue were fixed in 10 percent neutral formalin. All carcasses were weighed before and after dressing during 1955, thereafter weights were taken only before dressing. Also in 1955, adrenals, thyroids, thymuses, spleens, and ovaries of all animals were weighed. Thereafter only adrenal weights were recorded. Age was determined by the method of Severinghaus (1949) assuming that the wear and replacement of sika teeth were comparable to those in white-tailed deer. The teeth of the two species appear to be similar enough to warrant this treatment.

Histological sections of 5 micra or less in thickness were cut from the adrenals, kidneys, liver, thyroids, thymus, spleen, gonads and adnexa, heart, aorta, lungs, and other tissues that appeared to be of importance to the study. Sections were stained by Lillie's allochrome as well as haematoxylin and eosin (Lillie, 1954). In addition, frozen sections of the livers from the 1960 sample were stained with Sudan IV for fat. Sections supplied by Dr. Frank Hayes, from a deer which had succumbed during the die-off in 1958, also were examined.

Additional gross weights were supplied by the Maryland Department of Research and Education.

The data in this study were not evaluated statistically, as the differences in the critical data were so great that such treatment was not warranted.

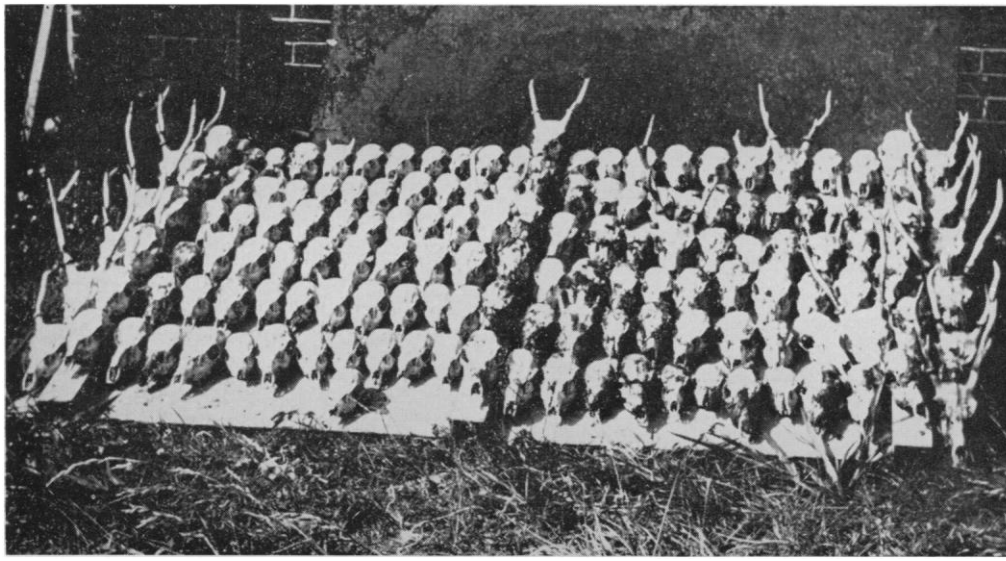

Fig. 2.—Skulls of the 147 sika deer, *Cervus nippon*, recovered on James Island, Maryland during the die-off in 1958. Reproduced from a kodachrome taken by V. F. Flyger.

Results

History and Habits of the Herd

This herd grew from an initial introduction of four or five deer in 1916 (Flyger and Warren, 1958; Flyger, 1960). The recent numerical history of the herd, in brief, is as follows: In April, 1955 the population was estimated to number between 280 and 300 deer. No change was noted in 1956 or 1957 but, from late January 1958 through March 1958, many deer died and 161 carcasses were recovered (probably none missed). Fig. 2 shows a collection of sika deer skulls taken during that period. A count of 109 surviving deer was obtained under unique circumstances following a deer drive in April 1958 (Flyger and Warren, 1958), hence the number of deer prior to the die-off was at least 270. Therefore the population had remained stationary at one deer per acre from April 1955 (or earlier) through December 1957. Counts indicated that the herd had declined further the following year to approximately 80 in 1959. Females and young were the predominant victims during the die-off (Table 1). The sex and age composition of the survivors is not known.

The diet of sika deer is more cosmopolitan than that of white-tailed deer. On the island sikas eat wax myrtle, grasses, red maple, red gum, loblolly pine twigs, needles, bark, and tree roots. Many plants have been grazed or browsed so heavily that they no longer exist or are rare on the island, and for this reason statements concerning food preferences cannot be made. Japanese honeysuckle and greenbriar, which are common on the adjacent mainland, are not seen on the island. Poison ivy leaves are found only on trees above the reach of the deer. Many of the loblolly pines on the island bear extensive scars where deer have stripped the bark from the trees. No change in the ap-

Table 1.—Sex and age composition of sika deer, *Cervus nippon*, carcasses found on James Island, Maryland from February through April, 1958.

Sex	Age Group					
	Calf (Less than 12 mos.)	1–2 years	2–3 years	3 years and over	Undetermined	Total
Male	—	6	8	6	—	20
Female	—	10	22	28	—	60
Unknown	50	1	8	8	14	81
Total	50	17	38	42	14	161

TABLE 2.—Temperature, precipitation and snowfall recorded at the Blackwater Wildlife Refuge, Dorchester County, Maryland, approximately 15 miles east by southeast of James Island, during the winters of 1956 through 1960.

	Mean Monthly Temperature				Total Precipitation				Total Snowfall			
	Jan.	Feb.	Mar.	Dec.	Jan.	Feb.	Mar.	Dec.	Jan.	Feb.	Mar.	Dec.
1956	33.2	39.3	42.9	46.4	2.2	3.7	4.1	2.4	3.0	0	0.5	0
1957	34.8	41.7	44.6	41.2	3.5	4.1	3.8	5.6	12.0	4.0	0	3.0
1958	33.6	31.2	40.7	32.6	3.6	4.5	6.0	2.1	4.0	7.0	7.0	10.0
1959	35.3	37.8	45.5	43.5	3.2	2.2	3.0	3.7	2.0	0	T	0
1960	41.0	39.6	34.9	—	2.7	3.6	1.7	—	T	5.0	15.0	—

pearance of the vegetation due to browsing could be detected from 1955 through 1960. In particular, there was no change in the degree of browsing on pine bark. Bark peeling has been a habit of the deer, at least as long as the authors have visited the island (1955). Consumption of tree bark apparently is a characteristic of the species. For example, in Denmark sika deer are considered a serious nuisance because of their habit of bark-peeling and browsing young trees (Jensen, 1959; and Flyger, 1959).

According to local residents and observations by the authors the deer are most active at night. Large herds of over 40 animals frequently swim to the mainland at night (a distance of about a mile) or wade across the water between the north and south island. Since the 1958 die-off most of the deer (probably 90 percent) spend the daylight hours on the south island. Sika deer probably are established on the mainland but some people believe that they reside only on the island and make excursions to the mainland. Because of their secretive nature and nocturnal habits observations on sika deer are difficult to make.

People on the mainland adjacent to the island (plus a lone resident on James Island) are interested in these unique deer and provide what little is known about these animals. On two occasions (January-February 1958 and March 1960) the deer did not commute when ice formed around the island or against the mainland. The die-off occurred at the time of, and following the ice conditions in 1958 (Table 2). However, following 10 days of severe cold and ice in March, 1960, two trips were made to the island for the purpose of examining the deer. On one occasion about 40 deer were seen at close range and all appeared to be in good condition. On the second trip five men walking about on the island found no dead deer. Again all deer seen appeared to be in good condition. Two subsequent trips to the island also failed to discover any dead or sick deer. Therefore we do not believe, as do Hayes and Shotts (1959), that ice conditions contributed directly to the mass mortality. Furthermore, there had been fairly frequent freezes prior to 1955 without any report of excessive mortality coming to our attention.

PHYSICAL CONDITION

The deer collected from 1955 through 1960 were, with one exception (※1961), in excellent condition and appeared well nourished. All animals had moderate amounts of fat in the mesenteric and subcutaneous depots. Their musculature was, without exception, well developed with no evidence of emaciation. Their pelage was shiny and dense, not patchy, loose, or ruffled. Further, numbers 1959, 1960, 1963, 2451, 2452, and 2454 were as large as any males seen at the time of their collection. Indeed, in ※2451 both hind legs had been shattered at the ankle joint, apparently by a shot well before collection. The animal was running on the exposed tibiae and yet appeared to be in excellent condition, wholly unimpaired by this degree of injury, as its body weight indicates (Table 3). Collections were made in the daytime when stomachs were full. The contents consisted largely of grasses, wax myrtle, and loblolly pine. We have noted one possible exception, ※1961, to the otherwise apparently good condition of these deer collected at the time of the die-off. An effort was made to collect sick deer but this individual was the only one which was in obvious distress. It staggered, appeared weak, and its pelage was somewhat ruffled. Nevertheless, this animal was not emaciated nor in poor condition at autopsy.

Significant changes in the size of these deer were found from year to year in spite

of the good general appearance and a moderate amount of fat. A general decline in body size occurred between 1954 and 1958, followed by a sharp increase in 1959 and 1960 (Table 3). Since the condition and relative amount of fat remained constant, and since all deer had full rumens at autopsy, it is reasonable to assume that weight is an indicator of growth. The live weight of the males was 34 percent greater in 1960 than in 1958, including ✻2453 which weighed only 24.1 k. This deer was born in 1955 and therefore experienced the conditions of the die-off during its period of most active growth. The growth of a male born in 1954 (✻2452) does not appear to have been inhibited. No does were collected in 1958, but those collected in 1960 were 28 percent heavier than the 1955 and 1957 females (Table 3). Body weights are obviously different, clearly indicating that growth of these deer was seriously impaired through 1958, but subsequently recovered.

The reproductive ability of the deer seemed unimpaired. All mature females collected (1955 and 1960) either were pregnant or lactating. The five embryos collected (three in 1955 and two in 1960) were viable and well developed. All mature males showed evidence of having undergone active spermatogenesis during the preceding rut. Sperm were found either in the testes and epididymes or in the epididymes only, depending on the date of collection. Therefore reproductive function apparently was normal in all samples.

No gross abnormalities of the abdominal or thoracic organs, brain, or pituitary were noted until 1960, when a mature male (✻2452) had a grossly scarred liver and yellow body depot fat. The external surface of the liver was coarsely granular, the organ was resistant to cutting, and its normal color was mottled with yellow. The fat was less deeply yellow in the second male (✻2454) and its liver was grossly normal. However, marked changes in the weights of the adrenals and the microscopic appearance of the adrenals, liver, and kidneys were found and will be discussed below. The sections of all other organs were essentially negative.

TABLE 3.—Weight of sika deer, *Cervus nippon*, and of their adrenals.

Date	Sex	Age	Weight	Status	Adrenal	Deer Number
			(*K*)		*mg/kg*	
December, 1952–54[1]	M	—	31.3	antlered	—	—
	M	—	29.5	"	—	—
	M	—	33.6	"	—	—
	M	—	28.6	"	—	—
	M	—	33.6	"	—	—
April, 1955	F	6+	23.6	1 embryo	10.5	1634
	F	2	19.1	1 embryo	14.3	1635
	F	2	19.5	1 embryo	9.3	1636
	F	4–5	23.2	lactating	8.7	1638
	F	1	11.4	immature	9.4	1637
					$\bar{x}$ = 10.7	
April, 1957	F	2	20.0	lactating	—	—
March 6, 1958	F	—	12.5	found dead	—	—
March 8, 1958	M	3	23.6	antlered	13.1	1959
	M	3	22.7	antlered	9.2	1960
					$\bar{x}$ = 11.2	
	M	1	8.2	immature	20.7	1961
	F	1	10.5	immature	17.4	1962
					$\bar{x}$ = 19.1	
February 13, 1959	M	3	34.5	antlered	8.8	1963
March 25, 1960	M	3–4	33.2	antlered	5.2	2451
	M	5–6	33.6	shed	7.4	2452
	M	3–4	32.7	antlered	5.1	2454
	M	4–5	24.1	antlered	6.4	2453
	M	1	15.0	immature	7.6	2455
					$\bar{x}$ = 6.0	
	F	2–3	27.3	1 embryo	5.8	2450
	F	2–3	27.3	1 embryo	6.5	2456
					$\bar{x}$ = 6.2	

[1] These data were supplied by the Maryland Game and Inland Fish Commission.

Detailed parasitological examinations were made in 1955 and 1958. Only gross examinations for parasites were made subsequently. The examinations were negative except for one female in 1955 which had nematodes in its lower colon.

HISTOLOGICAL DESCRIPTION

Adrenal glands: Changes in weight of the adrenal glands were associated with changes in population (Table 3). An increase occurred from 1955 to 1958, followed by a decline in 1960. Since there

is no difference in weight of adrenals from males and females the adrenal weights are combined for comparative purposes. Relative adrenal weights (mg/kg) remained relatively constant in mature deer from 1955 through 1958, at about 11 mg/kg, and dropped to about 6 mg/kg in 1960 (Table 3) a decrease of 46 percent from 1958 to 1960, associated with a decline in the population of approximately 60 percent. Adrenal weight in immature deer increased 78 percent from 1955 to 1958, and then declined by 1960 to 40 percent of the 1958 and 81 percent of the 1955 levels. Sample sizes were small, but the differences were marked and support one another. More specimens of immature animals from 1960 would have been desirable. It is notable that the deer with the heaviest adrenals in 1960 (♂1952) also had cirrhosis of the liver, a condition commonly associated with adrenal hypertrophy.

These changes in adrenal weight with changes in population size are appreciably greater than has been observed in any other species. It is evident that changes in the population were associated with greater changes in the adrenal weights of young than of mature deer.

Differences in adrenal weight with respect to age, population, and hepatic disease were found, microscopically, to be due to differences in the amount of cortical tissue, particularly of the fasciculata-reticularis, although no consistent significant differences were observed from sample to sample in the appearance of the cells of these zones (Figs. 3A, 3B, and 3D). However, the appearance of the cortical *zona glomerulosa* changed significantly during the study (Figs. 3A–3D). The glomerulosa of all deer in 1955 was relatively free of cells exhibiting pycnosis (Fig. 3A). However, by 1958 and persisting through 1959 and 1960, there was a marked number of cells showing pycnosis (Figs. 3B and 3D) which probably was most striking in ♂1963 of 1959 (Fig. 3B). Such pycnotic nuclei suggest over-stimulation accompanied by cellular degeneration. The condition of the *zona glomerulosa* of the deer which had died in 1958 was identical to those from deer which were shot (Fig. 3B).

In 1958 a young animal (♂1961), noted to be in a weak condition, showed marked degenerative changes in the glomerulosa characterized by necrosis of the cells with hemorrhage into cords, so that individual cords approached the appearance of blood sinusoids (Fig. 3C).

The adrenal medullas appeared normal in 1955 (Fig. 3E) but in 1958 were seriously altered by a marked irregularity in cell size and a striking number of pycnotic nuclei in the degenerating cells in the deer which were shot as well as in the one that had died, suggesting, as for the glomerulosa, over-stimulation with degeneration and repair. This appearance persisted but with some evidence of resolution in the medullae in 1959 (Fig. 3G), but a return toward normal was seen in all deer in 1960 (Fig. 3H).

Kidneys: Except for possibly a slight increase

Fig. 3A, B, C, and D of the outer *zona fasciculata, zona glomerulosa*, and capsule of the adrenals of sika deer, *Cervus nippon*, over a 6 year period, showing changes occurring before, during, and after the die-off. All × 212, H and E stain.

3A. ♂1635, typical of the appearance in 1955 and considered normal for the species. Note the general absence of pycnotic nuclei and cytoplasmic degeneration in the cells of the *zona glomerulosa*.

3B. ♂1963, illustrating typical appearance at the time of the die-off (1958) and the year after, including the deer found dead at the time of the die-off. Note the ragged appearance, often shrunken, and more deeply eosinophilic (darker) staining cytoplasm of the *glomerulosa* cells. Many of these have hyperchromic, irregular, often shrunken pycnotic nuclei. This illustration should be reversed.

3C. ♂1961. This immature deer was obviously in weak condition when shot at the time of the die-off. More advanced degeneration of the cells of the *zona glomerulosa* with several foci of hemorrhage are shown, suggesting over-stimulation.

3D. ♂2454, typifying the appearance of the adrenals two years after the die-off (1960). Degenerative changes are less marked than in 1958 and 1959, although pycnotic nuclei are still conspicuous.

Fig. 3E–H, a similar series of sections illustrating changes in the adrenal medulla before, during, and after the die-off, in the sika deer, *Cervus nippon*.

3E. ♂1635, illustrating the relatively normal apearance of the medulla typical in 1955.

3F. ♂1960, typical appearance of all deer examined at the time of the die-off, including the one which had died. Cellular degeneration with pycnosis is conspicuous; note the portion bordering the cortex in this section. Marked variations in cellular size suggest an active regenerative process as well as degeneration.

3G. ♂1963, shows evidence of recovery from the degenerative changes seen in 1958. Regeneration is indicated by the irregular nucleae and cell size, but evidence of active degeneration is largely lacking.

3H. ♂2450, typical appearance of medullas in 1960. Some evidence of degeneration and repair remains but, in general, the medulla appears normal.

in size the kidneys of these deer appeared essentially normal at autopsy. However, there were marked microscopic changes in the glomeruli.

The kidneys of two deer in 1955 (※1637, 1638) and all thereafter exhibited some degree of diffuse, non-exudative, proliferative glomerulonephritis. In its mildest form the disease was characterized by edema and swelling of the material in the intercapillary spaces (mesangium) of the tuft (Fig. 4B) compared to apparently normal or nearly normal glomeruli from 1955 (Fig. 4A). There was ischemia of the capillaries and partial collapse of the capillary basement membranes with restriction of their lumens. Some increase in the cellular elements of

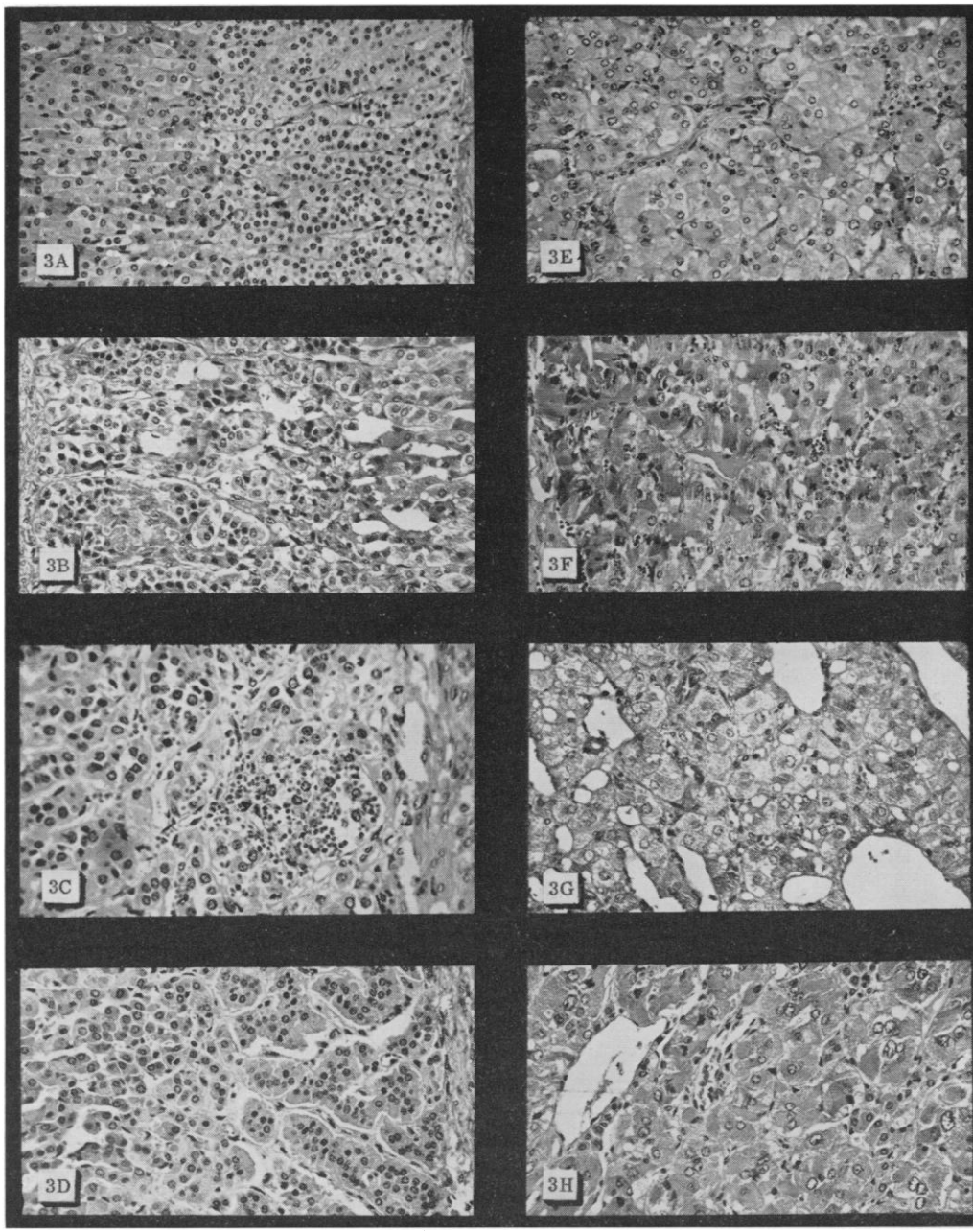

Fig. 3.

the mesangium and stalk was seen, especially in the periphery of the lobules, with distortion of the typical cloverleaf patterns of the capillaries. In more severe and probably more acute form these changes were all accentuated (Figs. 4C–4F). The capillary basement membranes generally were wrinkled and collapsed, frequently with total occlusion of the capillary lumens. Cellular hyperplasia was more marked and there was a marked deposition of PAS (Periodic Acid-Schiff) positive

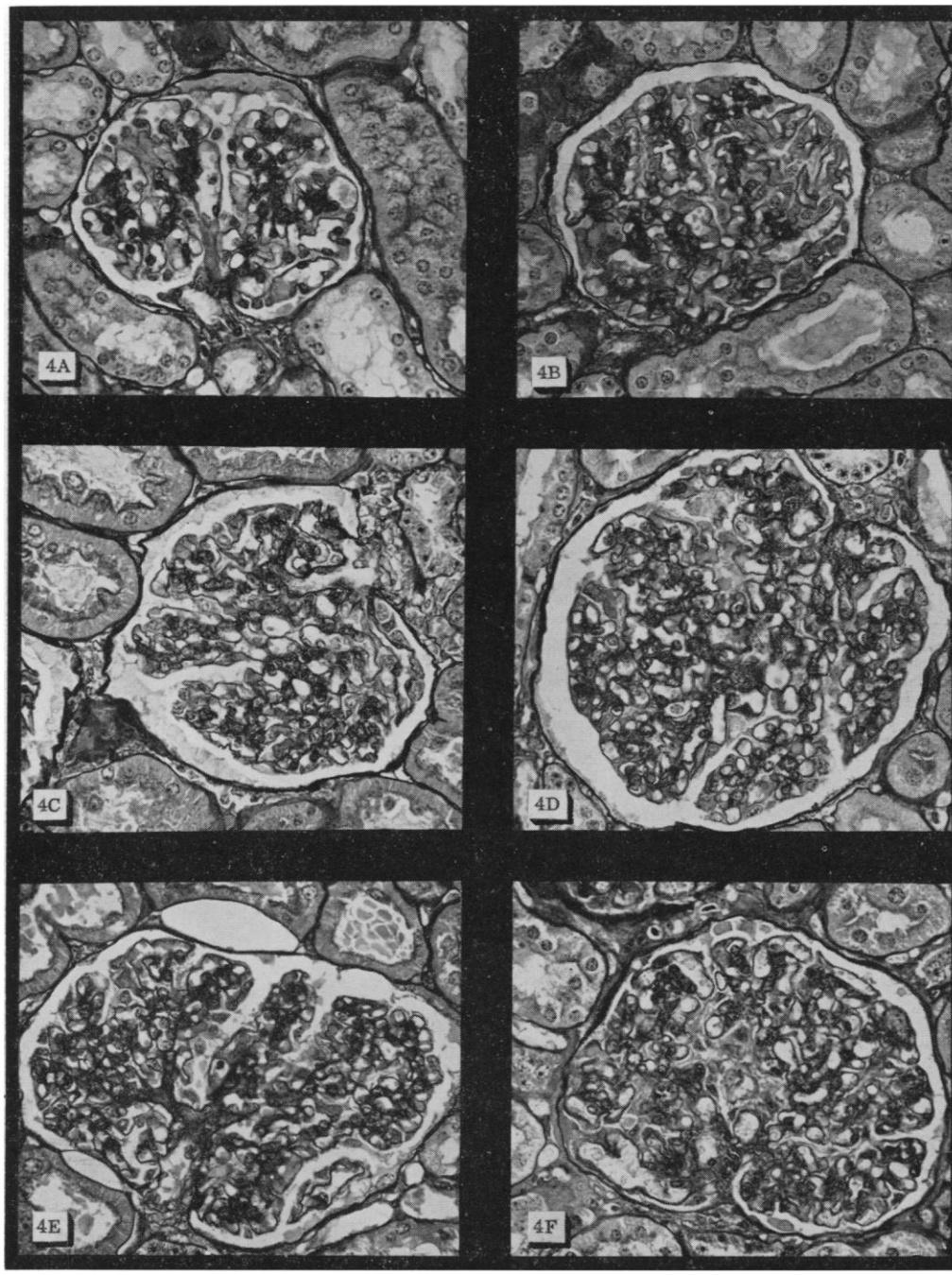

Fig. 4

fibrils in the mesangium, especially in the region of the bases of the capillaries. Infiltration by inflammatory cells or thickening of the capillary basement membranes was not observed. The presence of deeply eosinophilic, PAS positive casts in the lumens of many tubules, especially the collecting ones, indicated that the glomeruli were leaking protein. No epithelial crescents were observed. Adhesions between the tuft and Bowman's capsule were not seen. The changes seen in the glomerular structure of these deer are similar to those described for acute and subacute glomerulonephritis in humans (Grishman and Churg, 1957; Churg and Grishman, 1959) except for the absence of inflammatory infiltration and progression to a more severe form of the disease.

Involvement of the interstitial tissue and tubules was rare and evidently secondary to the glomerular changes, as it was observed only in the more severely involved kidneys. These changes assumed the form of occasional small foci of tubular atrophy associated with chronic inflammatory response, and were an inconspicuous part of the whole picture. This disease is notable for the general absence of an inflammatory response. In general, the tubules are entirely normal.

Juxtamedullary congestion was present in all deer shot.

Arteriosclerosis, characterized by moderate intimal proliferation in the larger arteries was seen in most of the more severely and apparently chronically involved kidneys (Fig. 6D). Arteriosclerosis also was present in a few of these.

The severity of the glomerulonephritis was graded on a basis of 1 to 5, 1 being entirely normal and 5 representing terminal, almost total destruction of the glomeruli. These gradings are given for each deer in Table 4. In 1955 the kidneys of only two deer (№1937, 1938) showed clear-cut involvement of the glomeruli (Fig. 4B). In these the glomerular tufts showed primarily an acute edematous glomerulonephritis with an increase in PAS negative intercapillary substance. In subsequent years the decrease became more pronounced with increased proliferation and scarring of the tufts (Figs. 4C–4F). The most severe subacute form of the disease was observed in the deer shot in 1959 (№1963) (Fig. 4E). All of the deer shot in 1960 showed renal disease even though some were not old enough to have experienced the population collapse (Fig. 4F), and the kidneys of №1961 which had been in weak condition in 1958 were not so

Fig. 4A–F illustrate the changes seen in the renal glomeruli of these sika deer, *Cervus nippon*, from 1955 through 1960. Renal tubules also are shown around the glomerulus in each figure. The normal appearance of the tubules is apparent irrespective of the changes in the population or degree of glomerular involvement. All sections 3 micra thick and stained by the allochrome method.

4A. №1637. Apparently normal glomerulus from an immature female in 1955. The renal glomeruli in three of the five deer shot in 1955 presented this appearance. Note the full expansion of the glomerular capillaries, the relative lack of intercapillary substance, and the lack of increased cellularity in the tuft in spite of it being an immature animal. However, note the pycnotic nuclei of the juxtaglomerular cells. × 440.

4B. №1638. Illustrates the glomerular disease as it was first observed in two deer in 1955. Cellularity is not increased noticeably, but a conspicuous increase in intercapillary substance with some wrinkling of the basement membranes of the capillaries and partial collapse of the capillary loops resulting in restriction of the lumens and an associated ischemia. These changes have been attributed to edema of the tuft in an early acute stage of glomerulonephritis (Grishman and Churg, 1957). However, since inflammatory cells are lacking the changes observed here perhaps more closely resemble those of nephrotic (non-exudative) glomerulonephritis. × 440.

4C. №1950. Illustrates more advanced glomerular changes in a mature female seen in 1957. Note the marked increase of cellularity of the mesangium with further restriction of the capillary loops and folding of the capillary basement membranes as compared to Fig. 4B. × 440.

4D. №1960. Renal glomerulus from a mature male at the time of the die-off. There is a marked increase in cellularity, especially in the mesangeal cells, with an increase in PAS positive fibrils (dark staining) in the intercapillary substance. Many capillary loops are almost totally occluded. Similar changes were seen in the deer which died, but they were less pronounced, especially the loss of potency of the capillary loops. × 440.

4E. №1963. Deer shot in 1959. All of the changes seen in 1958 (Fig. 4D) are present here, but scarring of the tuft, as indicated by the increase in PAS positive fibrils in the intercapillary substance is somewhat more pronounced. × 440.

4F. №2454. Illustrates the typical appearance of glomeruli in the deer in 1960. Changes in the glomerular tufts resemble those seen in the preceding year (Fig. 4E) except for the more open capillary loops. The tuft does not present the condensed appearance of the one preceding. However, scarring and increased cellularity of the tuft is prominent. These changes suggest a return of more normal function following severe involvement. Note the precipitate in Bowman's space. × 360.

TABLE 4.—Pathologic condition of adrenals, kidneys, and livers of sika deer, *Cervus nippon*.[1]

Deer Number	Adrenals		Kidneys					Livers			
	Pycnosis in glomerulosa	Casts	Inflammatory foci (atrophied)	Pycnosis	Arteriosclerosis	Arteriolosclerosis	Glomerulonephritis	Glycogen	Inclusions	Clefts	Severity of disease
1634	—	—	—	—	—	—	1.5	2	—	—	1
1635	—	—	—	—	—	—	1.5	2	—	—	1
1636	—	—	—	—	—	—	2.0	2	—	—	1
1638	—	1	—	—	—	—	2.5	2	—	—	1
1637	—	—	—	—	—	—	1.5	2	—	—	1
1950	—	1	—	—	1	—	2	—	—	—	—
1959	1	1	—	—	—	1	2.5	1	2	2	3
1960	1	1	1	—	—	—	3.0	1	2	1	3
1961	2	1	—	—	—	2	2.5	2	1	1	2
1962	1	—	—	—	—	1	2.5	1	1	2	2
1963	3	2	2	—	—	—	3.5	1	1	2	4
2451	1	1	—	1	2	1	2.5	1	2	2	3
2452	1	1	2	—	1	1	3.0	1	3	2	5
2454	1	1	—	—	2	2	3.0	1	4	2	4
2453	1	—	1	1	1	—	2.5	1	2	1	2
2455	1	—	1	—	—	—	2.5	1	1	2	2
2450	1	2	1	1	1	—	3.0	3	1	2	2
2456	1	1	—	—	2	—	2.5	4	1	1	2

[1] Grading of the severity of renal disease is on a basis of 1 to 5, where 1 is normal and 5 essentially total glomerular destruction. Renal grading is based on a scale established from other species. Hepatic disease is graded 1 to 5 where 1 is normal for the total series of these deer and 5 the most severe seen within this series.

severely involved as were those in later years. Except for a moderate degree of terminal passive congestion, the kidneys of the deer which died in 1958 resembled the others from that year, even with somewhat less severe glomerulonephritis. There appeared to be some return of circulation through the glomerular capillaries in 1960 (Fig. 4F), suggesting the beginning of resolution of the disease, although there was still a reasonably severe subacute or chronic glomerulonephritis. It is clear that these deer suffered from a chronic nonexudative, proliferative, intercapillary glomerulonephritis which began to make itself known in 1955, increased in prevalence and severity until 1958 (at the time of the die-off), and persisted for the following two years with an increase in severity, but showed some signs of resolution in 1960. This disease became evident at least three years prior to the die-off and continued for at least two years afterwards.

Liver: In 1958 hepatitis first appeared in the livers of the deer, those which were shot as well as in the one that died. This disease was characterized by active periportal necrosis and regeneration with cellular enlargement, and by the presence in many parenchymal cells of eosinophilic intranuclear inclusions closely resembling those seen in the cytomegalic inclusion disease of the renal tubules of urban rats (Figs. 5B, 5H and 6C). The presence of peculiar intracytoplasmic, semilunar clefts (Figs. 5B–5H and 6A–6C) in paraffin sections of the parenchymal cells was also characteristic. However, frozen sections showed these clefts to be refractile, yellowish, curved, double tapering rods which failed to take either nuclear or Sudan IV stains. Evidently the rods were soluble in one or more of the organic solvents used to process paraffin sections, but not in water. The clefts were largest and most conspicuous in the areas of the hepatic lobes uninvolved in the clearly acute phase of the disease, and were much smaller, more abundant, and less easily distinguishable in necrotic cells (Figs. 5B, 5F and 5H). None of these livers gave evidence of inanition. This statement is based on the lack of shrunken, deeply staining parenchymal cells with a loss of glycogen and cytoplasmic nucleic acid.

The intranuclear inclusions varied considerably in size and form and were more common in the periportal areas. The variations in the morphology of the inclusions are comparable to those described by Randall and Bracken (1957) for experimental hepatitis in hamsters, produced by equine abortion virus. They were eosinophilic, PAS negative, granular, round bodies producing margination of the chromatin and stained very pale blue with the allochrome stain. There was rarely more than one per nucleus.

Frozen sections stained with Sudan IV showed that intranuclear fat globules often, but not always, were associated with the inclusions. The fat globules varied from one to 20 or more per nucleus, but occurred in not more than a quarter of the cells.

Fat stains on frozen sections also showed that fatty degeneration of the hepatic cells was extremely rare, being found only in one small focus of one liver (※1963). What appeared to be fat vacuoles in some of the parenchymal cells in standard preparations did not stain for fat in frozen sections. The livers of mature males became much more severely involved than did those of mature females or immature deer, but this may have been partly due to age (Table 3). The liver of the male shot in 1960 (※2452, Fig. 5G), showed marked bile duct proliferation associated with a gross appearance of cirrhosis.

Hepatitis was not apparent in the deer in 1955 (Fig. 5A). At that time the livers contained moderate glycogen, roughly periportal in distribution, although the hepatic cells were shrunken and the blood sinusoids dilated.

The livers in 1958 showed a moderate degree of hepatitis, although inclusions were less common and the degree of degeneration and repair was less than that seen subsequently (Figs. 5C and 5D). However, there was pronounced cytomegaly and variation in nuclear size in involved areas (Fig. 5C). Little or no inflammatory response was seen. The intracytoplasmic clefts were conspicuous at this time. The level of glycogen in the 1958 animals

was somewhat reduced from the 1955 levels, but still present (Table 3).

The liver of the 1959 male (※1963) presented the most acutely involved of any (Fig. 5E and 5F). There was active necrosis and regeneration in the periportal areas, with marked enlargement of the cells and all stages of nuclear dissolution. Intracytoplasmic, semilunar clefts seldom were found in these acutely diseased cells, and glycogen was wholly lacking. Frozen sections showed no fat in the cytoplasm or nuclei of these cells, although intranuclear fat globules were present in many of the cells with clefts. Glycogen was present in the liver as a whole, in reduced amounts.

In 1960 the degree of involvement of the livers of mature males was obviously greater than in 1958, although less acute than in 1959. Table 4 and Figs. 5G–5H and 6A–6C summarize the results in 1960. Again, mature males were more severely involved than were does or young. Inclusions were most abundant in these deer and intranuclear fat vacuoles were conspicuous (Figs. 5H and 6C). All of the elements previously noted were seen in these deer, and in addition there was frank proliferation of the bile ducts of male (※2452) (Fig. 5G).

The lungs, GI tracts, and other organs were essentially negative except for a marked reduction in the lymphoid elements of the spleens of all animals (Figs. 6E and 6F).

Discussion

Ten years ago it was postulated that the growth and declines of mammalian populations might be controlled by physiological responses to population density (Christian, 1950). The importance of the pituitary-adrenocortical and pituitary-gonadal systems was stressed at that time. Since then considerable evidence has been collected from the laboratory and from natural populations to support this hypothesis, and it has been shown that social pressures (intraspecific competition) associated with increased density are the primary stimuli to increased activity of the physiological adaptive responses (Christian, 1959). It should be emphasized for the present purposes that these responses also result in decreased resistance to disease (Christian and Williamson, 1958; Davis and Read, 1958) and may play an important role in the production of other diseases of obscure etiology.

The data on this herd of sika deer present a number of questions, as well as provide some answers and a basis for tentative conclusions regarding several population factors.

This herd reached a density of one per acre sometime before 1955, then in two months of the winter of 1957–58, 60 percent died. The decline in population continued at a slow rate for another year, then the population began to increase again.

It often is stated that the decline in protein content of browse on winter range is critical (Longhurst *et al.*, 1952; Swank, 1956). However, the fact that the protein requirements of deer in good condition also decline (McEwen *et al.*, 1957) cast a considerable doubt on these conclusions. In terms of fat, glycogen, musculature, and general appearance the nutritional status of these deer was good and remained so with changes in the size of the population. In view of the present evidence the conclusions of Hayes and Shotts (1959) that malnutrition was an important factor contributing to the die-off of these deer seem untenable; so also does their statement that freezing of the mainland passage contributed to the development of malnutrition.

Disease often is said to be a major factor in population die-offs, but in this case disease could not have been a serious cause. Two diseases occurred in these deer—glomerulonephritis and inclusion hepatitis—but the history of each casts doubt on the possible role of either in the die-off. Hepatitis was first observed at the time of the die-off; it may have been present earlier but samples are not available to check. However, the hepatitis was relatively mild in 1958 and increased in severity in 1960. The available material suggests that this was a chronic, relatively mild hepatitis which would have been a factor in mortality only very late in its course, by the production of severe post-hepatitis cirrhosis. The data indicate that deer acquire the disease early in life and henceforth progresses slowly—probably a matter of years—to result eventually in a frank post-hepatitis cirrhosis. In no instance was cholemic nephrosis seen in association with the hepatitis, although there was a sufficient retention of blood pigments in numbers 1952 and 1954 to stain their adipose tissue yellow. There is no evidence to suggest that the hepatitis was severe in 1958, or that it directly contributed to mortality. The conclusion is

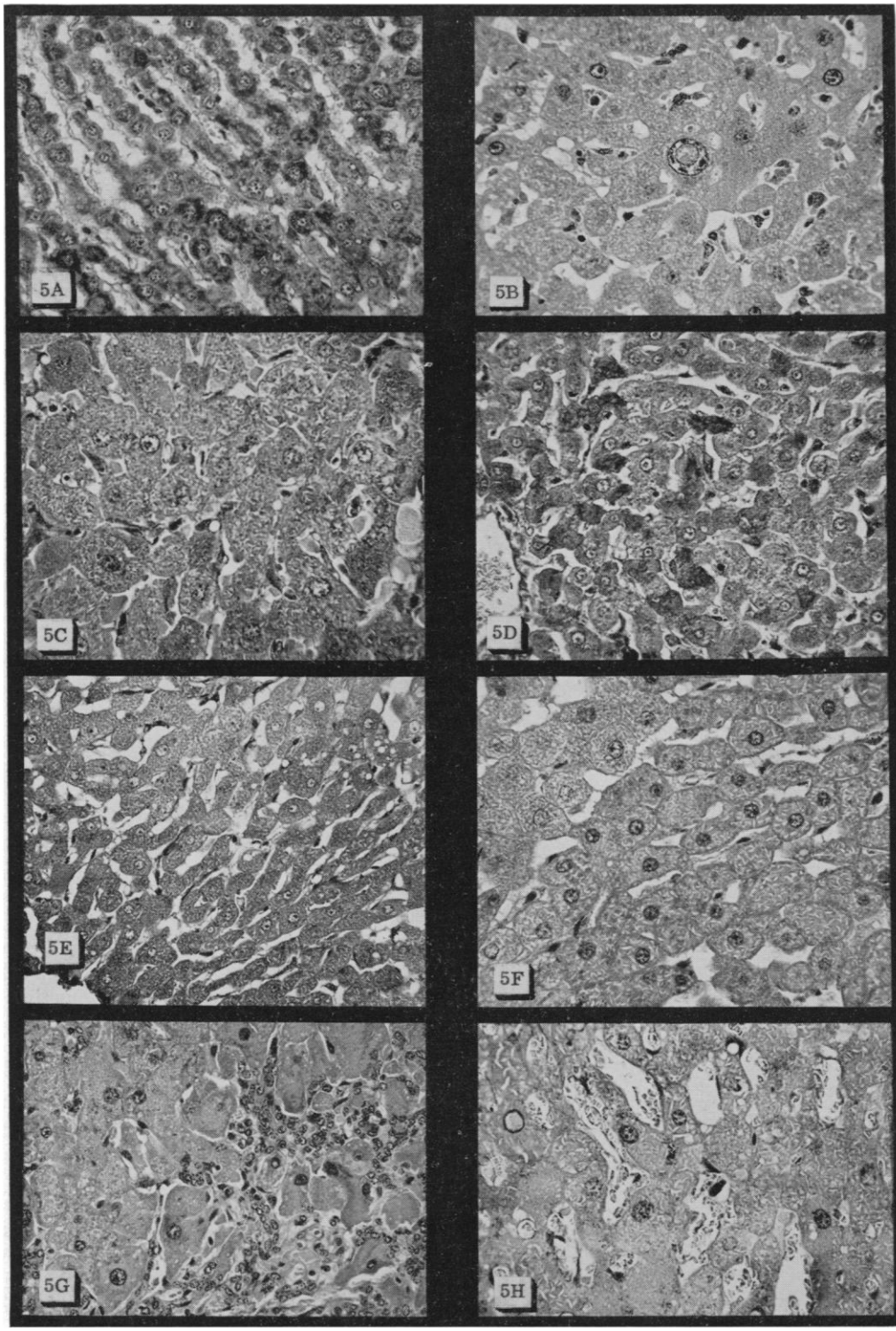

Fig. 5.

supported by deer ⁂1961, which appeared on the verge of collapse, yet had only a mild involvement of the liver (Fig. 5D).

Glomerulonephritis was first seen in mild form in some deer in 1955. It increased in severity through 1959, and remained severe through 1960. However, in no instance did it appear sufficiently severe to result in renal failure, although there was protein loss. Other studies on wild and captive mammals indicate that social strife is associated with the appearance of a similar glomerulonephritis (Christian, 1958 and unpub.) which may be associated with altered adrenocortical and other metabolic functions associated with increased density and strife.

It appears that neither renal nor hepatic disease contributed significantly to mortality. Indeed, it is much more likely that both of these diseases stemmed from altered metabolic factors concomitant with high population densities, which may play an etiologic role in the case of the renal disease and seriously lower resistance to infection in the case of hepatitis, as both diseases were much more serious a year or more after the die-off than at the time of its occurrence.

Hayes and Shotts (1959) have suggested that excessive browsing on pine, with the formation and absorption of pine oils, was responsible for the die-off by (1) direct toxicity and (2) sterilization of the rumen and consequent malnutrition. This diagnosis is unlikely for the following reasons: (1) the general well-fed appearance of the deer,

Fig. 5A–H illustrate conditions in the livers of sika deer, *Cervus nippon*, from 1955 through 1960, showing the inclusion hepatitis and its development from 1958.

5A. ⁂1638. Typical of the livers in 1958. The cells are somewhat shrunken and the sinusoids dilated in that year, a condition not noted subsequently. Note the uniform size of the nuclei and the presence of glycogen in the cytoplasm (appearing as black material in this photograph). Allochrome. × 360.

5B. ⁂1960. Liver of a 1958 deer with acute hepatitis. Note the large granular eosinophilic inclusions in the nucleus in the center of the field as well as bizarre chromatin pattern in some of the other nuclei. The liver cells are enlarged and the nuclei have almost disappeared from many. The beginnings of small semilunar clefts may be discerned in the cytoplasm, especially of the more peripheral cells. They appear to be absent from the cells in the center of the field which seem to be undergoing active degeneration. H and E. ×360.

5C. ⁂1960. Another field from the same section showing an area of less active degeneration. Darkly staining glycogen is present in the cytoplasm of many cells, the nuclei appear more nearly normal, and semilunar clefts in the cytoplasm are much larger and more conspicuous. One forms the impression that these less acutely involved cells may represent recovery from an active process. The appearance of the liver from the deer that died was essentially similar throughout to the condition shown in this figure. Allochrome. × 360.

5D. ⁂1961. Shows the comparatively normal appearance of the liver from the immature deer which appeared near collapse in 1958. There is some evidence of degenerative change, but is minimal compared to the preceding. Glycogen is abundant. Allochrome. × 360.

5E. ⁂1963. This liver from 1959 clearly had the most acute hepatitis of any seen. Note the nuclear vacuolation produced by fat. The cytoplasmic vacuolation shown was negative for fat. Semilunar clefts and necrotic cells are seen in this field. In spite of these alterations, glycogen is moderately abundant. Allochrome. × 212.

5F. Same deer. Active degeneration and regeneration are seen as well as an abundance of cells containing conspicuous clefts. H and E. × 360.

5G. ⁂2452. This liver from 1960 appeared grossly scarred and body and depot fat was deeply yellow. In addition to an active hepatitis, there is marked proliferation of the bile ducts. This liver had the appearance of having been involved in a chronic hepatitis of considerable duration. Degenerating and regenerating cells may be seen. H and E. × 212.

5H. ⁂2454. This liver from a mature male shot in 1960 had more inclusions and a more acute hepatitis than any other seen during that year. Large and small intranuclear inclusions of two types may be seen, as well as intranuclear fat vacuoles. The latter are perfectly clear, whereas the inclusions are pale grey (large), dark grey (small), or appear mottled. Aberrant chromatin patterns, semilunar intracytoplasmic clefts, and cells undergoing necrosis also are present. The fat of this deer also was stained yellow, but less deeply than in ⁂2452. Allochrome. × 360.

including an obviously weak one, (2) evidence that the degree of browsing on pine had not changed from periods before and after the die-off, (3) the absence of renal tubular lesions (Smith and Jones, 1957), (4) the absence of lesions in the liver consistent with poisoning by organic compounds, and (5) by the amount of pine bark or leaves which would have to be ingested in the winter to produce toxic amounts of pine oils, when it is considered that 3–15 cc of turpentine is the medicinal dosage for sheep (Stecher, 1960).

Factors correlated with changes in the population were growth, antler development, and changes in the adrenal glands. A remarkable decline in adrenal weight, representing a decline primarily in the

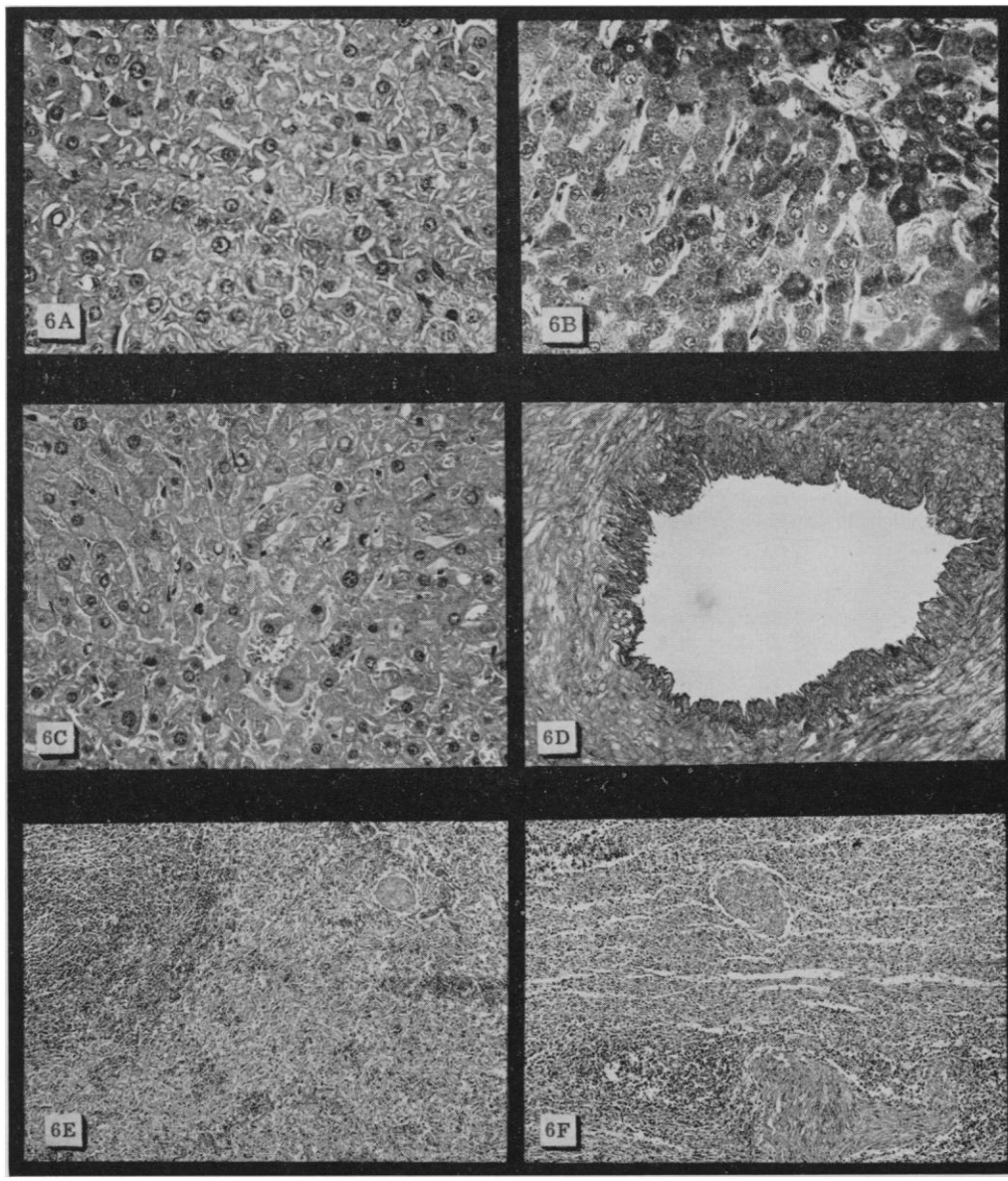

Fig. 6.

amount of adrenal cortical tissue, was seen after the die-off. Also, adrenal weight increased, particularly in the immature animals, from 1955 to the winter of 1958, and the subsequent decline was more marked than it was in the adults. These results coincide with those from other species in which a positive correlation between adrenal weight (as an index of cortical function) and population density has been shown, except that the magnitude of the response in deer is greater than had been observed heretofore. Inhibition of growth in association with increased pituitary-adrenocortical function is now well-established and is due, in part, to the growth inhibitory action of the corticoids, probably in part by inhibition of secretion of growth hormone from the pituitary (Christian, 1961).

The adrenal weight of male ✱2452 was greater than that of the others from the same sample, which is consistent with the presence of hepatic cirrhosis (Bloodworth and Sommers, 1958).

The marked pycnosis and apparent hyperplasia of the adrenal *zona glomerulosa* from 1958 through 1960 is not thoroughly understood. These changes suggest severe stimulation with active degeneration and regeneration. The glomerulosa secretes aldosterone, which regulates electrolyte fluid metabolism, and the secretion of aldosterone is stimulated by increased potassium, a fall in blood volume or, to a much lesser degree (and only for short periods) by ACTH. Therefore there is a suggestion in the morphological appearance of the glomerulosa that from 1958 on there were chronic fluid-electrolyte disturbances in these animals. Possibly the appearance of the glomerulosa reflects fluid and electrolyte disturbances in association with glomerulonephritis. The persistence of these changes in the glomerulosa through 1960 would be compatible with such an interpretation. Little can be said further except to note that the one animal (✱1961) which, in 1958, appeared on the verge of collapse, had "tubule formation" with hemorrhage of the *zona glomerulosa*, suggestive of extreme stimulation. The behavior of this animal would be compatible with acute adrenal insufficiency and electrolyte disturbance.

The cellular degeneration, degranulation, pycnosis, and variability in size of the adrenal medullas in 1958 suggest active degeneration and regeneration, resulting from excessive stimulation.

Hayes and Shotts (1959) reported degeneration of the medulla in the deer they examined, which had succumbed during the die-off. This condition was not noted in 1955, 1957, or 1960, and was only slightly evident in 1959, suggesting recovery. Therefore there is evidence of a marked increase in sympatho-adrenal activity in 1958. It has

Fig. 6A–C. Further illustrate conditions in the livers of sika deer, *Cervus nippon.*

6A. ✱2455. This liver of an immature male shot in 1960 showed less severe involvement than the two preceding livers. However, intranuclear fat vacuoles and intracytoplasmic clefts may be seen. Inclusions, although present, were not common. A small inclusion may be seen in the upper right corner of the field. The darker staining cells contain glycogen. The livers of numbers 2451 and 2453 appeared essentially identical to this except for a much greater number of inclusions. Allochrome. × 360.

6B. ✱2456. The liver of a pregnant female shot in 1960. Relatively mild hepatitis with the formation of clefts. The marked abundance of glycogen is shown by the darkly staining cells. The liver of the other pregnant female from 1960 (✱2450) was similar. Allochrome. × 212.

6C. ✱2454. A lower power photomicrograph of the same liver as shown in Fig. 5H, to illustrate the abundance of inclusions, variability in cell and nuclear size, and the wide distribution of clefts. These last are particularly noticeable in the smaller and seemingly less actively involved cells. H and E. × 212.

6D. ✱2454. A typical illustration of the relatively mild arteriosclerosis, characterized by intimal proliferation, seen in the larger renal arteries of a sika deer, *Cervus nippon,* in 1958, and many in 1959 and 1960. Allochrome. × 118.

6E and F. Spleens of sika deer from 1958 (numbers 1959 and 1962). Both show a marked reduction of the lymphoid elements in contrast to the amount one expects in a deer spleen. The spleens maintained this appearance from 1955 through 1960, perhaps somewhat more marked in the last year. H and E. × 85.

been established that physiological stress can result in a marked increase in the secretion of epinephrine and norepinephrine (Elmadjian *et al.*, 1958) and it is suggested that excessive sympathico-medullary stimulation and exhaustion, accompanying increased adrenocortical activity resulting from social competitive factors may have played a leading role in the die-off of these deer.

It is worth mentioning the diagnosis of malnutrition by gross inspection of the long-bone marrow at this juncture.

Loss of fat and gelatinous appearance of the marrow of long bones often is considered diagnostic of malnutrition in deer (Cheatum, 1949). However, there is reason to doubt the specificity of the disappearance of fat from the marrow. It is likely that any condition characterized by a negative nitrogen balance and loss of fat elsewhere can produce a fat-free, gelatinous appearance of the marrow. The marrow was examined in only two of the sika deer collected, therefore cannot be evaluated. However, changes similar to those ascribed to malnutrition frequently have been observed in the marrow of well-fed captive mammals. For example, at the Philadelphia Zoological Garden a barasingha deer and a white-tailed deer, each with quantitatively and qualitatively adequate food intake, died in extremely poor condition. They were wasted and their marrow was gelatinous with no fat. Both of these animals were subordinate males in their respective herds and were constantly subjected to attack and mistreatment, and, in fact, both were killed by the dominant male. These histories are typical for subordinate animals in penned groups. The value of the marrow as a specific diagnosis of malnutrition is open to serious question, unless one defines malnutrition in terms of basic anabolic-catabolic balances rather than in terms of food intake.

Malnutrition, pine-oil poisoning, and disease have been eliminated as important factors in causing the decline in size of the deer in this herd and their die-off in 1958. However, the data lead to the conclusion that adrenocortical, sympatho-adrenal, and other metabolic responses known to occur in increased density (therefore social strife) are primarily responsible for the die-off of the deer. Disturbed electrolyte and fluid metabolism may have been a part of this picture. The glomerulonephritis and helatitis described, more than likely are results rather than causes of the changes taking place in the population, and in the adaptive physiology of the deer.

Summary and Conclusions

The sika deer, *Cervus nippon*, introduced onto James Island, Maryland in 1916 had reached a population size of 280–300 by 1955, a density of one per acre. Sixty percent of the herd, primarily females and young, died in January-February, 1958. A further decline of 18 percent from the 1958 level occurred in 1959. A study of these deer was begun in 1955 in anticipation of the die-off, and continued through 1960.

All deer appeared well nourished and free of parasitism throughout the study, although there was a marked inhibition of growth at the time of the die-off, which primarily affected those in their first three years of age. Recovery of normal growth occurred following the die-off. Adrenal weight was high from 1955 through early 1958, and had declined 50 percent by 1960. Adrenal changes were more pronounced in young than in adults, which may be explained by behavioral-dominance factors. Beginning in 1958 and continuing through 1960, degenerative changes were seen in the adrenal *zona glomerulosa*, suggestive of over-stimulation. These changes may have been associated with some disturbance in fluid-electrolyte balances. The adrenal medulla showed marked degenerative changes suggestive of sympatho-adrenal over-stimulation, at the time of the die-off, with recovery in subsequent years.

A diffuse, non-exudative, proliferative, intercapillary glomerulonephritis is described. This disease began to make its appearance in 1955 and by 1958 involved all deer. Its peak evidently was reached in 1959, although all deer in 1960 were involved. At no time did this disease appear severe enough to be the direct cause of mortality.

A chronic, apparently viral, inclusion hepatitis, involving all deer collected, was first seen in 1958, but reached greatest

severity in 1959–1960. Its severity appears to have been related to the age of the deer —the older the more severe—and may affect males more than females. The history of this disease suggests that reduced resistance at the time of the die-off and peak population density accounts for its appearance. It was clearly not a cause of the die-off.

Malnutrition appears not to have been a factor in producing the decline of these deer, nor does toxic poisoning.

It was concluded that physiological disturbances, induced by factors associated with high population density, probably hierarchial-behavioral, were responsible for the deterioration and death of these deer, as well as for the manifestations of glomerulonephritis and hepatitis. These disturbances appear to be closely related to adrenocortical and sympatho-adrenal function.

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