To the memory of my

father

Frank Marston Clulow

1913-1964

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Figure 1 is reproduced by kind permission of the author, Dr. J.J. Christian.

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ABSTRACT

Populations of various densities of eastern chipmunks (*Tamias striatus lysteri* (Richardson) and white-footed mice (*Peromyscus leucopus noveboracensis* (Fischer) were formed by the introduction of wild-trapped animals onto islands of different size on Heney (Little Whitefish) Lake, Quebec.

The animals were subsequently recaptured and various indicators of adrenocortical activity, including levels of circulating eosinophils and plasma corticosteroids, were measured. The evidence indicated that activation of the adrenal cortex is directly correlated with population density in *T. striatus* but not in *P. leucopus*.

The conclusions are discussed in relation to current theories on the regulation of mammalian population including J.J. Christian's hypothetical behavioural-physiological control mechanism.

RESUME

Des Tamias (Tamias striatus lysteri (Richardson) et des Péromysques (Peromyscus leucopus noveboracensis (Fischer) ont été capturés à l'état sauvage et ont été introduits sur des îlots du lac Heney (petit lac Poisson Blanc), province de Québec, de façon à former des populations de densités différentes.

Ces animaux furent recapturés après un certain temps et des indices de la fonction surrénalienne ont été mesurés. Les résultats obtenus indiquent que l'activité du cortex surrénalien, chez les Tamias, semble influencée par la densité de la population alors que ce ne serait pas le cas chez les Péromysques.

Ces conclusions sont discutées à la lumière des théories actuelles se rapportant au contrôle des populations de mammifères, celle de J.J. Christian plus particulièrement.

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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

In 1950 Christian (22) suggested exhaustion of the adrenopituitary system as the cause of cyclic declines seen in mammalian populations. Arguing on a priori evidence, he sought to explain the occasional mass mortalities of animals which have been observed in the absence of disease, food shortages or other known causative factors (53, 57, 60). In particular he considered deaths from "shock disease" which occur in snowshoe hares.

Shock disease had been observed by Green et al. (57) in snowshoe hare populations in Minnesota. In these animals this disease is characterized by fatty degeneration and atrophy of the liver, reduced liver glycogen, hypoglycemia, and convulsions preceding death. These changes have been found in many of the animals which succumb during the periodic die-offs of the species. Animals which are in convulsions can be revived by injections of glucose.

Selye (98) had already suggested that shock disease resembled a disease of adaptation - a condition found in the exhaustion phase of his general adaptation syndrome. Christian (22) proposed that the stresses inherent in a high population

density, along with those associated with severe climatic conditions and the demands of the spring breeding season, stimulated and finally exhausted the adrenopituitary system. He postulated that the pituitary was maximally stimulated at the beginning of the year to produce adrenocorticotropic hormone (ACTH). With the onset of the breeding season there was further stimulation of the pituitary, this time for the production of gonadotropins. As is known from the work of Selye (97) the pituitary cannot respond maximally to more than one stimulus. Christian suggested that in this case ACTH production was reduced to favour gonadotropin secretion leading to adrenal insufficiency. Gonadotropin secretion was presumed normal because the hares reproduced at the start of the population crash.

Christian subsequently produced other papers which altered his original hypothesis (26, 27, 28, 30, 34, 38). The modified hypothesis was that behavioural - emotional reactions to population density are the stimuli for endocrine responses within the animals constituting the population. These endocrine responses, occuring within the pituitary-adrenocortical axis, result in changes in mortality and natality rates which serve to maintain population stability or adjust the population level to new environmental conditions (38).

The scope of the hypothesis has also been changed. When first advanced it was to explain only sudden population declines. It is now applied to mammalian population regulation in general.

Proponents of the hypothesis agree that, at certain times and under special conditions, extrinsic factors may also bring about population regulation.

It is integral to Christian's hypothesis (Figure 1), that behavioural forces, stemming from the population itself and related to its density, are able to effect changes in the activity of the adrenal cortex which might affect mortality and natality. I shall show later that interactions between animals can cause such changes in the endocrine system. Before doing so, the control and function of the adrenal cortex will be briefly discussed.

A) Nervous system - anterior pituitary relationships.

Harris (62) points out that one of the ways the central nervous system alters the endocrine system in vertebrates is by integrating sensory messages which reflect changes in the "milieu intérieur" and conveying the need for certain endocrine regulatory factors to the glands concerned. This activity includes the transmission to the endocrine system of changes in the psychological state of the animal. It is precisely this portion of the "milieu intérieur" which

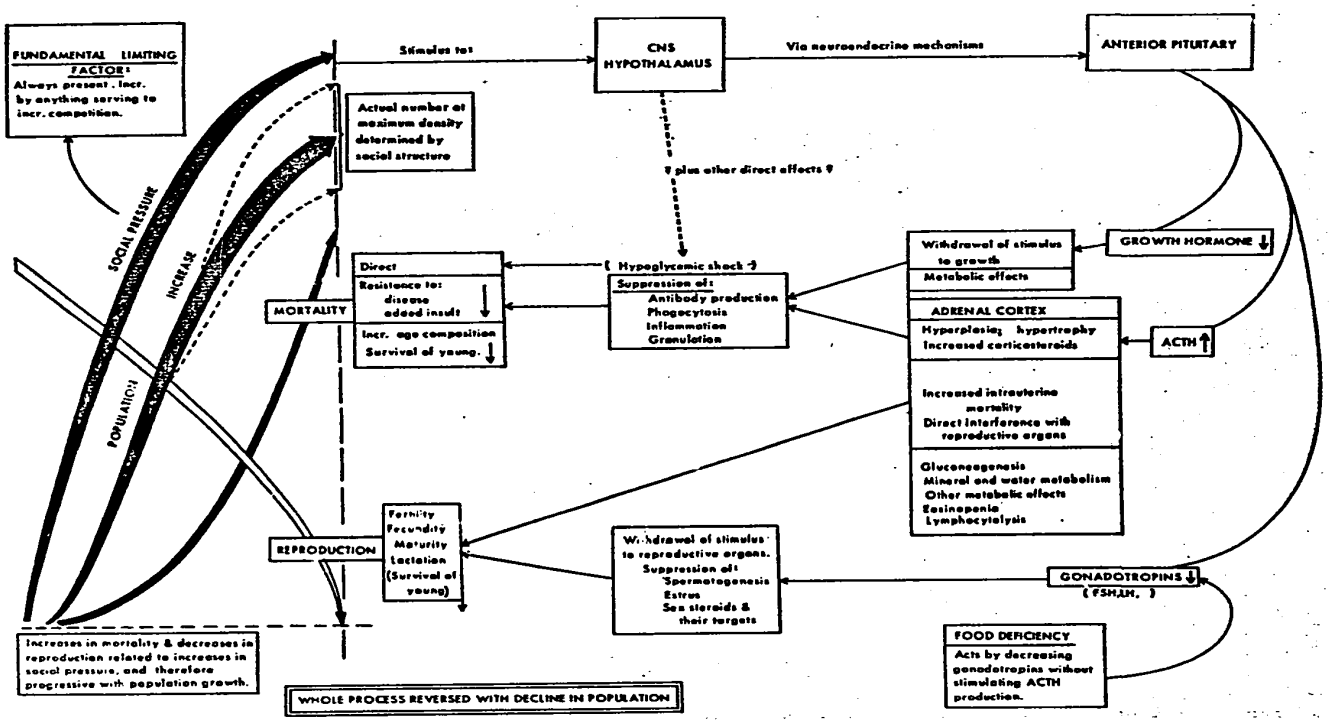


Fig. 1 Schematic summary of Christian's hypothetical feedback mechanism for regulating population growth (From Christian 1961)

is changed by the common interactions of animals within a population. As will be shown later, agonistic behaviours, i.e., those associated with challenge and fighting, are especially effective in changing the endocrine activities of an individual.

There is close physical contact between the brain and the endocrine system at the level of the hypothalamus - hypophysis complex. This complex has been considered as a functional unit by some authors (55,56). The limbic system (rhinencephalon), amygdaloid nuclei and the hippocampus have been shown to be the source of control impulses regulating the function of the hypothalamus in endocrine studies utilizing ether stress (68).

It is generally agreed that the hypothalamus is involved in the neural control of ACTH secretion from the adenohypophysis. Nalbandov (80) cites a number of workers who report blockage of stress-induced increases in ACTH production when they damaged parts of the median eminence or tuberal region of the hypothalamus in a variety of species. Stimulation of the median eminence enhances ACTH secretion.

From the knowledge gained from anatomical research and experimental surgery, it is certain that a neurosecretory mechanism is involved in the transmission of messages from the hypothalamus to the anterior pituitary (56). Anatomists have reported a portal system extending from the

median eminence of the hypothalamus to the adenohypophysis. It has been established that the blood in this portal system flows towards the hypophysis (56).

Subtle control of the anterior pituitary by direct nervous pathways can not be ruled out. Ganong (55) observed that potentials are evoked in the anterior pituitary after stimulation of the fornix, hippocampus or reticular formation. It is possible that these potentials are activity potentials of secretory cells.

Activation of the hypothalamopituitary unit is discussed by Ganong (55) who states: 'Emotions are particularly strong stimuli and it seems that all degrees of anxiety, fear, apprehension, anger and anticipation are equally effective'.

Ganong then proceeds to summarize the factors affecting the secretion rate of ACTH in the stressed animal. Two opposing forces act upon the anterior pituitary. Hypothalamic activation leads to an increase in the secretion rate of the hormone. Suppression of release is brought about by the circulating levels of adrenocortical hormones acting upon some unknown sites. The circulating level of ACTH also affects its own release from the adenohypophysis (63, 67).

B) Structure, function and control of the adrenal cortex.

Examination of the adrenal cortex reveals three morphologically distinct regions, the zonae glomerulosa, fasciculata and reticularis. There is equivocal evidence that these are also functional zonations. The relative sizes of the zones vary from species to species.

The outermost region, the zona glomerulosa, produces 18-aldocorticosterone and 11-deoxycorticosterone. Both are concerned with water and electrolyte balance. Of the two, 18-aldocorticosterone is the more biologically active.

Numerous hormones are released from the zona fasciculata. They include: hydrocortisone, corticosterone, cortisone, 11-deoxycorticosterone and 11-deoxy-17-hydrocorticosterone. These compounds are referred to as glucocorticoids since they principally affect carbohydrate metabolism. The zona fasciculata also produces a number of mildly androgenic C_{19} ketosteroids.

The zona reticularis is thought to produce the same hormones as listed for the zona fasciculata but under conditions of rest. When the cortex is stimulated the main release of corticoids is from the zona fasciculata (114).

Secretion of the glucocorticoid and androgenic hormones is controlled by the circulating level of ACTH. This hormone has two effects on corticoid secretion. First, an immediate increase in secretion rate follows an elevation in level of

circulating ACTH. If this high level of ACTH is sustained its effect is to augment the secretory capacity of the adrenal.

Associated with enhanced adrenocortical function is an enlargement of the gland. This is accomplished by cellular hypertrophy and/or hyperplasia. Caution must be exercised when interpreting cortical size and morphology because a larger size does not necessarily reflect a higher activity (25). Growth hormone from the pituitary can effect hypertrophy of the adrenal cortex (8). For this reason, data on adrenal gland size are only conclusive when considered along with histological information and other indices of adrenocortical function.

Cortisone, hydrocortisone and corticosterone, as previously mentioned, affect carbohydrate metabolism. Injection of the compounds produces the same effect as injecting ACTH. There is stimulation of gluconeogenesis from protein with resultant hyperglycemia and glycosuria with increased glycogen deposition in the liver. Through their effect of increasing deamination of protein residues, which are used in glucose formation, they also depress protein anabolism (52) with the result that there is an increase of the non-protein nitrogen in the blood and urinary nitrogen excretion (99). They also have an inhibitory effect on

lipogenesis; this is poorly understood.

The effect of these hormones upon growth is suppressive since they lower the rates of osteogenesis, chondrogenesis and connective tissue growth. Cortisone and hydrocortisone reduce considerably resistance to disease and injury. This is brought about by lowering inflammation, phagocytosis, granulation and antibody formation. Inflammation is reduced by failure of fibroblasts and lymphoblasts to appear at the site of injury. Injections of the compounds can cause mild diseases to become severe in animals (23).

High physiological doses of cortisone and hydrocortisone during pregnancy lead to an increase in the incidence of congenital malformations, especially cleft palate. In rats the compounds increase the rate of intrauterine mortality. In nursing rats injection of the compounds produces stunting in the suckling progeny. Newborn rats are affected in the following ways; growth is slowed, eruption of the teeth and opening of the eyes are advanced and morphological changes are induced in the skull and brain.

Involution of the thymus and lymphoid tissue generally is caused by glucocorticoids through degeneration and fragmentation of lymphoid cells and reduction of differentiation and lymphocytopoiesis (118). The resultant reductions in thymus weight, lymph nodes and malpighian corpuscles of the spleen can, if carefully used, serve as indicators of adrenocortical

activity. In some species, splenic hypertrophy follows stimulation of the pituitary-adrenocortical axis. This may be due to increased haematopoiesis. The level of eosinophils in circulation is reduced by increased adrenocortical activity and this parameter may also be used as an indicator of adrenocortical function (109).

Adrenocortical sudanophilia, resulting from staining sections out on the freezing microtome with one of the Sudan stains, is useful in evaluating the secretory activity of the region. The sudanophilia seen in the resting cortex is reduced in acutely stimulated glands (28).

The net result of the above effects, if brought about in a population of wild animals in their natural habitat, would probably result in increased mortality.

The indirect effects of adrenocortical function on reproduction is through the suppressive effect of the mild sex hormones, 11β-hydroxy-Δ4-androstene-3,17-dione and 11β-hydroxytestosterone and other C_{19} steroids, on gonadotropin production or release from the anterior pituitary. In hypophysectomized mice these steroids do not have enough potency to maintain the sexual accessories. However, they lower the pituitary secretion of gonadotropins in the rat. Selye (97) showed that alarming stimuli, which result in increased ACTH production and adrenocortical activity, also inhibit reproduction in the female rat. This is due to ovarian atrophy

or permanent anestrus.

Christian (29) showed that ACTH injections, at high physiological levels, can prevent maturation of intact female mice. The precise hormonal mechanism involved in this inhibition is not known.

The literature pertinent to the hypothesis published prior to 1963 was comprehensively reviewed by Christian (28). Recent literature, not included in his review, has been incorporated into the following survey. The review is presented under the same headings as those used by Christian.

C) Experiments in the laboratory with populations of fixed size.

Psychological factors, acting alone, have been shown to affect adrenocortical function in voles (21,40), mice (48), Norway rats (5), dogs (50), rhesus monkeys and humans (76).

Bronson and Eleftheriou (14) recently demonstrated that the mere sight of a trained fighter is a sufficient stimulus to elicit an elevation of free circulating corticosterone in mice which have had previous experience of fighting and defeat. It was previously shown that the bound fraction of plasma corticosterone in mice remains constant when the animals are stressed (15, 51).

Adrenocortical and other physiological changes have been related to group size in albino mice (23,25,94), white-footed mice (25), and woodchucks (10,11). The response of all animals to group size is not the same, the dominant animals are observed to be less stressed than the subordinates (26). This differential response was well illustrated in the experiment of Welch and Klopfer (119). An increase in mean adrenal weight, related to increase in group size in mice, was associated with an increase in its variance. This was due to the skewed frequency distribution curves.

Christian (26), working with house mice, and Bailey (4), working also with mice, have both demonstrated that the physiological responses seen in grouping experiments are not dependent upon the amount of space available to each animal. They obtained similar responses when populations of equal size were allowed to disperse over widely different areas.

The experiments of Christian (23), Southwick (108), Bronson (12) and Bronson and Eleftheriou (13) reveal that the responses of animals to group size is also related to the aggressiveness of the animals being used.

In other experiments it has been shown that grouping produces changes which are secondary to activation of the adrenal cortex. Individual growth rate is progressively reduced as population size is increased in house mice (25).

This suppression is less in the dominants than in the subordinates (26). Circulating antibody titre decreases with increase in group size (117), once again the dominant animals are less affected. Grouped as compared to isolated animals form less granulation tissue at the site of subcutaneous injection of turpentine (39). Resistance to invasion by larvae of the trichina worm Trichinella spiralis is reduced in subordinate animals as population size is increased in mice (49). Similarly, resistance to infection with intestinal parasites decreases directly with group size (84,85). Survival time for animals with tuberculosis decreases with grouping in animals (116). Reactivation of rabies virus has been observed in a guinea pig when it was exposed to the stress of grouping (106). Survival time, after exposure to X-rays, is less in grouped than in isolated animals (58); the mechanism involved is unknown.

Circumstantial evidence (90,92) suggests a direct relationship between group size and the incidence of coronary arteriosclerosis in animals and birds kept under zoological garden conditions.

The effects of crowding upon birds is similar to that upon mammals. Siegel (102, 103) has shown a positive correlation between adrenal weight and grouping in chickens and cockerels. Ratcliffe has shown a direct relationship between

grouping and coronary arterial disease in chickens (91). He also presented evidence (88) that the incidence of tuberculosis in captive wild birds increases with population density.

D) Freely growing populations.

In experiments conducted on populations of fixed size there is a sudden exposure of strange animals to each other. The question arises whether or not the same responses to grouping will be elicited from animals which have had time to adjust to the situation or which have been together since birth. To investigate this possibility, experiments have been performed in which animals are permitted to breed in a confined area. Such populations follow a sigmoid growth curve (121) and are self-limiting at maximum size (24, 25, 27, 41, 45, 69, 73, 93, 107, 113).

As a population increases the constituent animals show correlated increases in pituitary and adrenocortical function. These increases are greatest at maximum population size and approximately half as great at half the maximum size (24,73).

A relationship between sodium availability in the soil and population density of microtine rodents has recently been established. Aumann (2) noticed that natural populations of these rodents reach their greatest density in regions where there is an abundance of sodium present. Experiments

subsequently confirmed that sodium availability affected confined populations of Microtus pennsylvanicus (3). The authors suggest that access to unlimited sodium alleviates the stress caused by social interactions in high population densities. They speculate that failure of the adrenocortical regulation of sodium in dense populations may bring about population regulation.

Reproductive functions in the male and female are progressively inhibited as population level increases in confined populations. Inhibition is less in the dominant than in the subordinate animals (24). In males, inhibition of involution of the X zone of the adrenal cortex and decreased spermatogenesis are observable (24). In females the changes include: a reduction in birth rate (24, 25, 26, 38, 41, 45, 69, 78), an increase in the incidence of anestrus (45, 78), a greater number of females with adult size but juvenile reproductive organs (24), a reduction in the number of females which are pregnant at any one time (24) and an increase in the number of resorbing embryos (24, 78). Crowcroft and Rowe (46) found that the non-fecund females in a dense population become fecund when the population is permitted to disperse over a larger area.

Associated with female physiology at higher population levels of mice and voles is a decrease in litter survival (16, 24, 41, 69, 73, 107, 113). This may be due to a quanti-

tative decrease in lactation (24) or to changed parental behaviour (16, 73, 107).

Ratcliffe (89) presents data which show that a Myocastor coypus population, kept in the Philadelphia Zoological Gardens, was self regulatory through changes in infant survival.

£) Natural populations.

Observations and experiments on natural populations are few in number. Frequently, in a natural situation, it is impossible for an experimenter to control all the variables or to obtain samples of large enough size. It is often difficult for the observer to know which of the many variables to measure.

Adrenal weights have been observed to increase with population density in Norway rats (33). Artificial reduction of the population brought about a reduction in adrenal weight (32). Introduction of alien animals into an established population caused an increase in intraspecific competition (47). This was followed by cessation of population growth and an increase in mortality (47).

In the vole, Microtus montanus, adrenal weight follows population density very closely (1). At high population levels the heavy adrenals of Microtus pennsylvanicus are associated with eosinopenia (74). This is evidence of higher

adrenocortical function. Kalela (66) has presented evidence to show that reproductive rate is related to population density in wild vole populations (Clethrionomys rufocans).

A decrease in adrenal weight has been correlated with a decline in population density in house mice (21).

Adrenal weight has been related to population density in rabbits (123). Increased adrenal weight in dense wild populations of rabbits in New Zealand was not related to a decrease in general condition, as judged by fat deposition (123). In natural populations of rabbits which had been markedly reduced by an epidemic of myxomytosis, there was a higher reproductive activity as compared to that seen in the previous dense conditions (71).

In Marmota monax populations a reduction of adrenal weight followed a change in social structure brought about by trapping and removal of animals (72). This was interpreted as a reflection of reduced social pressure within the population.

Changes in adrenal histology and the incidence of renal disease has been observed in animals dying in a population of deer (Cervus nippon) during a period of mass mortality (36). It was later shown that ACTH injections can produce renal glomerular disease in woodchucks (38) and mice (31).

F) Criticisms of the hypothesis.

A number of workers have stated that extrapolation from dense laboratory populations to natural populations is not valid (19, 45, 82).

Christian and Davies (35) counter this by pointing out that only a very brief daily contact between animals in the laboratory is necessary to elicit activation of the adrenal cortex.

Other criticisms of the hypothesis, particularly those based on experimental work, are considered later in this thesis when they are discussed in connection with the results.

I wished to test that part of the hypothesis which relates adrenocortical activity to population density. In view of the paucity of field experimentation I decided to work with wild animals under near natural conditions.

CHAPTER II

METHODS AND MATERIALS

A) Experimental design.

The experiments involved exposing wild animals to different population densities and subsequently comparing their physiological conditions. Populations of known density were formed by placing animals on islands of measured, different areas. Most or all of the native mammals were removed from the islands, by intensive trapping, prior to the start of experimentation.

Vegetation, meteorological and topographical conditions were considered as constants in the experiments. To ensure the standardization of food supply, an excess was made available to the animals at all times. Population size was a constant in the experiments conducted in 1966.

The populations of different density which were produced were considered as points on hypothetical population growth curves. From Christian's hypothesis it was anticipated that the degrees of adrenocortical activity in the animals would be related to the respective

population densities.

A preliminary experiment, using Tamias striatus (Linnaeus), was performed in 1965 to test the experimental approach and techniques.

Experience gained in this test was utilized in designing the subsequent experiments in 1966.

3) Experimental areas.

a) Description

Islands were used in the experiments in order to reduce migration of animals to a minimum. Natural island populations of both species used have been reported by Werner (120) and Sheppe (101). Three islands (A, B and C) on Heney (Little Whitefish) Lake, near Gracefield, Quebec, (Figures 2,3 and 4) were chosen for their similarity and accessibility.

The vegetation of the area is typical of the Great Lakes - St. Lawrence Forest Region of Halliday (59), more specifically the Middle Ottawa Section (L4c)* of Rowe (95), in which it is included.

The islands themselves are elevated, rocky in places, and they support mixed woodland. Frequently occurring species of softwoods on the islands are eastern white pine (Pinus strobus), eastern white cedar (Thuja occidentalis)

*See map of Rowe (95)

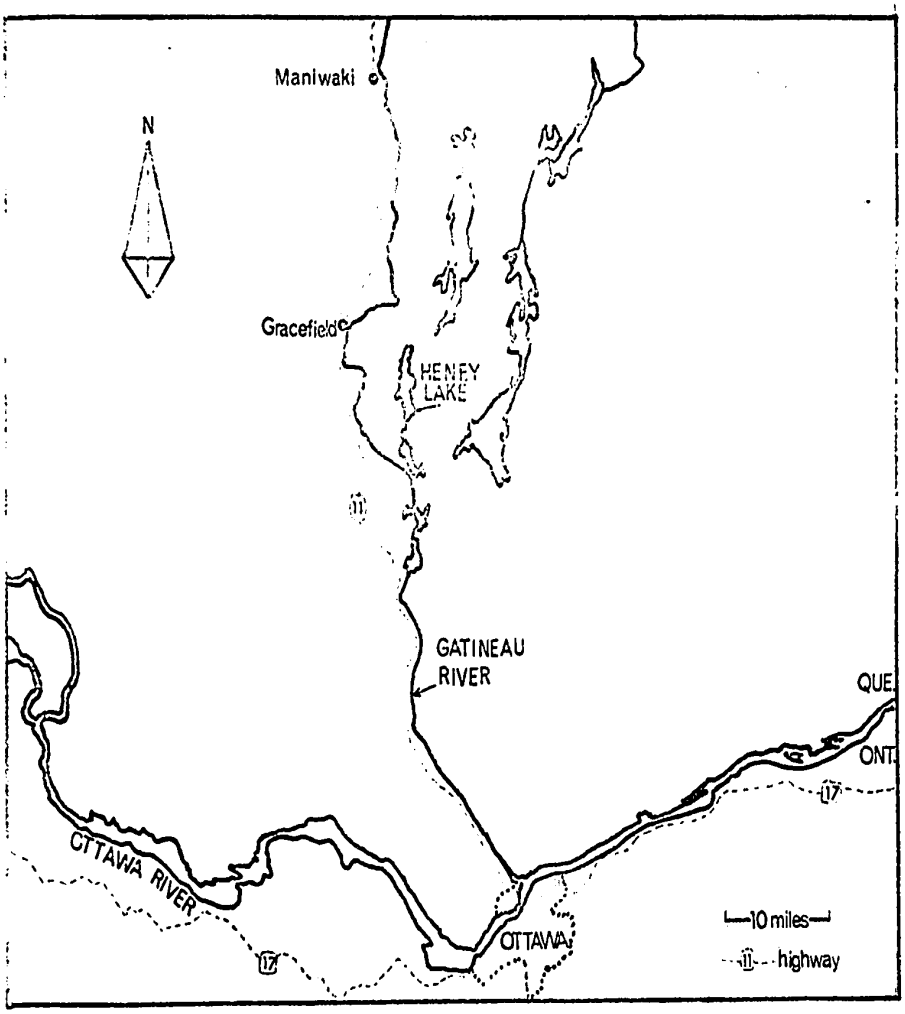


Fig. 2. Sketch map showing the location of Heney (Little Whitefish) Lake, Quebec.

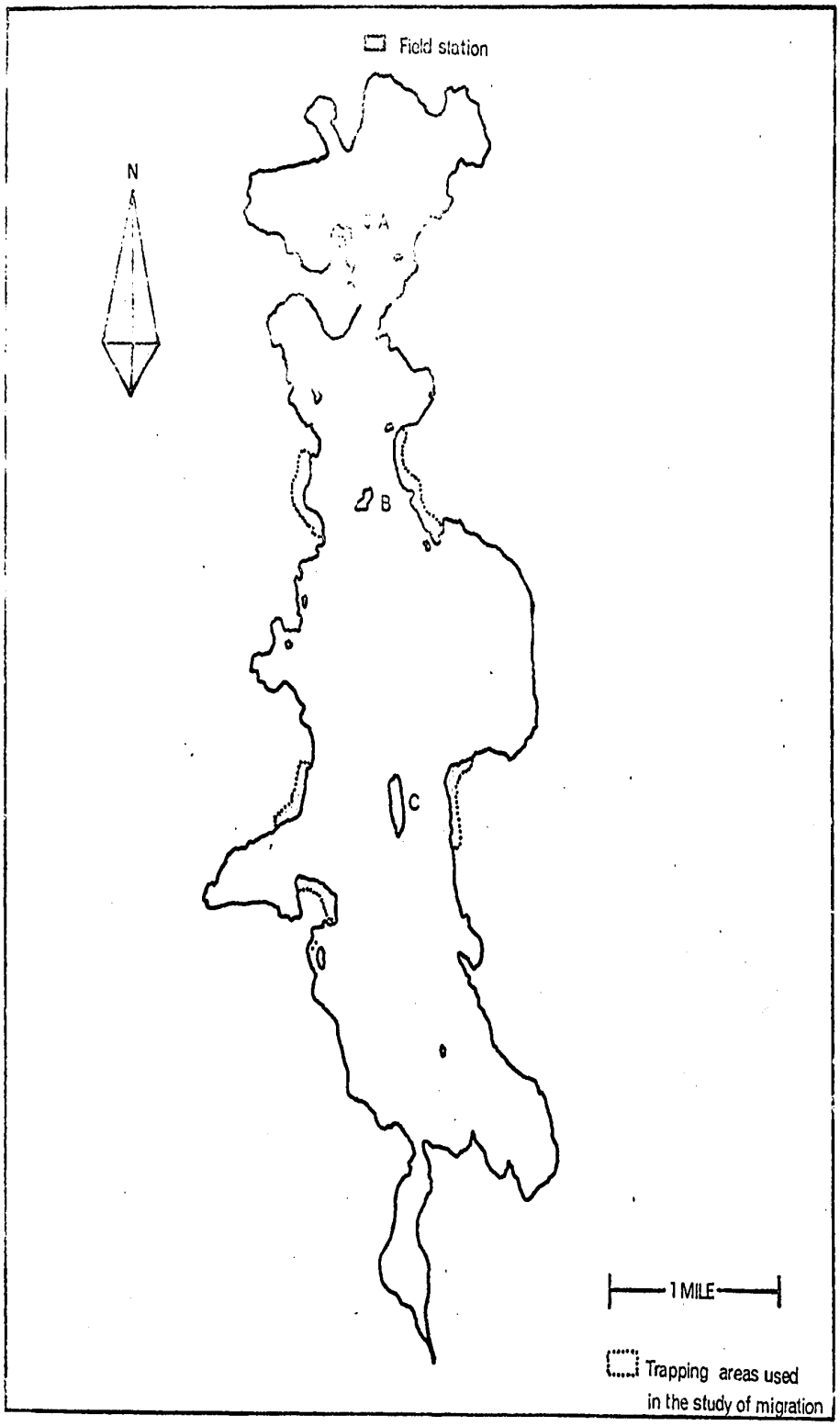


Fig. 3. Sketch map of Honey (Little Whitefish) Lake, Quebec, showing the location of the experimental areas.

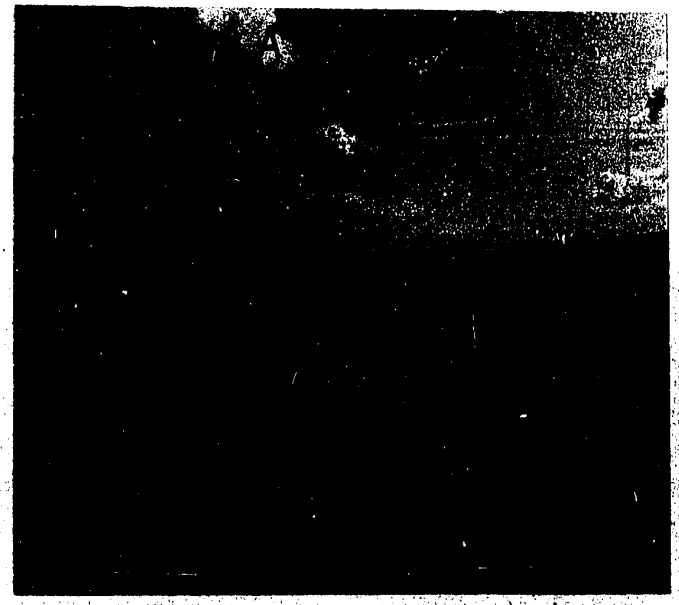
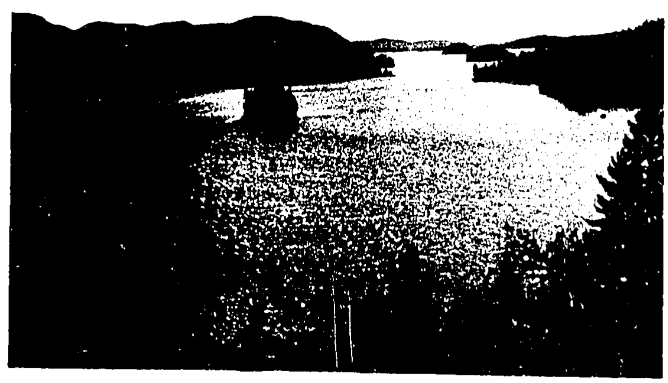


Fig.4 Heney (Little Whitefish)Lake, Quebec: View from the north showing experimental islands.



[illegible] Idaho, [illegible] from the north [illegible] islands.

and balsam (Abies balsamea). Deciduous trees include white birch (Betula papyrifera), basswood (Tilia americana) and northern red oak (Quercus rubra). The islands provide ample cover for small mammals in the form of undergrowth and fallen trees.

The islands have the areas and locations shown in Table I.

TABLE I

Sizes and distances from the closest shore of the islands

Island	Area (Acres)	Area (Hectares)	Distance from closest shore (feet)	Distance from closest shore (metters)
A	0.3	(0.12)	540	(165)
B	1.2	(0.49)	890	(271)
C	9.1	(3.68)	1,350	(412)

b) Preparation of the islands.

In an effort to remove the native small mammals, a trapping programme was carried out on each of the islands prior to the introduction of the experimental populations. Islands A and B, which were used both in 1965 and 1966, were found to have no native populations in either year.

Island C, used only in 1966, yielded numbers of mice, voles and squirrels during this preliminary trapping period. The voles and squirrels were released on the mainland, the mice were used in the experiments.

Each island was mapped and staked with a trapping grid. A 25' (7.6 meters) grid was marked on islands A and B, a 50' (15.2 meters) grid on C.

The supplementary food supply consisted of 'Purina Rat Chow' (Regd. T.M.). This was placed in 20 oz. cans, one of which was put at each grid marker. The food was replenished from time to time when the supply in the cans was becoming low. Experience with keeping and raising chipmunks and white-footed mice in the laboratory established that this food was an adequate diet for weaning and growth of the animals.

C) Experimental animals.

a) Source.

A total of 246 eastern chipmunks (Tamias striatus lysteri (Richardson) and 165 white-footed mice (Peromyscus leucopus noveboracensis (Fischer) were used in the two years of experimentation. All these animals, with the exception of a few chipmunks, were trapped in the immediate vicinity of Heney Lake. Animals trapped in the spring and

summer were used in experiments of the same year.

While in the laboratory, the animals were kept in individual cages with food and water ad libitum.

b) Preparation.

Before being released the animals were weighed, examined for sexual condition and individually marked (ear tags and/or clipping of the toes) for later identification.

D) Experimental procedures.

a) Trapping.

All trapping was done with Sherman Live traps, 12 x 3 x 3" (Figures 5 and 6); a mixture of rolled oats and peanut butter with a trace of almond extract was used as bait.

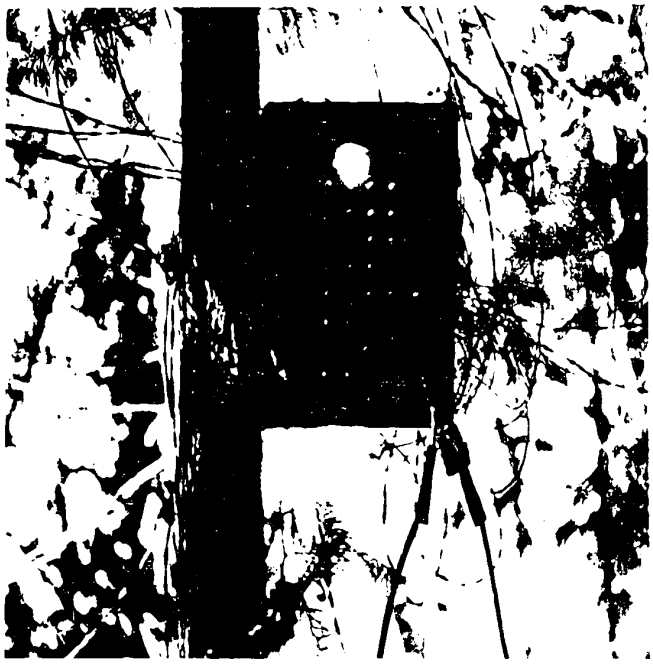
I wished to study a number of labile physiological variables which are probably affected by handling and trapping. To reduce the effects of these treatments to a minimum I devised a system of indicator lights which came on when a trap was sprung. When an animal entered a trap the time was noted and steps taken to retrieve the animal and anaesthetize it with ether as soon as possible. The efficiency of the procedure is discussed later.

A central console supported an array of flashlight bulbs, each one being connected to a separate trap (Figure 7).

Fig. 5 Typical arrangement of trap, stake, and food can. Trap in open position. (Note switch above door.)

Fig. 6. Typical arrangement of trap, stake, food can. Trap in closed position. (Note switch above door.)

Fig.7 Central console on island A. (The lighted bulb indicates that trap no.1 is closed).



A switch at the trap completed a circuit when the door closed (Figures 5 and 6). A 12 volt battery was the power source.

b) Formation of the populations.

The captured animals were divided as shown in Tables 2 and 3. Animals were first sorted according to sex and then assigned at random to the groups. In the chipmunk experiment of 1966 it was first thought that only enough animals could be captured to form two populations. With this in mind the animals trapped by the middle of June were divided into two populations (Islands B and C) with some remaining. Sufficient animals were subsequently captured in the course of mainland trapping that I was able, by adding them to the residue of the laboratory pcol, to form the population of Island A. Unfortunately, I was forced to include a disproportionate number of females, especially small ones, in order to arrive at the required number. This accounts for the mean body weight of the females of Island A being lower than either of the others. The sex ratios of the various populations were dictated by the availability of animals.

The initial densities of the populations assembled in 1966 (Tables 2 and 3) were chosen for two reasons:

1) I was concerned with the size of the populations which I could expect to retrieve at the end of the experiment. I knew, from the experiment of 1965, that popula-

Table 2. Populations of T. striatus

Populations	1965 Males	1965 Females	1965 Sex Ratio	1965 Density (Animals per acre)	1966 Males	1966 Females	1966 Sex Ratio	1966 Density (Animals per acre)
Island A	25	20	1.25:1	149.8	17	23	0.74:1	133.2
Island B	15	10	1.50:1	20.7	20	20	1:1	33.6
Island C	-	-	-	-	20	20	1:1	4.4
Laboratory	7	7	-	-	10	19	-	-
Wild	8	5	-	-	-	-	-	-

Table 3. Populations of P. leucopus

Populations	Experiment I				Experiment II			
	Males	Females	Sex Ratio	Density (Animals per acre)	Males	Females	Sex Ratio	Density (Animals per acre)
Island A	30	15	2:1	150.0	2[illegible]	[illegible]0	[illegible]	[illegible]0.0
Island B	30	15	2:1	37.4	20	10	[illegible]	24.9
Laboratory	8	7	-	-	-	-	-	-

tions would decline in numbers whilst on the islands.

2) I wanted to have population levels which bore some similarity to those which might be found in nature. The density of at least one population of each species falls within the range reported in the literature (Table 4).

When possible, samples of animals were killed to obtain information about their physiological state and variability under conditions other than those experienced on the islands. Samples of animals kept under both laboratory and wild conditions were taken.

c) Duration of the experiments.

The decline of artificial island populations of chipmunks was studied by means of census trapping in 1965. At that time it was noted that a fifty percent decline could be expected between twenty and forty days after introduction. In 1966 I decided that an exposure of about twenty-five to thirty days might be long enough to produce measurable changes and at the same time allow me to retrieve enough animals to obtain meaningful results.

The mice experiments had a duration of eleven to fourteen days. It has been shown that one week is adequate to obtain measurable stress-induced responses in laboratory populations of mice (108).

For reasons of convenience, the periods spent on the island by the various populations were sometimes staggered.

TABLE 4

POPULATION DENSITIES OF T. STRIATUS AND P. LEUCOPUS

	Reported Density (animals/acre)	(animals/hectare)	Author
T. striatus	20.0 - 30.[illegible]	8.1 - 12.2	Seton (100)
	4.0 - 15.0	1.6 - 6.1	Williams (122)
	0.8 - 8.7	0.3 - 3.5	Bole (9)
	0.8 - 3.6	0.3 - 1.5	Burt (17)
	1.1 - 3.2	0.5 - 1.3	Smith (104)
	0.1 - 0.7	0.1 - 0.3	Manville (75)
	4.1 - 29.4	1.7 - 11.9	Yerger (128)
	(4.4 -149.8)	(1.8 - 60.7)	(This study)
P. leucopus	3.08--10.87	1.3 - 4.4.	Burt (17)
	6.00- 7.00	2.4 - 2.8	Stickel (111)
	1.8 - 8.8	0.7 - 3.6	Hansen (61)
	1.1	0.5	Howell (65)
	0.5 - 2.9	0.2 - 1.2	Snyder (105)
	34.2	13.9	Bendell (6)
	4.1	1.7	Stickel (112)
	7.7	3.1	Sheppe (101)
	(24.9 -150.0)	(10.1 - 60.7)	(This study)

This permitted me to arrange for transportation of our equipment and field station from island to island and to stay on each for a few days to terminate the experiments.

The starting and finishing dates of the various experiments are given in Table 5. Because of the impossibility of trapping all the constituent animals of a population on a given day, the termination of each experiment was not clear-cut. The terminal dates which are given were arrived at as follows: trapping was continued until no more animals could be taken. The trapping records were then consulted and the day by which half of the total number of animals which were retrieved had been captured was stated to be the finishing date of the experiment.

Island C was not used during the experiments with P. leucopus because the wiring of its indicator light system had been damaged by vandals.

d) Investigation of migration.

Emigration of chipmunks from the islands to the mainland was studied in 1966 by placing dense batteries of traps on the mainland near the islands. The location of these trapping sites is shown in Figure 3.

Evidence of immigration of animals was looked for

TABLE 5

DATES AND DURATIONS OF THE EXPERIMENTS

	Start	End	Duration (days)
T. striatus			
Experiment I (1965)			
Island A	27 June	26 September	91
Island B	27 June	28 September	93
Experiment II (1966)			
Island A	11 July	4 August	25
Island B	27 June	22 July	26
Island C	2 July	29 July	28
P. leucopus			
Experiment I (1966)			
Island A	15 August	26 August	12
Island B	15 August	25 August	11
Experiment II (1966)			
Island A	27 September	10 October	14
Island B	21 September	3 October	13

in the trapping records at the end of the experiment.

e) Terminal phase of the experiment.

All animals of the same species were killed during the same time period each day in order to eliminate a potential variable. Chipmunks were trapped and killed on the islands between 17.00 hours and dusk. Mice, which are nocturnal in their habits, were taken between dusk and 3.00 hours.

The time elapsing between trapping and subsequent etherization was calculated for each animal. When most of a population had been captured, as judged by a sharp decrease in the number of animals being trapped each day, the timed trapping procedure was discontinued. Subsequently, captured animals remained in traps for an unknown period of time. Treatment of data obtained from these animals is discussed in the next chapter.

After being anaesthetized with ether the animals were weighed, on a diet scale, with a margin of error of 0.5 mg. Blood was then withdrawn into a heparinized syringe from the jugular veins and heart. A blood smear was prepared from each animal.

The rest of the blood was chilled by standing the tube in an ice-bath, and then centrifuged in the field. The plasma was kept cool in a similar manner until frozen later.

Internal organs were removed from the animals and fixed in buffered formalin. These organs were weighed to 0.1 mg at a later date.

f) Laboratory determinations.

The unfixed blood smears obtained in the experiment were stained with Giemsa stain. This procedure resulted in the destruction of all but the leukocytes. Differential leukocyte counts were then made on 500 cells and the percentage of eosinophils determined. Blood eosinophil levels were used as one criterion of adrenocortical activity.

The plasma samples from individual animals were analyzed for their plasma corticoid content by the micro adaptation of the method developed by Pelletier, Labrie and Fortier (87) for differential estimation of cortisol and corticosterone. This was a second index of adrenocortical function.

Sections were prepared of the adrenal glands using the freezing microtome. These sections were then stained with Sudan Black B for lipid. Photomicrographs were taken of these sections when interesting features were seen.

E) Statistical Techniques.

Student's 't' test was employed when only two means were to be compared. Analysis of variance was used where applicable in the analysis of the data. When significance was indicated, specific pairs of means were compared using the least significant difference method for unequal replica-

tion as described by Steel and Torrie (110). Results were declared significant when $P < 0.05$.

CHAPTER III

RESULTS*

A) Study of migration.

No alien animals were trapped on the islands after the start of experimentation. Trapping at the mainland locations shown in Fig. 3, during 1966, resulted in the capture of 38 T. striatus (and 108 P. leucopus) in approximately 4,300 trap-nights. None of these animals had been released on the islands. These observations led to the conclusion that migration was minimal or absent during the experiments. Bendell (6) reported only occasional movements of P. leucopus to and from the islands on which he was conducting his experiments. Bendell's islands were only 200 feet from the mainland.

B) Utility of the indicator-light system.

The system of indicator lights, for timing captures of animals, worked as designed. The only slight difficulty which was encountered was due to the formation of a white deposit between the galvanized steel of the trap door and the copper contact of the switch. This deposit prevented the circuit from being completed and the contact surfaces had to be cleaned with steel wool every two days or so.

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* Tables numbered with the suffix A are to be found in the Appendix.

Using the system, the elapsed time between trapping and killing of the animals was kept down to a few minutes (Table 6). The differences between mean elapsed times for the three populations of T. striatus (1966) were not significant ($P > 0.05$). In the case of P. leucopus the differences were significant in Experiment I ($P < 0.05$) but not in Experiment II ($P > 0.05$).

C) Retrieval of animals.

Census trapping of island populations during the T. striatus experiment of 1965 yielded the data plotted in Figure 8. The decline in numbers was due to either mortality or emigration. These two factors could not be distinguished.

The retrieval rates for the animals in the various experiments are seen in Table 7.

Data concerning animals which had spent unknown lengths of time in traps or which were captured a long time after the rest of a population had been killed were ignored. This was done because of the unknown effect of trapping upon such labile variables as blood eosinophil and plasma corticosteroid levels. Animals which were taken after the rest of the population had been killed had experienced a period of low population density conditions and were thus likely to be quite different from the other members of the population.

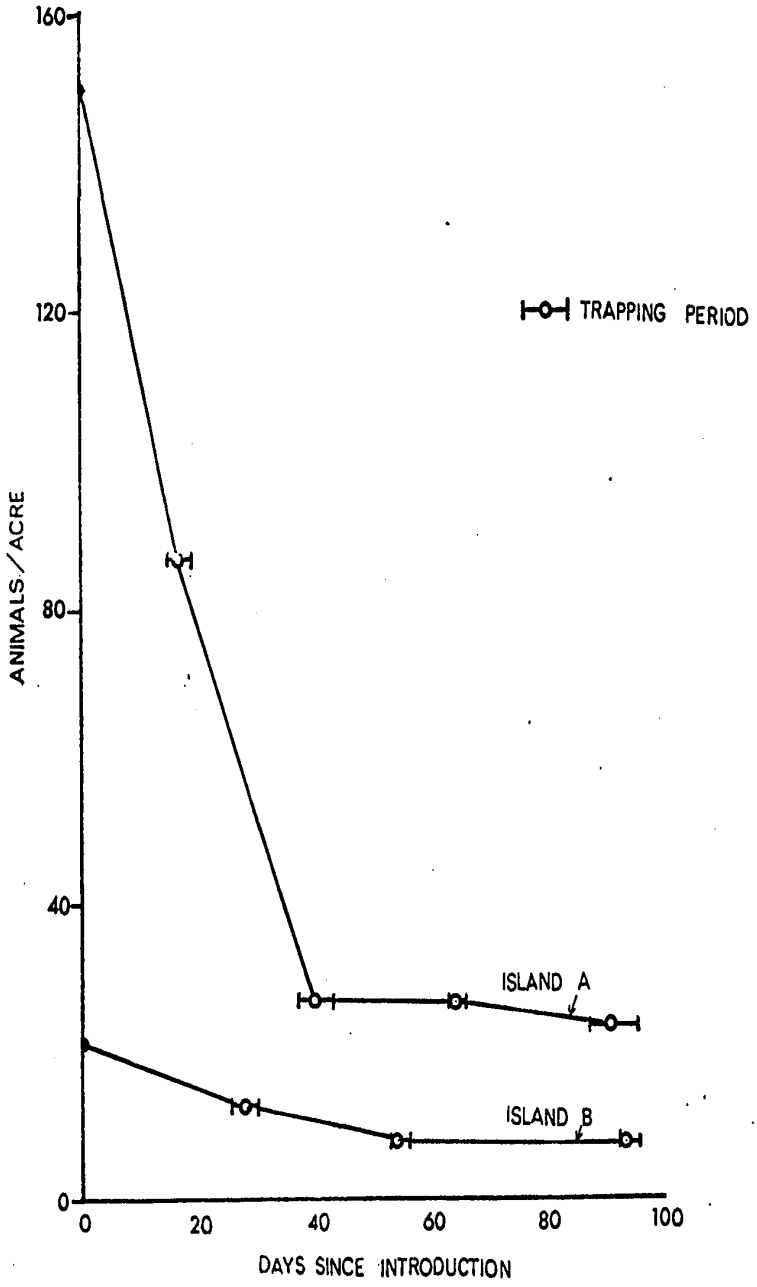


FIG. 8. Declines of island populations of T. striatus (1965).

Table 6 Elapsed time (minutes) between trapping and killing the animals

	T. striatus	P. leucopus	
	1966	1966	
		Experiment I	Experiment II
Populations			
Island A	1.4±0.3* (12)**	1.0±0.2 (10)	2.1±0.5 (13)
Island B	2.4±0.5 (14)	2.2±0.3 (16)	3.6±0.7 (8)
Island C	2.5±0.9 (10)	-	-

* = Mean ± 1 Standard Error

** = Sample Size.

Table 7. Retrieval of Animals (Animals recaptured/Animals released)

Populations	T. striatus		P. leucopus	
	1965	1966	1966	
			Experiment I	Experiment II
Island A	11*/45	17/40	32/45	19/30
Island B	9/25	20/40	32/45	21/30
Island C	-	27**/40	-	-

* = Includes 4 animals which were accidentally killed during the experiment.

** = Includes 5 animals which were accidentally killed during the experiment.

About a third of the chipmunks which were retrieved were found to be carrying the larvae of a parasitic dipteran bot fly of the genus Cuterebra. Cuterebra eggs are deposited on vegetation and gain entry to their mammalian host at an early stage in their larval development, possibly through wounds in the skin. The larvae migrate under the skin and most come to lie in the inguinal region where they remain for three or four weeks during which time they grow. Mature larvae force their way out of the host and drop to the ground where they burrow in and pupate. The flies overwinter in this condition and emerge as adults the following summer.

D) Results from the T. striatus experiments.

a) Body weights.

The mean body weight, at the time of release of all animals in the each population is given in Fig. 9 and Table 1A. The only significant difference ($P < 0.05$) is that between the females on island A in the 1966 experiment, and the females of the other two populations of the same year.

The body weights of the survivors, at the time of introduction were compared to see if body size was a factor influencing the survival of animals in different populations (Fig. 9 and Table 2A). There was no difference between the populations in this respect, except as stated previously in the case of the females of Island A, 1966, which were lighter than all others.

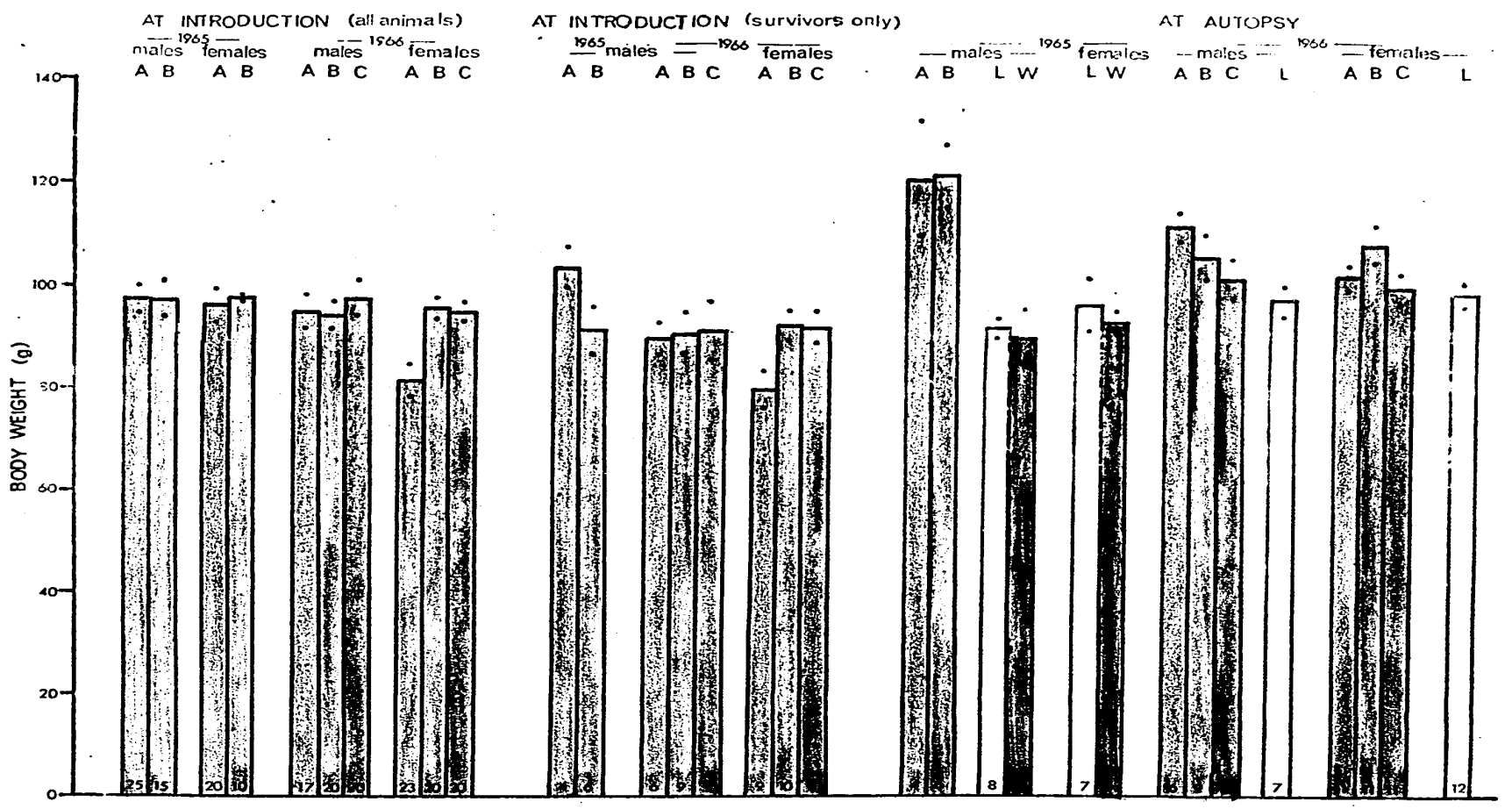


Fig. 9. Body weights of T. striatus (Mean ± 1 S.E. shown for each group)

The mean body weights of all populations just before death are given in Fig. 9 and Table 3A. In 1966 the females of Island C were lighter than those of both Islands A and B. This was the only significant difference ($P < 0.05$) noted between the island populations. Some of the differences in body weight between island, wild and laboratory populations were significant.

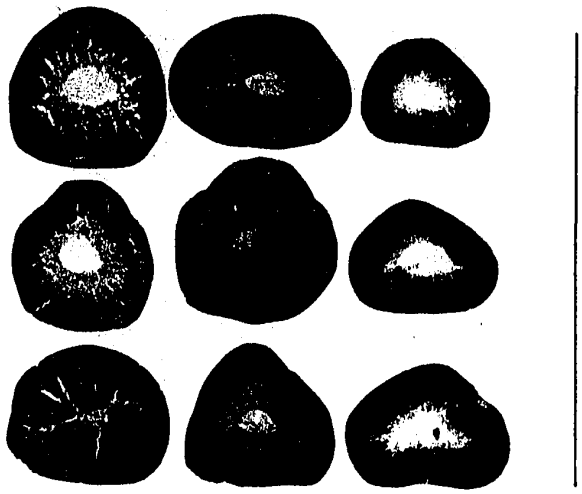
A comparison of the mean initial weights (Table 2A) with the autopsy weights (Table 3A) revealed that significant weight gains occurred in the animals of Island B 1965 (males $P < 0.01$), Island A 1966 (males $P < 0.01$; females $P < 0.01$) and Island B 1966 (males $P < 0.01$; females $P < 0.01$). None of the populations showed a reduction in mean body weight. (The conclusion drawn from these data is that the food supply available to the animals while they were on the islands was sufficient to maintain them.)

b) Adrenal weights and sudanophilia.

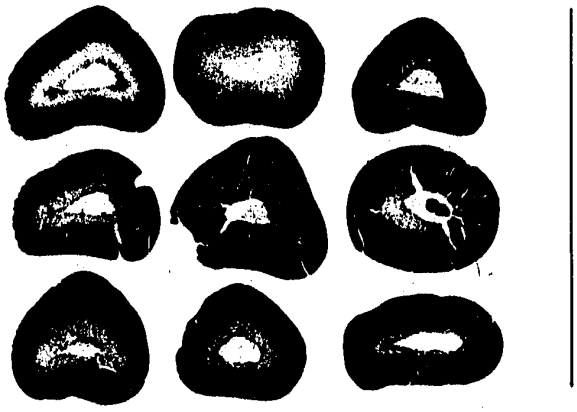
The adrenal weight data for the two years are given in Fig. 10 and Table 4A. The data obtained in 1965 were so few that it was necessary to pool those from males and females. In that year the adrenals of the Island A population were significantly heavier ($P < 0.01$) than Island B, laboratory or wild samples. In 1966 it was not possible to demonstrate significance between the populations.

The adrenal sections stained with Sudan Black B

PLATE I Adrenal sudanophilia in T. striatus



ISLAND A

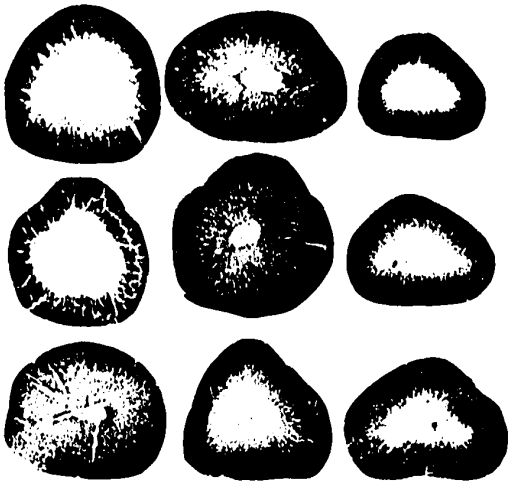


ISLAND B

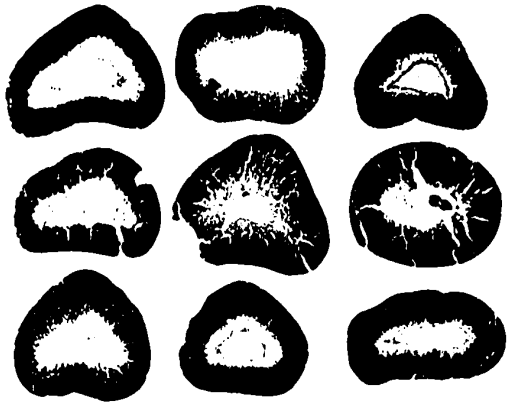
ISLAND C

(X 10)

PLATE I Adrenal sudanophilia in T. striatus



ISLAND A



ISLAND B

ISLAND C

(X 10)

were studied and differences in the intensity and distribution of sudanophilia were noticed. Sample sections from the island populations were photographed (Plate 1). There can be seen a density-related decrease in thickness and intensity of the sudanophilic region of the cortex.

c) Levels of circulating eosinophils.

Levels of circulating eosinophils, expressed as a percentage of total white blood cell count (Figure 11 and Table 5A), revealed differences related to population density. The density-related trend seen in the eosinophils counts of males animals was not significant ($P > 0.05$). For the females, those of Islands A and B were not significantly different ($P > 0.05$). However, Island C females were different from both those on Island A ($P < 0.01$) and on Island B ($P < 0.05$).

Trypanosomes were seen in the blood smears from about a third of the total number of animals. Omission of the data obtained from these slides did not affect the significance of the results in any way.

d) Plasma cortisol.

Cortisol was the principal plasma corticosteroid, as judged by the speed of development of fluorescence in the fluorometric method of Pelletier, Labrie and Fortier (87). The concentrations of cortisol in the plasma are given in Figure 12 and Table 6A.

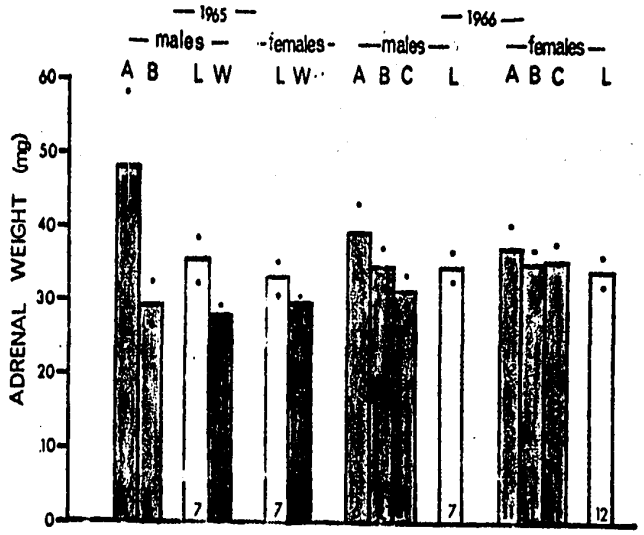


Fig. 10. Adrenal weights of T. striatus. (Mean ± S.E. shown for each group)

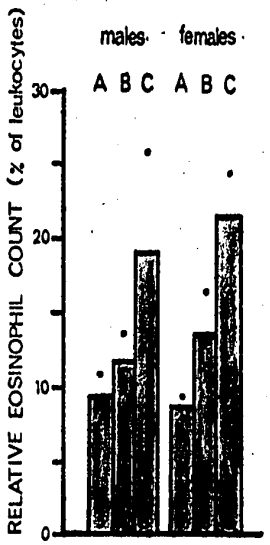


Fig. 11. Eosinophil levels of T. striatus, 1966. (Mean ± 1 S.E. shown for each group.)

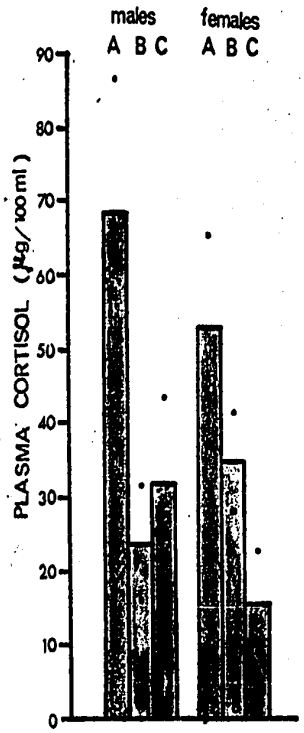


Fig. 12. Plasma cortisol levels of T. striatus (1966) (Mean ± 1 S.E. shown for each group.)

Analysis of variance revealed no population-related differences, when the data was divided by sex. Analysis of variance showed that significant ($P<0.01$) differences existed between pooled samples. The least significant difference test showed that the Island A value is greater ($P<0.05$) than that of either B or C.

e) Thymus weights.

Thymus weights(Figure 13 and Table 7A)showed no significant variations between island populations. Significant differences were noted between the laboratory sample of 1965, Island A ($P<0.01$), Island B ($P<0.01$) and the wild sample ($P<0.05$).

f) Spleen weights.

Spleen weights of male animals(Figure 14 and Table 8A) were not significantly different between island populations. Female animals from the densest population in 1966 had heavier spleens ($P<0.01$) than those of either of the other two populations. Islands B and C were not significantly different. Differences were noted between the island populations and the others in both years.

Removal of data, relating to animals which were infected with trypanosomes or which were infested with larva(e) of Cuterebra sp. in 1966, and reanalyzing the remaining data, increased only slightly the number of significant differences between island populations (Island A males $>$ Island C males: $P<0.05$).

ADDENDUM

g) Testes

Histological examination of section 5 of testes revealed a lack of spermatogenesis and a picture identical to the photomicrographs of Neff and Anthony (1964) showing the chipmunk testis when quiescent.

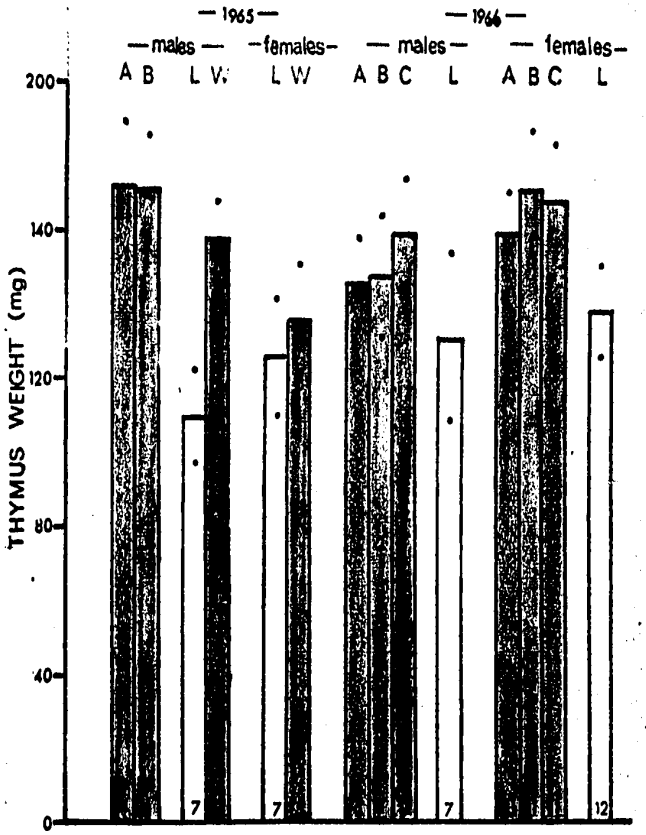


Fig. 13. Thymus weights of T. striatus. (Mean ± 1 S.E. shown for each group.)

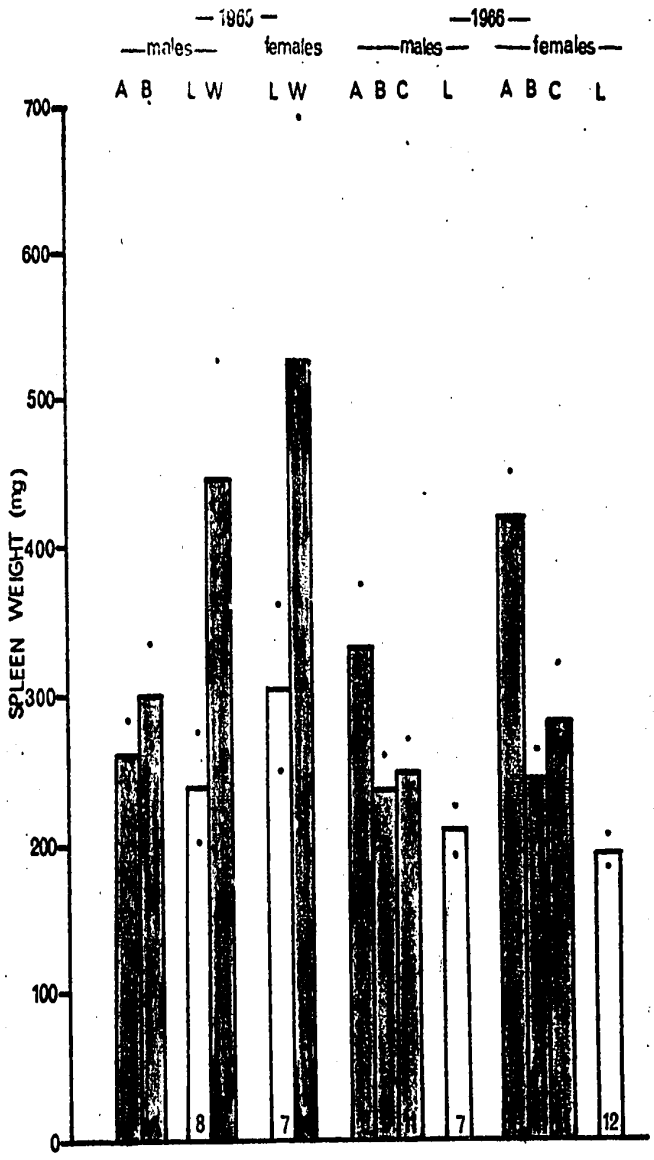


Fig. 14. Spleen weights of T. striatus. (Mean ± 1 S.E. shown for each group.)

E) Results of the P. leucopus experiments in 1966.

a) Body weights.

The mean starting weight of all animals in each population is given in Figure 15 and Table 9A. The records of the body weights of 5 of the animals released in the second experiment were accidentally lost. Student's 't' test revealed no significant differences between the populations in each experiment ($P > 0.05$). Similarly, no differences in mean initial weight of the survivors, shown in Figure 15 and Table 10A, could be found.

The body weights of the animals just prior to death (Figure 15 and Table 11A) showed no significant variation within experiments ($P > 0.05$).

b) Adrenal weights and sudanophilia.

Adrenal weights of animals in the different groups (Figure 16 and Table 12A) showed no significant differences ($P > 0.05$) within each experiment.

Examination of the Sudan Black B stained sections showed no differences between groups.

c) Levels of circulating eosinophils.

Relative eosinophil levels expressed as a percentage of total white blood cells seen in Figure 17 and Table 13A, showed no significant variation correlated with population density ($P > 0.05$).

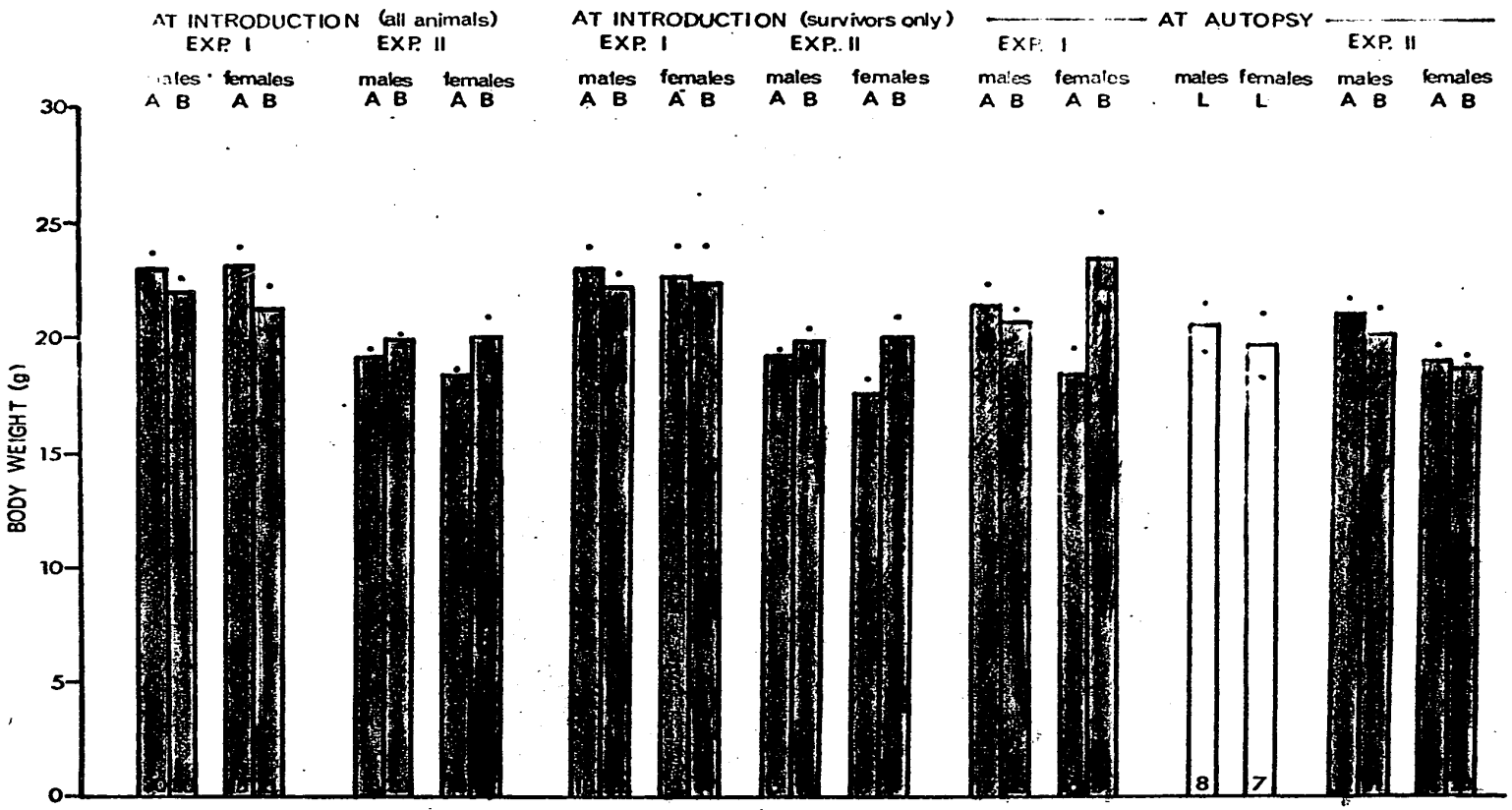


Fig. 15. Body weights of P. leucopus. (Mean ± 1 S.E. shown for each group.)

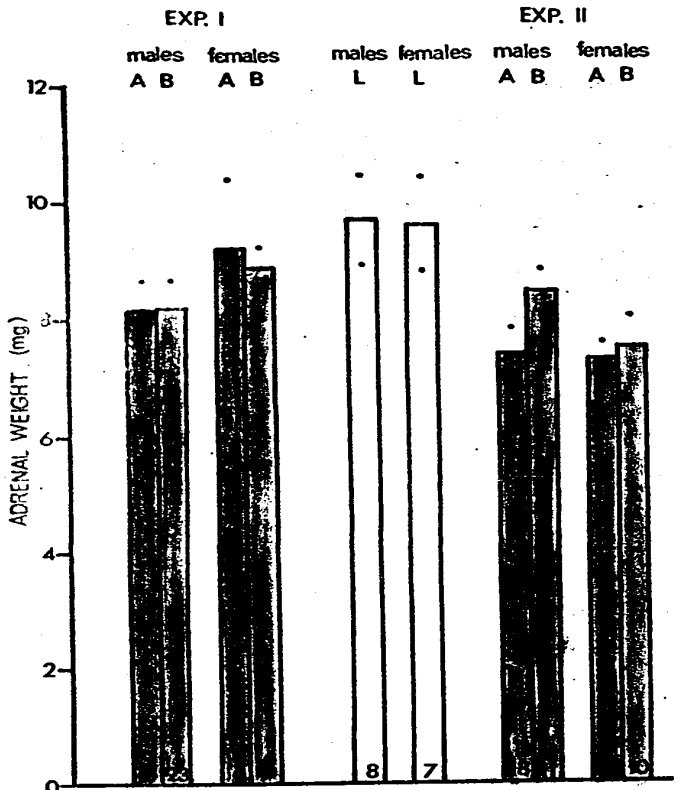


Fig. 16. Adrenal weights of P. leucopus. (Mean ± 1 S.E. shown for each group.)

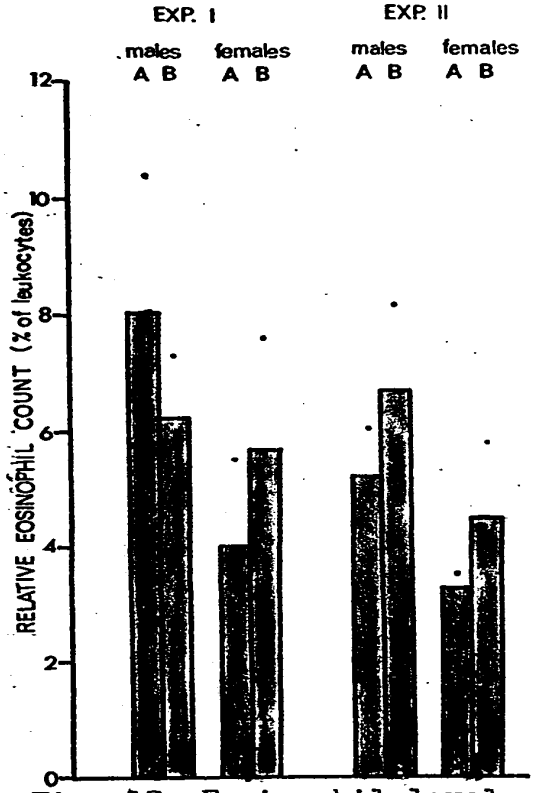


Fig. 17. Eosinophil levels of P. leucopus. (Mean ± 1 S.E. shown for each group.)

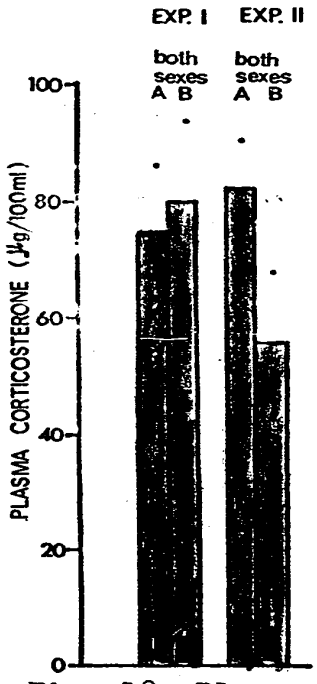


Fig. 18. Plasma corticosterone levels of P. leucopus. (Mean ± 1 S.E. shown for each group.)

d) Plasma corticosterone.

Corticosterone, which was found to be the only corticosteroid present in the plasma, showed no significant variation in concentration with population density ($P \geq 0.05$). Due to the small number of samples data obtained from both sexes have been pooled (Figure 18 and Table 14A).

e) Thymus weights.

Thymus weights, given in Figure 19 and Table 15A, showed the following differences. In Experiment I, the thymus glands of the females on Island A were significantly heavier ($P < 0.05$) than those of both Island B and the laboratory sample. In Experiment 2, the males of Island A had heavier thymuses than those of Island B ($P < 0.05$).

f) Spleen weights.

The spleen weights, shown in Figure 20 and Table 16A, were not significantly different between island populations. The spleens of females of Island B in Experiment I were heavier than those of the laboratory sample ($P < 0.05$).

g) Testes

Histological examination of sections of testes revealed active spermatogenesis.

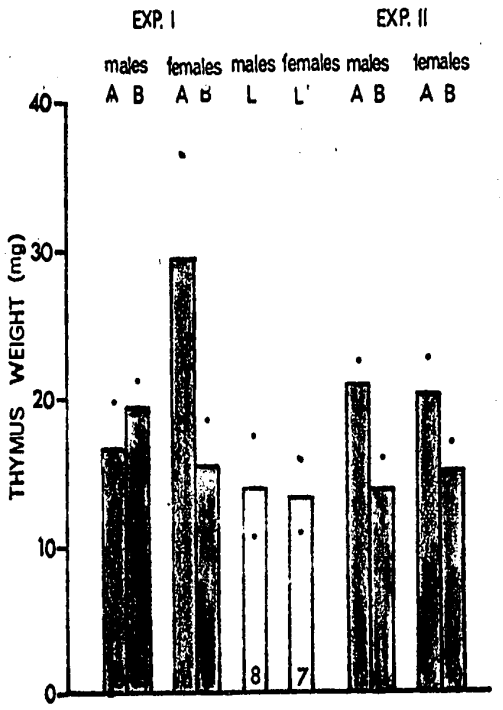


Fig. 19. Thymus weights of P. leucopus. (Mean ∓ 1 S.E. shown for each group.)

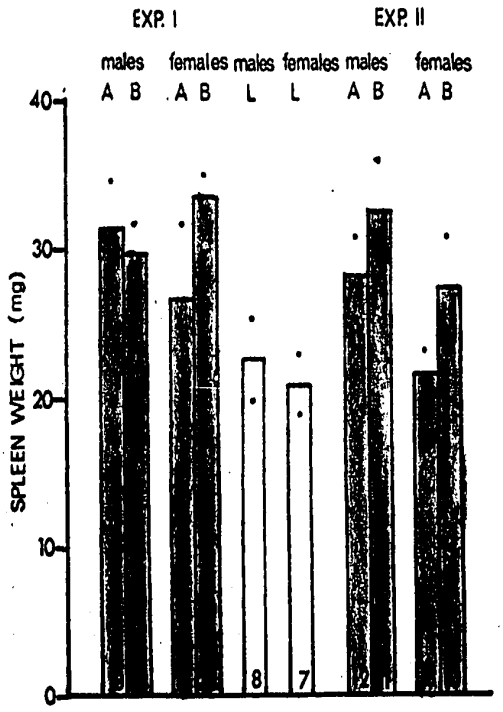


Fig. 20. Spleen weights of P. leucopus. (Mean ± 1 S.E. shown for each group.)

CHAPTER IV

DISCUSSION AND CONCLUSIONS

The null hypothesis tested in each of my experiments was that the physiological states of the animals would not be related to the densities of the populations to which the animals belonged.

In the case of T. striatus, the null hypothesis was disproved by our findings in both years. In 1965, the increase in adrenal weight, which was related to population density, was taken as an indication that there had been difference in the degree of activity of the adrenal cortices of the animals sometime during the course of the experiment. In 1966, the observed differences in adrenal weights were non-significant. In spite of this lack of significance, the progressive increase in adrenal weight seen in the males, the loss of sudanophilic material from the adrenal cortex, and the increases in eosinopoenia and plasma cortisol indicated that increased adrenocortical function accompanied increased population density in the species. The spleen and thymus weights were variable. The slight splenomegaly seen in animals from the densest population was interpreted as confirmation of higher adrenocortical activity in this group.

In the case of P. leucopus no consistent differences, related to population density, were found in any of the parameters which were considered. The null hypothesis therefore remains intact. The differences which were seen in the thymus and spleen weights were discounted as indicating different degrees of adrenocortical function because of the absence of corroborating evidence.

My conclusions are: a) In the case of T. striatus density-dependent behavioural-physiological factors may operate under natural conditions to regulate population density; b) in P. leucopus population density does not affect the physiological parameters which were measured. This being the case, it is difficult to see how Christian's mechanism can effect population regulation in this species. This conclusion is in agreement with the findings of Bendell (6) who observed no correlation between population density and adrenal weight in experimental island populations of P. leucopus.

This difference in response of the two species is difficult to explain. In both species there was overlap of home ranges of individual animals. The home ranges reported for T. striatus vary from 0.25 acres (0.10 Hectares) to 3.10 acres (1.26 Hectares) (Table 8). P. leucopus has a home range of about a third of an acre

TABLE 8

Home ranges of T. striatus and P. leucopus

	Author		Mean Home Range (Acres)	Mean Home Range (Hectares)
T. striatus	Burt	(17)	1.04 - 2.04	0.42 - 0.83
	Blair	(7)	2.31 (Males) 2.15 (Females)	0.94 0.87
	Smith	(104)	3.10 (Males) 1.66 (Females)	1.26 0.67
	Manville	(75)	0.25	0.10
	Yerger	(128)	0.26 (Males) 0.37 (Females)	0.11 0.15
P. leucopus				
	Burt	(17)	0.27	0.11
	Howell	(65)	0.29	0.12

ADDENDUM

My histological observations, reported on pp. 50 and 56 confirm that the chipmunks were sexually quiescent and the mice were sexually active during the experiments.

experiments. This could have resulted in more numerous social interactions in the chipmunks than in the mice. Even so, the mice did not respond at all to the overlapping of home ranges.

Another possibility is that the reproductive activity of the animals influenced their aggressiveness and thus the degree of stress within the populations. The breeding season of P. leucopus starts in March or April with the last young being born in September or October (6, 17, 44). The picture is not so clear for T. striatus. The breeding season is reported to start in March or April in Ohio (43). Maximum spermatogenic activity is observed in Pennsylvania in February and March with subsequent testicular regression in April and May, which is complete by June (81). Forbes (54) reports that no reproductive activity is observable after July in Minnesota. Panuska and Wade (86) report that very few pregnant females can be found from late June or early July onwards in Wisconsin. There is however the possibility of a summer breeding season for the species, at least over part of its range. Condrin (43) reports such mating occurring in late June in Ohio.

My experiments were performed at a time when reproductive activity, and probably also social interaction, was at a

(Table 8). The degree of overlap of home ranges may have been greater in T. striatus than in P. leucopus in the experiments. This could have resulted in more numerous social interactions in the chipmunks than in the mice. Even so, the mice did not respond at all to the overlapping of home ranges.

Another possibility is that the reproductive activity of the animals influenced their aggressiveness and thus the degree of stress within the populations. The breeding season of P. leucopus starts in March or April with the last young being born in September or October (6, 17, 44). The picture is not so clear for T. striatus. The breeding season is reported to start in March or April in Ohio (43). Maximum spermatogenic activity is observed in Pennsylvania in February and March with subsequent testicular regression in April and May, which is complete by June (81). Forbes (54) reports that no reproductive activity is observable after July in Minnesota. Panuska and Wade (86) report that very few pregnant females can be found from late June or early July onwards in Wisconsin. There is however the possibility of a summer breeding season for the species, at least over part of its range. Condrin (43) reports such mating occurring in late June in Ohio.

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ADDENDUM

My histological observations, reported on pp. 50 and 56 confirm that the chipmunks were sexually quiescent and the mice were sexually active during the experiments.

high level in P. leucopus. On the other hand, the chipmunks were sexually quiescent during the experiment. It is reasonable to suppose that social interactions within the chipmunk population were milder during this period than in the breeding season. If reproductive activity increases social interaction then it would have influenced adrenal responses to population density. In which case results opposite to those actually obtained could have been expected.

Another alternative is that P. leucopus is intrinsically different in its manner of population regulation than T. striatus. Various authors have reported on other species which seem to share this lack of adrenal responsiveness to population density. They include: a) Negus et al (82), who could find no correlation between adrenocortical activity and population density when working with the rice rat, Oryzomys palustris. Their conclusions have been questioned by Christian and Davies (35) on the grounds that the samples were too small, their variability was too great and that the authors had indiscriminately pooled samples of different sexes and ages. Negus and Gould in turn have criticized Christian's data on deer (83). b) Houlihan (64), who found no relationship between population density and adrenocortical activity when observing voles in enclosures. In his experiments he observed that food limited population size under certain circumstances. He did note that in a population crash which

occurred in his experiment the animals showed signs of adrenal insufficiency.

c) Clough (42), who found no correlation between gland weight and population density also when studying voles. He concluded that changes in viability, due to dense population conditions, play a minor role in the fluctuations of rodent numbers.

d) Terman (115) who observed that animals from a dense, freely growing laboratory population of Peromyscus maniculatus bairdii had the same mean adrenal weight as isolated controls. The reproductive organs from the group animals were smaller than the controls.

e) Lidicker (70), who found no correlation between body, adrenal, thymus and spleen weights on one hand and population density on the other when investigating a declining feral house mouse population. He concluded that the population reduction which he observed was due to a number of factors including reduced viability and failure of reproduction.

f) Rudd and Mullen (96), who concluded that the response to population density in the pocket gopher, Thomomys umbrinus, is centered in the medulla and not in the adrenal cortex.

g) Myers (77) who demonstrated that in the presence of excess food, rabbit populations continued to grow in enclosures, to produce densities of up to 200 per acre. This density

is in excess of those found in nature. He could find no effect on growth rate of young rabbits or body weights of adults which were attributable to population density. Neither was there any increase in mortality which could be attributed to physiological upsets. Myers and Poole (79) had previously pointed out density-related changes in fecundity, mortality and social stress, but they indicated that these were insufficient to regulate the population.

For these species it is obviously necessary either to modify Christian's hypothesis, possibly by inserting a threshold density clause, or to look to some of the alternative mechanisms which might bring about population regulation which have been suggested. These include food supply, which has been shown to limit the levels of populations of P. leucopus (6), predation and disease.

Although [illegible] experiments were not designed to test them, the hypotheses of Wynne-Edwards (124, 127) and Chitty (18, 20) are worthy of mention at this point. Both involve behavioural factors and reproduction.

Wynne-Edwards (124) proposed that animals have evolved mechanisms of social behaviour (conventions) which serve to reduce reproduction under conditions of dense population thus stabilising population levels.

Chitty proposed (18, 20) that under conditions of high breeding density, contemporary and subsequent generations of animals undergo changes which lower their resistance to mortality factors such as severe weather or disease, thus bringing about population reduction or stabilization. Chitty (20) assumes a change in genetic constitution of populations before and after these have undergone reduction. He suggested selection of different genotypes at different points in the population cycle; these genotypes being related to the ability to reproduce and survive under conditions of mutual interference.

The complete elucidation of population regulation will have to wait until more species have been subjected to experimentation. The final conclusion will probably be a synthesis of the mechanisms mentioned above and others as yet unknown.

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APPENDIX

CONTENTS

Table 1A Body weights (gm) of T. striatus populations at time of introduction: All animals (1965, 1966).

	1965		1966	
	Males	Females	Males	Females
Populations				
Island A	97.68 ±2.55* (25)**	96.00±3.01 (20)	94.71±3.22 (17)	81.52±2.69 (23)
Island B	97.40 ±3.43 (15)	97.25±0.31 (10)	93.90±2.73 (20)	95.15±2.16 (20)
Island C	-	-	97.05±3.02 (20)	94.25±2.26 (20)

* = Mean ± 1 Standard Error

** = Sample Size.

Table 2A Body weights (gm) of T. striatus populations at time of introduction: Survivors only (1965, 1966).

	1965		1966	
	Males	Females	Males	Females
Populations				
Island A	103.63±3.64* (4)**	***	89.67±2.64 (6)	79.44±3.57 (9)
Island B	91.00±4.25 (6)	***	90.44±4.22 (9)	92.00±2.83 (11)
Island C	-	-	91.00±5.98 (9)	91.55±3.99 (11)

* = Mean ± 1 Standard Error

** = Sample Size

*** - Sample too small

Table 3A Body weights (gm) of T. striatus populations at time of autopsy: all animals (1956, 1966).

	1965		1966	
	Males	Females	Males	Females
Populations				
Island A	120.50±11.44* (4)**	***	111.17±2.77 (6)	101.55±2.00 (11)
Island B	120.83±6.51 (6)	***	105.56±3.75 (9)	107.36±3.40 (11)
Island C	-	-	101.09±3.78 (11)	98.00±3.33 (11)
Laboratory	89.86±5.31 (7)	96.00±5.16 (7)	96.43±3.12 (7)	98.00±2.69 (12)
Wild	91.38±2.83	92.00±2.68	-	-

* = Mean ± 1 Standard Error

** = Sample Size

*** = Sample too small

Table 4A Adrenal weights (mg), T. striatus (1965, 1966).

	1965	1966	
	Males and Females	Males	Females
Populations			
Island A	53.20±10.89* (6)**	39.12±3.16 (6)	37.50±3.08 (11)
Island B	30.66±2.44 (8)	34.72±2.48 (9)	35.23±1.98 (11)
Island C	-	31.64±1.84 (11)	35.98±1.93 (11)
Laboratory	31.34±1.89 (14)	34.33±2.14 (7)	34.23±1.93 (12)
Wild	28.49±0.89 (13)	-	-

* = Mean ± 1 Standard Error

** = Sample Size

Table 5A Levels of circulating eosinophils (% Total white Blood cells): T. striatus (1966).

	1966	
	Males	Females
Populations		
Island A	9.40±1.34* (5)**	8.57±0.82 (7)
Island B	11.93±1.51 (6)	13.58±2.83 (8)
Island C	19.00±6.83 (4)	21.48±2.99 (5)

* = Mean ± 1 Standard Error

** = Sample Size

Table 6A Plasma Cortisol (µg/100 ml) T. striatus (1966).

	1966		
	Males	Females	Both sexes
Populations			
Island A	68.64±17.75* (5)**	52.50±12.51 (7)	59.23±10.16 (12)
Island B	23.78± 7.78 (4)	34.50± 6.78 (8)	32.22± 4.66 (12)
Island C	32.00±11.40 (5)	15.68± 6.50 (5)	23.84± 6.50 (10)

* = Mean ± 1 Standard Error

** = Sample Size

Table 7A Thymus weights (mg), T. striatus (1965, 1966)

	1965		1966	
	Males	Females	Males	Females
Populations				
Island A	172.23±16.79* (4)**	***	146.63±12.05 (6)	159.78±11.56 (11)
Island B	171.33±13.54 (6)	***	148.11±16.47 (9)	170.87±16.39 (11)
Island C	-	-	159.47±14.67 (11)	167.36±16.07 (11)
Laboratory	109.86±12.23 (7)	126.16±14.90 (7)	131.09±16.29 (7)	138.90±11.02 (12)
Wild	158.52± 9.08 (8)	136.76±14.80 (5)	-	-

* = Mean ± 1 Standard Error

** = Sample Size

*** = Sample too small

Table 8A Spleen weight (mg), T. striatus (1965, 1966)

	1965		1966	
	Males	Females	Males	Females
Populations				
Island A	260.55±24.13* (4)**	***	332.43±42.66 (6)	418.69±29.45 (11)
Island B	300.53±34.98 (6)	***	235.09±23.21 (9)	241.62±19.12 (11)
Island C	-	-	246.51±24.60 (11)	279.91±39.63 (11)
Laboratory	237.77±36.44 (7)	303.33±55.38 (7)	208.00±15.58 (7)	190.07±12.27 (11)
Wild	446.15±69.46 (8)	525.24±163.84 (5)	-	-

* = Mean ± 1 Standard Error

** = Sample Size

*** = Sample too small

Table 9A Body weights (gm) of P. leucopus populations at time of introduction: All animals (1966).

	Experiment I		Experiment II	
	Males	Females	Males	Females
Populations				
Island A	20.00±0.70* (30)**	23.20±0.60 (15)	19.11±0.38 (18)	18.40±0.30 (10)
Island B	21.90±0.64 (30)	21.10±1.10 (15)	19.80±0.40 (20)	20.00±0.85 (7)

* = Mean ± 1 Standard Error

** = Sample Size

Table 10A Body weights (gm) of P. leucopus populations at time of introduction: Survivors only (1966).

	Experiment I		Experiment II	
	Males	Females	Males	Females
Populations				
Island A	23.00±0.98* (18)**	22.58±1.39 (6)	19.08±0.48 (12)	17.60±0.75 (5)
Island B	22.07±0.75 (22)	22.29±1.78 (7)	19.82±0.62 (11)	20.00±0.85 (7)

* = Mean ± 1 Standard Error

** = Sample Size

Table 11A Body weights (gm) of P. leucopus populations at time of autopsy (1966).

	Experiment I		Experiment II	
	Males	Females	Males	Females
Populations				
Island A	21.41±0.97* (17)**	18.50±1.09 (6)	21.00±0.69 (12)	19.00±0.68 (6)
Island B	20.52±0.75 (23)	23.43±2.03 (7)	20.09±1.27 (11)	18.60±0.54 (10)
Laboratory	20.50±1.10 (8)	19.71±1.39 (7)	-	-

* = Mean ± 1 Standard Error

** = Sample Size

Table 12A Adrenal weights (mg) P. leucopus (1966).

	Experiment I		Experiment II	
	Males	Females	Males	Females
Populations				
Island A	8.11±0.50* (15)**	9.20±1.17 (6)	7.38±0.42 (13)	7.33±0.26 (6)
Island B	8.15±0.49 (23)	8.84±0.41 (7)	8.46±0.37 (11)	7.49±0.56 (10)
Laboratory	9.69±0.77 (8)	9.60±0.84 (7)	-	-

* = Mean ± 1 Standard Error

** = Sample Size

Table 13A Levels of circulating eosinophils (% Total White Blood Cells), P. leucopus (1966).

	Experiment I		Experiment II	
	Males	Females	Males	Females
Populations				
Island A	8.06±2.33* (8)**	4.00±1.50	5.15±0.88 (10)	3.25±0.25 (2)
Island B	6.23±1.04 (13)	5.67±1.92 (3)	6.70±1.46 (5)	4.44±1.34 (4)

* = Mean ± 1 Standard Error

** = Sample Size

Table 14A Plasma corticosterone (µg/100 ml), *P. leucopus* (1966).

Populations	Experiment I	Experiment II
	Both sexes	Both sexes
Island A	67.29±18.63* (4)**	81.72±8.17 (12)
Island B	79.93±13.14 (10)	55.23±12.72 (8)

* = Mean ± 1 Standard Error

** = Sample Size

Table 15A Thymus weights (mg), P. leucopus (1966).

	Experiment I		Experiment II	
	Males	Females	Males	Females
Populations				
Island A	16.77±3.14* (18)**	29.43±6.88 (6)	20.94±1.70 (12)	20.02±2.67 (6)
Island B	19.34±1.99 (23)	15.33±2.80 (7)	13.89±1.90 (11)	15.09±1.86 (10)
Laboratory	13.92±3.16 (8)	13.06±2.50 (7)	-	-

* = Mean ± 1 Standard Error

** = Sample Size.

Table 16A Spleen weights (mg), P. leucopus (1966).

	Experiment I		Experiment II	
	Males	Females	Males	Females
Populations				
Island A	31.53±2.99* (19)**	26.53±5.00 (6)	27.90±2.78 (12)	21.37±1.56 (6)
Island B	29.89±1.93 (23)	33.41±1.45 (7)	32.37±3.20 (11)	27.38±3.23 (10)
Laboratory	22.42±2.62 (8)	20.96±1.98 (7)	-	-

* = Mean ±1 Standard Error

** = Sample Size