overlapping experimentation and allow researchers to diversify more rapidly within a given area. Further, an investigator would not have to peruse each individual journal as it appears. On a subscription basis the service might become relatively self-supporting.

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Endocrines, Behavior, and Population

J. J. Christian and D. E. Davis devote the latter half of their article on "Endocrines, behavior, and population" (*1*) to discussing data from other investigators that are contrary to their theory that "the behavioral-endocrine feedback" is of primary importance in the regulation of mammalian populations. Concerning our work (*2*), for example, they make the following statement:

> In some situations no correlation has been shown between adrenocortical function and changes in population, but so far the cases fall into two categories. The first is that where the sample is too small to demonstrate any correlation. For instance, Negus studied only 98 animals over a 2-year period, of all ages and both sexes.

While we would not debate that a larger sample would have been desirable, our concern here is the considerable confidence these authors express in much weaker data of their own in support of their hypothesis. For example, in the paragraph preceding the one quoted above, the authors apparently place complete confidence in a paper published on sika deer by Christian, Flyger, and Davis (*3*), in which a total of 17 adrenal weights, collected over a 6-year period, is reported. These weights were recorded from animals of various ages and both sexes, and in 4 of the 6 years of study the sample consisted of either one or two adrenal weights. Histological evidence for adrenal changes is presented, but the critical period during the decline of the population is represented by adrenals from only three adult deer and two immature deer. In the summary of this study (*3*) the authors write:

> 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.

Yet no attempt to study the deer's behavior is reported. The authors give two reasons for believing that excessive browsing on pine bark was unrelated to the decline in population: (i) that the deer appeared well fed and (ii) that the degree of browsing on pine was the same before and after the decline. The critical period 1958–1959 is represented by only three adults. Of these, two in 1958 were 8 and 9 kilograms lighter than the average of five adults during a favorable year. No data on browsing of the sika deer were collected. In view of the absence of behavioral data and the presence of numerous environmental circumstances relating to the decline, the conclusions reached in a previous paper by Flyger and Warren (*4*) seem more tenable. With reference to the identical declining deer population, they summarize the circumstances leading to the catastrophe as follows:

> (1) A large herd of animals had built up which severely overbrowsed their range.
> (2) A substantial amount of food was lost in a fire.
> (3) Severe weather conditions required greater food consumption.
> (4) The feeding area was restricted by an ice barrier around the island.
> (5) The deer were forced to eat unpalatable materials including loblolly pine bark containing pine oils.
> (6) The condition of very little food, severe weather and consumption of poisonous substances resulted in mass mortality.

We have here criticized the data relating to deer, but some of the other data offered in Christian and Davis's article are equally controversial (*2*). The self-contained nature of the Christian hypothesis has probably accounted for its widespread acceptance in textbooks. The validity of their theory is not at issue here, but it needs to be pointed out that the data on population dynamics are complex and variable, and the resolution of the problems will not be furthered by overzealousness in the defense of a particular hypothesis.

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Any reference to the validity of numbers implies a consideration of variability, comparability of pooled samples, adequate numbers of samples, and other conditions imposed by statistical considerations. Negus and Gould's discussion (*2*) of the relationship of adrenal weight to population status in *Oryzomys* is based on five samples containing both sexes and one of males only, taken irregularly in the approximately 3-year period of study. Moreover, they have pooled adrenal weights of male and female, young and adult rats in unknown proportions, a procedure that their own data indicate is invalid, as there are clear differences in adrenal weight, in the directions one would expect, between males and females in the two samples taken during times of reproductive activity. The variability of adrenal weights in most of their samples is such that much larger numbers would be required to demonstrate significant differences between samples. However, this variability could no doubt be reduced greatly by putting weights from mature females, mature males, and immature males and females in separate groups for each sample, if their first sample consisting entirely of males can be used as a criterion. The data as published do not permit a conclusion in favor of either their hypothesis or ours.

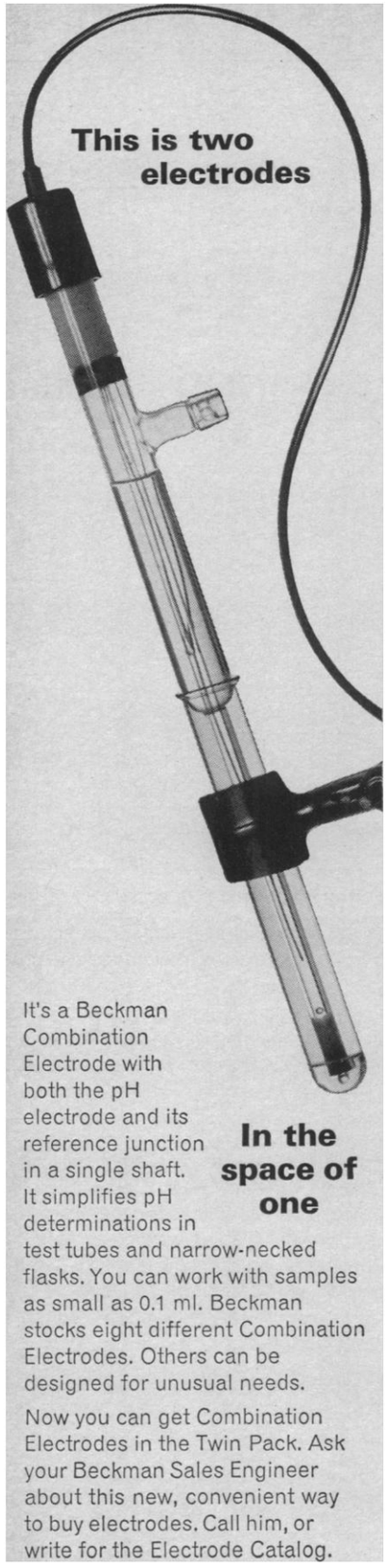
Aside from the fact that large numbers of deer are difficult or impossible to obtain, we submit that the data from our study of sika (*3*) are strong despite the small numbers, for the following reasons:

1) The numbers required are determined by the magnitude of the differences and the variances of the samples. In our samples there was no overlap in the adrenal and body weights by sex and age between samples taken before and after die-off.

2) Complete autopsies were performed and were supported by histological studies.

3) All samples were collected at comparable times of the year (late winter to early spring).

Regarding the decline in weight of the sika, the appropriate "favorable



year" for comparisons is 1960, not 1952–54, data from which are cited by Negus and Gould. However, this makes little difference so far as the data are concerned. In mature males 8 to 9 kg represents a 25-percent decline in total body weight from maximum weights or conversely a 44-percent increase over the weights at the time of the die-off—hardly an insignificant change, especially when there were adequate stores of fat in the usual depots, abundant for the time of year. These weight differences represented differences in the size of the animals, therefore reflected effects on somatic growth, not gain or loss of fat or other labile tissue mass.

With regard to the last point, we have recently learned that, owing to an editorial error, the illustrative photograph in our original paper (*3*) was one taken in 1959, not in 1958 as there indicated. The statement in the legend is, however, valid. Photographs taken in several years are available to illustrate our statement that the deer were not emaciated or suffering from a deficient food supply.

The earlier conclusions of Flyger and Warren (*4*) cited by Negus and Gould were based on superficial gross inspection without benefit of subsequent histological studies or of the sample collected and studied in 1960. To complicate matters, many of the dead deer were in advanced stages of autolysis, and all were autolyzed to some degree. We did not collect tissue from any dead deer, as we thought that in even the most recently dead animals autolytic changes were sufficient to impair reliable interpretation. Therefore we arranged to shoot a sample on our second visit during the die-off.

A second publication (*5*) presents strong presumptive evidence implicating potassium deficiency, secondary to prolonged hyperadrenalcorticalism, in the death of these deer. The deer feed extensively on submergent or emergent plants surrounding the island, of which there is a great supply, and the "ice barrier" existed for approximately 10 days in February, whereas the die-off began in January and continued through March. We cannot say that climate did not play an augmentive role in the die-off, but circumstantial evidence would tend to rule it out as an important factor. No die-off occurred in the years before or has occurred since, despite recurrent episodes of climatic adversity. However, we can state with assurance that the die-off was not caused by an inadequate intake of food, unless the deer died of malnutrition in the presence of adequate fat and liver glycogen stores and without emaciation.

We made observations, although not detailed ones, of social rank. Our supposition regarding social rank was based on the differential death of adult females and young of both sexes, and on reports in the literature on social rank in deer of several species (see *6*).

In support of our conclusions, Welch has shown a good relationship between adrenal change and population density in white-tailed deer (*7*).

In sum, it seems to us that the evidence provided by detailed autopsies and histological studies must assume greater validity than superficial gross examination. It would be difficult, if not impossible, to explain the appearance of renal glomerular disease well before the die-off on the basis of malnutrition; the presence of adequate fat stores in deer dying supposedly of malnutrition; the fact that the deer were stunted 3 years prior to the die-off and growth increased promptly afterwards; the chronic adrenal enlargement for 3 years preceding the die-off; the absence of indications of emaciation in the deer that was staggering and obviously dying (with hemorrhages in the adrenal zona glomerulosa); and, finally, the evidence, albeit presumptive, of potassium deficiency.

We expect variability and have never claimed that only endocrine mechanisms are capable of limiting population growth. We agree with Negus and Gould that further experimentation must provide the ultimate answers.

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