giotensinases." We, therefore, suggest this plasma and red-cell enzyme from normal human beings be called angiotensinase A.

With respect to substrate specificity, angiotensinase A resembles aminopeptidase A, an enzyme found in a particulate fraction from rat kidney that splits α-aspartyl- but not β-aspartyl-β-naphthylamide, and which is also activated by calcium ions (*13*). Identity between these two peptidases has not been established.

The fact that β-aspartyl-angiotensin is not destroyed by angiotensinase A may lead to erroneous results since the α-aspartyl compound is easily converted to the β-aspartyl linked compound. For example, when angiotensin II which had been dissolved in 0.1*N* HCl for several months was used undiluted plasma did not destroy angiotensin II (*1*). Subsequently, we found that this treatment of angiotensin yielded a peptide with electrophoretic mobility the same as that of β-aspartyl-angiotensin. We would again recommend that paper electrophoresis at *p*H 2.1 be used on all angiotensin II samples (*9*) since this can separate the α- and β-linked aspartyl peptides. The β-linked peptide obtained in this way retains full pressor activity, but is not split by plasma or red-cell enzyme. The possibility of a mixture may account for some of the disparity in results reported.

When measuring angiotensinase concentrations, several angiotensin analogs should be used to differentiate between angiotensinase A and other enzymes which may destroy angiotensin. For example, the plasma of a hypertensive patient in uremia who had been subjected to biweekly hemodialysis showed increased destruction of angiotensin activity. But since it destroyed deaminoangiotensin (Fig. 3), an angiotensin-destroying enzyme or enzymes other than, or in addition to, angiotensinase A were present in this patient's plasma.

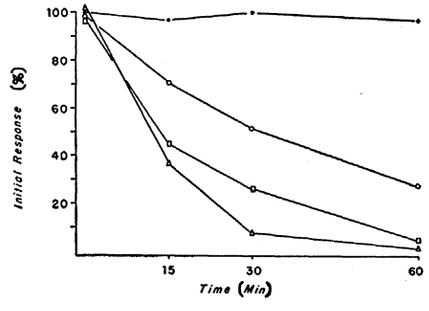

Fig. 3. Normal and abnormal plasma angiotensinase. Open circles, normal plasma plus angiotensin II; closed circles, normal plasma plus deaminoangiotensin II; triangles, abnormal plasma plus angiotensin II; squares, abnormal plasma plus deaminoangiotensin II.

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Lemming Cycle at Baker Lake, Canada, during 1959–62

Abstract. *Brown and varying lemmings of the central Canadian arctic showed changes in reproduction, mortality, and the properties of the individuals over a 4-year period of fluctuating population. These changes were not due to starvation or malnutrition, nor were there any obvious symptoms of stress. They may have been associated with changes in the behavior of the animals.*

Periodic fluctuations in small mammal populations have not yet been explained. The lemming cycle of the tundra is a classic example of the 3- to 4-year cycles found in many rodent populations (subfamily Microtinae) of temperate and arctic regions. Elton (*1*) has described the early work on periodic fluctuations.

Since 1949 the brown-lemming (*Lemmus trimucronatus*) cycles of northern Alaska have been studied in detail (*2*). Populations of the Norwegian lemming (*L. lemmus*) and the Siberian lemmings (*L. sibiricus* and *Dicrostonyx torquatus*) have been studied only sporadically and not in detail.

The purpose of this paper is to summarize a 4-year study on brown- and varying-lemming (*D. groenlandicus*) populations of the central Canadian arctic, and to relate these findings to three currently held hypotheses. The main area studied was at Baker Lake, 425 miles north of Churchill, Manitoba. Data were obtained from about 4000 animals (*3*).

Beginning in the summer of 1959 and continuing throughout the winter of 1959–60, both species increased in number. The crude estimates of this winter increase were 25- to 50-fold in *Lemmus* and 5- to 10-fold in *Dicrostonyx*. Little further increase occurred during the summer of 1960, when the population was highest. The population decreased over the winter of 1960–61, so that only 5 to 10 percent of the *Lemmus* population and 20 to 30 percent of the *Dicrostonyx* survived from August 1960 to June 1961. In the main area this decline continued and accelerated during the summer of 1961. The population changed little during the winter of 1961–62 but began to increase again in the summer of 1962. This cycle was synchronous in both species over a very wide zone of the central Canadian arctic.

But not all areas showed this sharp decline in 1961. At Aberdeen Lake, 115 miles west of Baker Lake, the decline was very gradual, as it was at several islands in Baker Lake within a few miles of the main study area.

Two components of reproduction varied with the cycle—length of breeding season and weight at sexual maturity. Winter breeding occurred in the period of increase but not in the period of decline; summer breeding ended in early August in the peak year and in the decline, rather than continuing into September. There was rapid sexual maturation in the period of increase. In particular, young *Lemmus* males born in the summer matured in the summers of increase at 25 to 35 g but did not mature during the peak summer or decline even though they reached weights of 40 to 50 g. Females matured in the summers of increase and

decline at 20 to 25 g, but in the peak summer most did not mature and those which did weighed 25 to 35 g. There was no change during the cycle in litter size or midsummer pregnancy rates.

Juvenile mortality was very high in the summer of the decline in both species. The young of the first litter of that summer nearly all died, but more young survived in the latter part of the summer. Adults survived less well in the summer of decline, but this change was relatively slight compared with the changes in juvenile mortality.

Mean body weights of the adults increased 20 to 30 percent in the peak summer (Table 1). The change in body weight represents change in growth rate of the lemmings and is not the result of fat deposition or age differences. High body weights occurred in the gradual decline at Aberdeen Lake and were accompanied by improved recruitment of young. In the sharp decline adult body weights were low and few young survived. Increased body weight in peak years has been described for several cyclic microtines (*4*) and may be considered a defining characteristic of these cycles.

The role of the extrinsic factors of food, predators, disease, and parasites was investigated. Lemmings reduced the forage crop outside wire enclosures by about 15 percent in the peak and decline summers (Table 2). After the crucial winter of 1960–61 30 percent or less of the vegetation in wet habitats was cut, and forage utilization in dry habitats was negligible. Thus there was no evidence of quantitative food shortage, nor was there any suggestion of deficiencies in food quality. Lemmings in the spring of the decline were as fat as usual.

Table 1. Relative density changes and mean adult body weights in *Lemmus* and *Dicrostonyx*, 1959-62. All data are from 16 to 31 July of given year.

Year	Relative density*	Mean adult body weight (g)	
		Main study area	Aberdeen Lake
		Lemmus	
1959	<1	56.6 ± 3.0	
1960	43	81.8 ± 4.5	84.7 ± 3.6
1961	3	62.8 ± 3.5	79.8 ± 3.0
1962	1	63.4 ± 1.5	67.3 ± 1.4
		Dicrostonyx	
1959	~2	53.0 ± 0.5	
1960	29	75.0 ± 0.8	76.9 ± 2.9
1961	6	63.0 ± 2.7	80.9 ± 3.3
1962	7	62.3 ± 1.6	60.0 ± 1.8

* Based mainly on live trapping in the main study area.

Avian predators were uncommon. Weasels (*Mustela erminea*) were the only common mammalian predators but they could not have accounted for observed mortality changes because the numbers of weasels on the study area did not increase until late in the summer of 1961 when the mortality rate of young lemmings decreased. Disease and parasites seemed to play no significant role in the cycle.

The role of the intrinsic factors of behavior and physiology was also considered. In an attempt to find a physiological index which was correlated with the population changes, adrenals, spleens, and testes from about 3000 lemmings were weighed. For both species neither adrenal nor spleen weights showed any relationship to the cycle, although they showed seasonal change. Testes weights showed some relationship to the cycle.

Intraspecific strife, as measured by wounds on skins, showed strong seasonal changes which were not a simple function of density. The amount of wounding in adults during July and August did not change significantly in the period from 1959 to 1962, which implies that severe interactions between animals may occur even at very low densities. Sexually mature young males from summer litters suffered more wounding during the summer of increase than did immature young males born at the same time.

Chitty (*5*) has discussed reasons for viewing cycles as a single class of events having some common explanation. We are dealing with a system of changes in reproduction, mortality, and the properties of the animals which is manifested by cyclic changes in numbers.

With this background of information I have attempted to evaluate three current hypotheses for explaining microtine cycles.

The food-supply hypothesis of Pitelka (*6*) and Lack (*7*) explains the cycle by qualitative and quantitative changes in the vegetation which result in changes in reproduction and survival of the animals because of nutrient deficiencies. But there was no extensive forage depletion or any evidence of starvation during this cycle. Thus any food deficiency, if there is any, must be qualitative. Yet there was no macroscopic evidence of deficiency diseases in the young, and it seems highly unlikely that the reproductive and mortality changes are the result of subtle qualitative changes in forage. For these reasons I rejected the food-supply hypothesis as an adequate explanation of this lemming cycle.

Table 2. Standing forage in grams per 0.5 m² dry weight at the end of summer. Figures are means for ten quadrats.

Year	Enclosed	Open
1959	45.8	38.8
1960	63.5	48.3*
1961	77.0	58.5*
1962	80.3	74.4

* Lemmings significantly depressed forage production, paired *t*-test, $.01 < P < .05$.

The stress hypothesis of Christian (*8*) explains the cycle by the "general adaptation syndrome" of Selye, associating declines with changes in adrenal-pituitary functions and shock disease. If this hypothesis is correct, there should be increased adrenal activity and decreased reproductive activity at high population densities. This increased adrenal activity should cause an increased death rate. I have found no consistent relationship between adrenal weights in the summer and the phases of the cycle. There is no evidence from field populations in favor of this hypothesis (*9*) and it cannot be considered an adequate explanation for the events described here.

The polymorphic-behavior hypothesis of Chitty (*10*) is the most recent attempt to explain these cycles. Chitty proposed that populations change in quality during changes in abundance and suggested that the mechanism was mutual antagonism associated with high breeding densities which brings about a change in the properties of the population through selection.

Evidence from my study supports the general concept that populations change in quality during changes in abundance. Peak populations showed these qualitative differences by high body weights and reproductive changes which carried over into the decline. There is no direct evidence from this study to test the genetic mechanism proposed by Chitty. None of my results are either good evidence against or good evidence for this mechanism.

The most important conclusion from this study has been the remarkable similarity between the cyclic changes in this Canadian study and those in rodents of such ecologically diverse places as Alaska, England, Finland, and Germany.

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Somatic Inheritance of Habituation of Responses to Light in Planarians

Abstract. *Planarians show long-lasting reversible decrease of responses to a photic stimulus repeated 25 times each day. This habituation was found to be significantly faster in regenerated offspring of previously habituated planarians, and was also transferred by cannibalization, thus resembling light-shock conditioning. Habituation provides a new situation in which to study the somatic inheritance of learning in planarians.*

Previous descriptions of planarian behavior include their reactions to light (*1*), vibration, and to changes in *p*H, temperature, oxygen tension, and so forth.

Studies on classical conditioning (*2*) and instrumental conditioning (*3*) in planarians with light (conditioned stimulus) and shock (unconditioned stimulus) have greatly extended knowledge of their behavior. Of particular current interest is the inheritance and cannibal-transfer of learning in planarians (*4*), and the possible chemical basis of these memories (*5*). The significance of these findings in view of the very simple nervous system planarians possess has been stressed (*2*, *4*, *5*).

The present study was undertaken to determine whether planarians show diminution or loss of responses to repeatedly applied sensory stimuli, that is, whether they show "habituation" (*6*). Light was chosen as the stimulus because there were earlier indications (*2*, *7*) to suggest that habituation to this sensory modality might occur in planarians, in addition to the environmental familiarization demonstrated by Best (*8*).

Sixty-six planarians of the species *Dugesia dorotocephala* used in the study were kept in individual dishes of tap water in the laboratory. They were allowed normal diurnal light fluctuations and were fed every 4 days. The apparatus and procedures used were similar to those described by McConnell (*9*) except for the inclusion of a heat filter between light-source and animals. Transfer and handling were by gentle suction, water jet, and occasionally fine brush. Daily tests were given only between 1:30 and 5:00 P.M. to minimize diurnal rhythm effects. After 5 minutes of familiarization to the test trough each day, 25 presentations of incandescent light were given daily to each planarian, with intertrial intervals of 30 to 60 seconds. Each planarian was returned to its home dish after 25 trials. The light intensity during each 3-second stimulus reached 400 foot-lamberts from the previous level of 40 ft-lam with ambient room lighting. Testing in all groups was single blind, except for the cannibal habituation group in which double blind testing and matched pairs were used because of the small numbers in this group.

Naive planarians ($N = 35$) averaged 26 percent responses to light in this situation on the first 2 days. This is comparable to previous figures for light control groups during conditioning studies (*2*).

Figure 1 shows the time course of the habituation to light for 35 normal controls (group A), in which the percentage response to light on successive days declined with several small plateaus. The criterion of full habituation for a single planarian was arbitrarily set at 2 successive days (50 trials) with zero responses. Included in this group are eight planarians that were habituated in the manner described but in the absence of a heat screen, and seven taildrop offspring of naive planarians, allowed to regenerate from 9 to 14 days and then habituated. Since there were no significant differences between results for either of these groups and the other naive planarians, all were

Table 1. Responses and habituation to light by *Dugesia dorotocephala*. Criterion of habituation is 50 consecutive trials with zero responses. *P* values refer to differences from the corresponding figure for group A, except in the case of group D, where *P* values refer to differences from their matched controls ($N = 6$), not shown in the table.

Responses in first 50 trials (Mean %, ± S.D.)	Time to criterion (Mean No. of trials, ± S.D.)
Group A (normal controls; N = *35)*	
26 ± 10	386 ± 132
Group B (taildrop offspring; N = *15)*	
15 ± 12 ($P < .01$)	218 ± 65 ($P < .001$)
Group C (regenerated cut tails; N = *10)*	
16 ± 8 ($P < .001$)	245 ± 80 ($P < .001$)
Group D (cannibal habituation; N = *6)*	
13 ± 9 ($P < .01$)	243 ± 87 ($P < .01$)

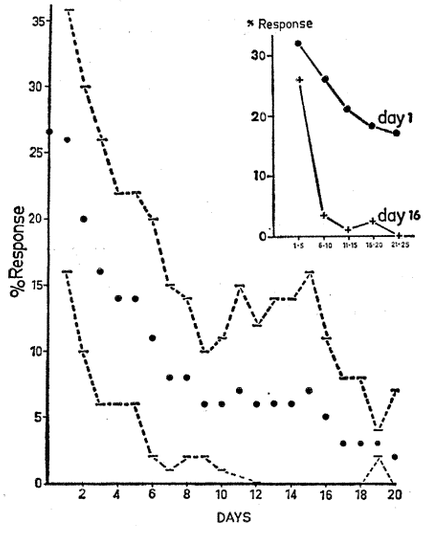

Fig. 1. Habituation of *Dugesia dorotocephala* to light. Each of 35 planarians received 25 presentations of light daily, each of 3 seconds duration. The ordinate shows the percentage of responses occurring each day, and training time in days is plotted along the abscissa. Each point is the mean of the percentages of responses of the 35 planarians on the day in question, and the horizontal bars above and below the points represent standard deviations of these means. The inset depicts receptor adaptation during 25 trials on day 1 (circles) and day 16 (crosses). Percentage response is shown on the ordinate for each of the five groups of five trials designated along the abscissa. Each circle is the mean of the percentages of responses of 35 planarians, and each cross is the mean of the percentages of responses of 16 planarians.