POLSKA AKADEMIA NAUK – KOMITET EKOLOGICZNY

EKOLOGIA POLSKA

SERIA A

Tom XI Warszawa 1963 Nr 3

Kazimierz PETRUSEWICZ

POPULATION GROWTH INDUCED BY DISTURBANCE IN THE ECOLOGICAL STRUCTURE OF THE POPULATION*

Confined populations of mice were used in experiments (repeated 264 times) in which either several virgin mice were added or several mice removed for the period of one week. In 65 percent of the experiments (versus 32% of controls) there was a numerical growth of the population. Numerical growth started in experiments at higher population levels than in controls. Population fecundity and survival of the young were after an experiment greater than before. The suggestion is made that the experimental shock breacks down the ecological structure of the population and thereby promotes numerical growth.

In an earlier paper (Petrusewicz 1957) transfer of an entire population to a different cage has been shown to cause numerical growth of the population irrespective of whether the new cage was larger, of the same size, or even smaller. The induced growth took place in 43 of 54 experiments, this result

* From Institute of Ecology, Polish Academy of Sciences, Warszawa.

being beyond the limits of error. Population fecundity and litter survival have also been shown to be after the experimental stimulus larger than before (difference statistical significant). It has been proposed that change of cages was a stimulus breaks down the population's ecological structure. The latter was construed as the complex whole of relations between population components. Upsetting of the population's structure caused the mice to behave more or less like a new-established population, and in these numerical growth is a rule.

To check this surmise, a number of experiments have been made in the Polish Academy of Sciences Institute of Ecology, where the stimulus did not involve a change of the habitat, but could have upset relations between individuals (a preliminary communication has already been published – Petrusewicz, 1960a).

1. METHODS AND MATERIAL

White laboratory mice were bred in cages of the following sizes:

37.5 × 15 × 15 cm. (type *A*)
80 × 80 × 15 cm. (type *Z*)
160 × 80 × 15 cm. (type *B*)

In cages of types *Z* and *B* were kept as "stock" populations 3 males and 8 females and in cages *A* 3 males and 5 females in each. The males used as "stock" were litter mates, or were at least kept together in a cage until three weeks old. The mice used as stock were allowed to breed selfranging without either addition or removal of any. Fully adequate feed, water, and shavings were always supplied in excess.

Daily records were kept of the number of births, survivals, and deaths. The mice were weighed fortnightly, and litters were marked by toe clipping when 3-week old. Over a period the food supplied and removed were weighed daily, to calculate per capita food in take. In some populations fights were counted before and after an experiment.

In all, the observations covered:
80 populations of type *Z*, which lived jointly 2225 months,
40 " of type *A*, " " " 379 months, and
7 " of type *B*, " " " 284 months.

Between July 1957 and June 1961, the following experiments were made 264 times:

Variant I. Several virgin females (4–10) were released into a cage inhabited by a population, and removed again after a week (gestation lasts in mice 20 days).

Variant II. Several (4–7) females and or males were removed from a population, kept separately according to sex and restored after a week.

In either variant the mice were added or removed in the proportion of about 1 : 4 or 1 : 5 of the actual population.

Concurrent experiments were regarded as a series, of which there were 14 (Tab. I). Cages of types *Z* were used in 8 series, covering a total of 181 experiments, and cages of type *A* and *B* in 5 (70 experiments) and 1 series (13 experiments) respectively.

For control, population dynamics before and after 476 random-chosen time points (dates) were determined as in experiments combined into 14 series for analysis, so that each series of experiments should have controls made at possibly the same time[1].

The time of experiment was chosen at random, with some qualifications. In neither experiments nor controls were recorded events to take place before the and of the first peak of a newly started population, for the first peak differs somewhat in character from others (Petrusewicz 1960). It usually exceeds subsequent ones and is obligatory, occurring invariably within ten weeks after the populations has been started[2]; another peak may follow the first not at all, or after a very long time.

In series *Z*1 and *B* (Tab. I), experiments were made in each population at a different time. The moment was chosen at random, but so that no experiment should succeed the previons by less than four months. Control was provided by control dates falling in the same span of time at a ratio of 3–4 in one year of the population's life and at intervals not shorter than 4 months after an experiment.

In series *Z*2–5 and 8, and *A*1, the experiment was made simultaneously in all populations at a random-chosen moment four to seven months after the previous experiment. Control data were chosen two to four months before and after the experiment (Tab. I).

In series *Z*6–7 and *A*2–5 an experiment was made at a single random-chosen moment in all even-numbered populations, and odd-numbered populations were used as controls. After two to four months the situation was reversed: even-numbered populations served as controls, and experiments were made in odd-numbered populations, and so on alternately (Tab. I).

2. THE GENERAL COURSE OF THE EXPERIMENTS

In general course, the development of the populations in which experiments were made was much like described earlier (Petrusewicz 1957, 1960).

[1] These random-chosen time points will be called "control dates" further below. The experimental procedure it self, i.e. the adding or removing of mice as in variants either I or II, will be colled hereafter "experimental interference". Jointly, control dates and experimental interference as in variants either I or II of the actual experiments will be referred to by the term "recorded event".

[2] Growth begins within 10 weeks. End of growth (peak and passing into equilibrium or decline) may take place even 10 months after the establishment of the population.

The series of experiments and controls, and their timing

Tab. I

Experiments			Date	Controls		
Basis for choice the moment	Series No	Number of replications		Number of replications	Series No	Basis for choice the moment
Time chosen arbitrarily	E.1	33	Varia	210	C.1	Time random-chosen
All populations	E.2	16	Sep. 6th, 1958 – Feb. 1st, 1959	19	C.2	All populations
All populations	E.3	21	April 1st, 1959 – July 15th, 1959	22	C.3	All populations
All populations	E.4	20	Oct. 1st, 1959 – Dec. 1st, 1959	20	C.4	All populations
All populations	E.5	20	Feb. 1st, 1960 – May 1st, 1960	21	C.5	Odd-numbered populations
Even-numbered populations	E.6	20	Sep. 1st, 1960	19	C.6	Even-numbered populations
Odd-numbered populations	E.7	19	Jan. 15th, 1961 – Dec. 1st, 1960	33	C.7	All populations
All populations	E.8	32	Jan. 15th, 1961 – March 1st, 1961	28	C.8	All populations
All populations	E.1 *A*	10	May 15th, 1959 – Sep. 1st, 1959	10	C.1 *A*	All populations
Odd-numbered populations	E.2 *A*	15	Feb. 1st, 1960	15	C.2 *A*	Even-numbered populations
Even-numbered populations	E.3 *A*	15	May 1st, 1960	15	C.3 *A*	Odd-numbered populations
Odd-numbered populations	E.4 *A*	15	Sep. 1st, 1960	15	C.4 *A*	Even-numbered populations
Even-numbered populations	E.5 *A*	15	Jan. 15th, 1961	14	C.5 *A*	Odd-numbered populations
As E, 1	E.*B*	13	varia	35	C.*B*	As C.1

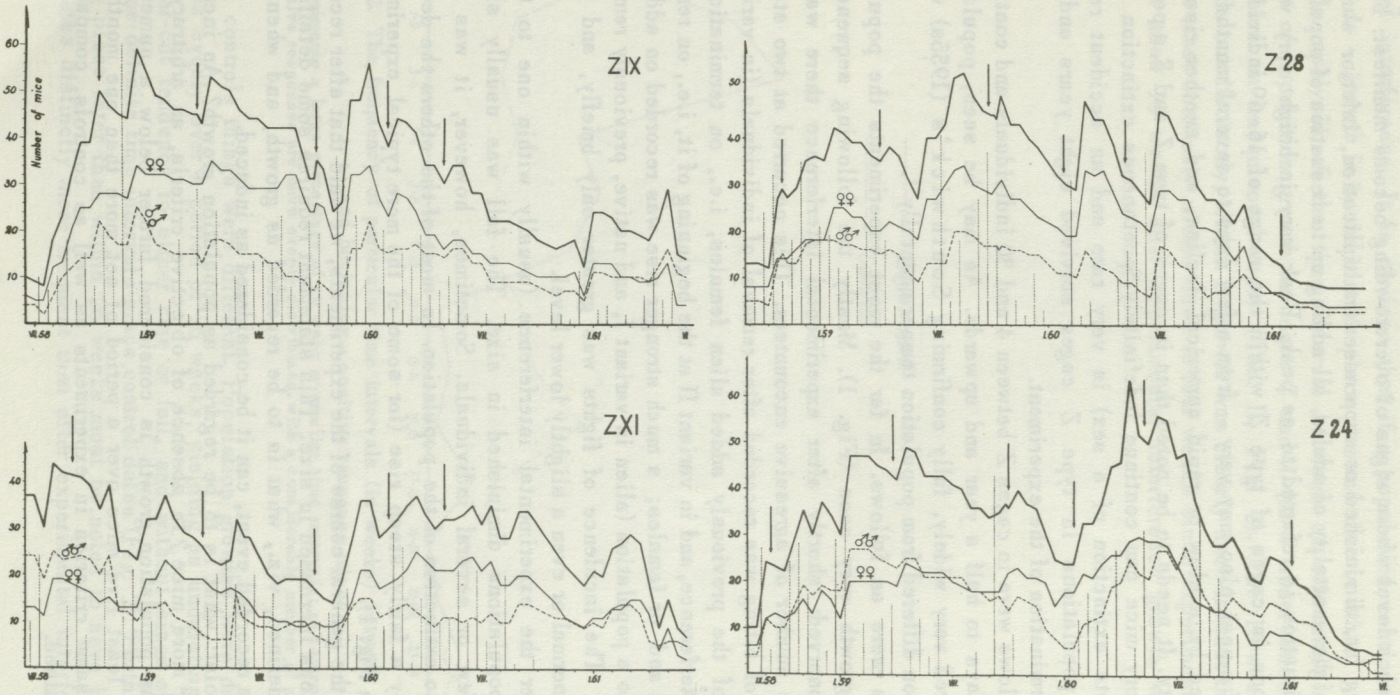

Fig. 1. Examples of quantitative dynamics and course of experiment in populations *Z*

Arrows – experimental interference; verticals total height – number of newborn in 2 weeks; solid vertical – survived up to 3 weeks

In short, 3–10 weeks after the starting of the population the mice began to multiply to reach a level where signs of overcrowding become manifest: biting, shaggy sweating fur, diminished or suppressed multiplication, inferior survival of litters, and higher mortality of adults, all adding up to cessation of population growth. This situation is referred to as peak. Peak vary in height very widely – for populations in cages of type *Z* within the range of 16–69 individuals. The duration of a peak also may vary – from a few days to several months. The peak is followed by a slow or rapid numerical decline, and another rise, and so on alternalety. It needs to be noted than in cages of type *Z* and *B* a population of laboratory mice can continue indefinitely. Spontaneous extinction (e.g., through complete extinction of a sex) is very rare and an accident rather. Some of the populations in type *Z* cages survived eight years and were destroyed on termination of the experiment.

Population lows were in cages *Z* between 4 and 30 individuals and continued from several days to half a year and upwards. As may be seen, population dynamics differed very widely, fully confirming Southwick's (1955a) words that: "Population differed from population temperamentally".

The results were as follows. In far the most experiments the population responded by growth very soon (Fig. 1). Mostly the following sequence of events was observed: shortly after experimental interference there was an increase in the number of agressive encounters. This occurred at two stages: a smaller rise of biting was recorded after removal of individuals (in variant I after removal of the previously added alien females, i.e., on termination of experimental interference, and in variant II at the beginning of it, i.e., on removal of native males and or females; a much stronger rise was recorded on addition of individuals to a population (alien in variant I, and native, previously removed, in variant II). The incidence of fights was greater only briefly, and then returned to the normal or even a slightly lower level.

Shortly after the experimental interference (usually within one to three weeks) most populations diminished in size. The fall was usually slight, mostly by a few or several individuals. Sometimes, however, it was very deep and lead to extinction of the population. In most of the others the decline was followed by a fairly steep rise (for some of the more typical experiments this is shown in Fig. 1).

Describing the general course of the experiments, we said that after recorded events populations increased in size. This statement requires some details and criteria to be defined, viz., what is to be regarded as growth, and when, i.e. how long after a recorded event, can it be considered as induced.

The first point: what is to be regarded as population growth? An increase by one, two, or more mice ? In absence of objective criteria, an arbitrary one was chosen: as population growth is considered further below a numerical incerase by at least five mice over a period of not more than one month. By applying the same criterion in experiments as well as controls, comparable results are obtained.

The other point is at what time can growth be regarded as due to experimental interference. It is obvious that if a population continues – as far the most do – growth is bound to take place at some time or other.

An analysis of the time interval between experimental interference and onset of population growth in cages *Z* and *A* (in *B* observations were too few) supplies an unequivocal reply (Tab. II, Fig. 2). The 10-week time interval stands out distinctly; within it growth was clearly more frequet than after, taking place most commonly within three to four weeks since experimental interference. Later than 10 weeks after experimental interference growth was much rarer.

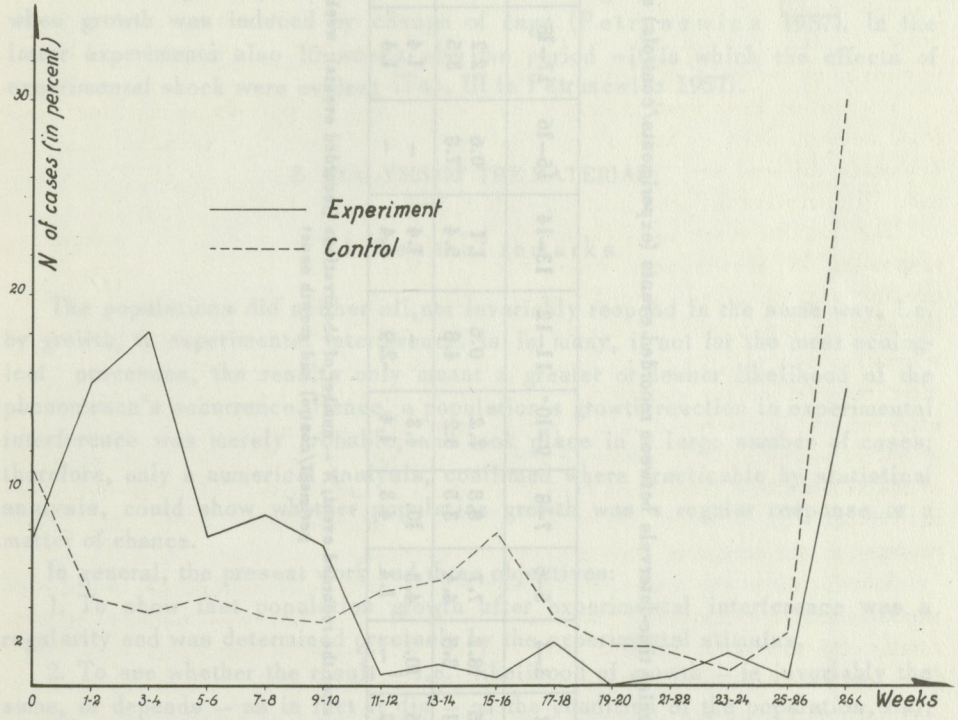

Fig. 2. The frequency of particular time intervals (in weeks) between a recorded event and beginning a positive response onset

In experiments a large percentage of 10 week or smaller time intervals; in controls the percentages of positive responses are more evenly distributed, with a considerable number of negative responses

In controls things were different. Population growths were distributed more or less evenly over a period of 26 weeks, yielding an undulating curve without a clear-cut tendency to either rise or fall, and without sings of concentration in some definite time intervals since control dates (Fig. 2). Furthermore, growth taking place later than within 26 weeks must be noted to have been after control dates distinctly more frequent than after experimental interference. To pu

Frequency of particular time-intervals between recorded events (experiments/controls) and growth onset

Tab. II

	Weeks of eksperiments	0	1–2	3–4	5–6	7–8	9–10	11–12	13–14	15–16	17–18	19–25	> 26	*D*	*N*
Z	E. 1 – 8	7.7	15.5	18.2	7.7	8.8	7.2	0.6	1.1	0.6	2.2	2.8	15.9	11.7	181
	C. 1 – 8	11.1	4.3	3.8	4.4	3.5	3.2	4.8	5.4	7.8	3.5	6.5	35.0	6.7	372
A	E. 1 – 5	8.6	8.6	10.	4.3	10.	4.3	–	1.4	–	1.4	1.4	32.9	17.1	70
	C. 1 – 5	4.3	2.9	–	1.4	4.3	1.4	2.9	4.4	–	1.4	–	65.3	11.7	69

D – population died within 26 weeks after the recorded event; *N* – number of observations (recorded events); weeks – time- nterval between experiment/control and growth onset

it differently,we may say that 26 weeks is in the life-time of a mouse so long a period that if population growth after an event fails to take place within it, that event may be said to have been followed by no growth at all; now, in controls "no growth" was clearly more frequent than in experiments. The relevant figures were for type *Z* 32 percent versus 14 percent, and for type *A* 65 percent versus 32 percent (the difference is statistically significant: $P = .0000$).

With this as a basis, populations were assumed to have responded to experimental interference by growth if the beginning of growth was recorded not later than 10 weeks after[3].

Worth noting, the present results were in full agreement with those obtained when growth was induced by change of cage (Petrusewicz 1957). In the latter experiments also 10 weeks was the period within which the effects of experimental shock were evident (Tab. III in Petrusewicz 1957).

3. ANALYSIS OF THE MATERIAL

3.1. General remarks

The populations did neither all,nor invariably respond in the same way, i.e. by growth, to experimental interference. As in many, if not far the most ecological processes, the results only meant a greater or lesser likelihood of the phenomenon's occurrence. Hence, a population's growth reaction to experimental interference was merely probable, and took place in a large number of cases; therefore, only a numerical analysis, confirmed where practicable by statistical analysis, could show whether population growth was a regular response or a matter of chance.

In general, the present work had three objectives:

1. To show that population growth after experimental interference was a regularity and was determined precisely by the experimental stimulus.

2. To see whether the result – i.e. likelihood of growth – is invariably the same, or depends – as in fact it did – on the condition of the population, i.e., its phase of development or other characteristic qualities.

3. To discuss the presumable causes and mechanism (course) of the processes observed.

The reply is not on all three points of the same character. As to the first two, analysis and discussion warrant an unequivocal reply; as to the third, it will be largely guesswork and hypotheses.

[3] Hence, whenever the expression "positive response" is used further below, it is to be understood as meaning growth of a population that started in either actual experiments or controls not later than within ten weeks after a recorded event, i.e., experimental interference or control date. Conversely, the expression "negative response" "no response" will signify that there was no growth of the population within that period.

Results experiments (percentage of positive responses and statistical analysis)

Tab. III

Series No:	Experiments		Controls		No – fit probability between			Growth preceded by decline (%)		
	N of replicat.	Positive responses (in percent)		N of replication						
		P_1	P_2		P_1, P_3	P_2, P_4	P_1, P_2	Experiment	Control	P
E. 1/C. 1	33	67	34	210	.0040	–.0000	.0001	64	14	.0000
E. 2/C. 2	16	94	32	19	.0000	–.0147	.0000	75	16	.0000
E. 3/C. 2+3	21	81	34	41	.0000	–.0000	.0000	52	15	.0000
E. 4/C.3 +4	20	65	43	42	.0474	–.1869	.0530	65	21	.0004
E. 5/C. 4+5	20	85	46	41	.0000	–.5093	.0004	50	17	.0088
E. 6/C. 5	20	85	43	21	.0010	–.3524	.0019	70	5	.0000
E. 7/C. 6	19	47	21	19	–.7480	–.0000	.0785	26	5	.0001
E. 8/C. 7+8	32	25	13	61	–.0001	–.0000	.1337	22	5	
E. 1–6/C. 1–5	130	78	36	292	.0000	–.0000	.0000	62	15	.0000
E. 7–8/C. 6–8	51	33	15	80	–.0004	–.0000	.0193	24	6	.0000
E. 1–8/C. 1–8	181	65	30	372	.0000	–.0000	.0000	51	13	.0000
E. 1A/C. 1A	10	70	20	10	.0042	–.0000	.0000	60	0	.0002
E. 2A/C. 2A	15	80	33	15	.0006	–.0534	.0052	27	7	.1331
E. 3A/C. 3A	15	47	33	15	–.7114	–.0534	.4286	13	7	.5824
E. 4A/C. 4A	15	27	0	15	–.0022	–.0000	.0183	13	0	.1322
E. 5A/C. 5A	15	14	0	14	–.0000	–.0000	.1471	13	0	.1322
E.1–5A/C.1–5A	70	45	17	69	–.4078	–.0000	.0000	23	3	.0000
E. B/C. B	13	69	18	35	.0018	–.0000	.0000	46	11	.0092

$P_3 = 100\% - P_1$ and $P_4 = 100\% - P_2$ – (percent of negative responses in experiment P_3 and in control P_4).

The line of thought further below does not follow rigidly the points already mentioned. Analysis was applied to particular processes and phenomena, and most of these shed some light not on one only but on two or even all three of the points.

In spite of ample and deliberately collected material it is difficult to claim definitively that population growth after experimental interference is the result of that interference. Even more, the claim can be confirmed only indirectly and cannot become probable except on analysis of many data that support it indirectly; therefore, in the analysis below many and closely related indexes were checked, for only concurrence of conclusions drawn from various indexes warrants the highly probable general reply: growth is the consequence of experimental interference.

The point is that a particular population growth is never certain to heve been due to experimental interference. We can never predict with absolute certainty the shape of a population curve, and therefore we can never be absolutely sure that the growth observed within ten weeks after experimental interference would not have taken place anyway at that particular moment even without that interference. From a previous trend (growth, stabilization, or numerical decline), fecundity, survival of litters, number of fights, condition of the mice, and size of the population, we may indeed predict a population's further development with some probability, but a mere probability it will remain, because the numerical dynamics of confined populations of mice is known to be irregular and variable (Southwick 1955a, Petrusewicz 1957, 1960). Variable is the size at which growth may begin (Southwick 1955a, Petrusewicz 1957, 1960, cf. also Fig. 1). When a large number of cases is considered, we can see there is a certain size at which growth occurs more frequently (Fig. 4 – population size at growth in controls), but when one particular population is considered, forecasts are rather unreliable. Growth may follow decline (to an indeterminate low) directly, or after prolonged stabilization on a low level continuing often several months even. It also is difficult to foretell at any given moment when a decline will cease, and at what level.

We may predict with considerable confidence that within a proximate short span of time there will be no growth (no births, low litter survival, many fights). However, there have been casses where irrespective of low fecundity and high mortality of the young, some litters attained adulthood and distinctly swelled the population's size. But though forecasts of no growth for the proximate future may not infrequently be accurate, a reverse prognosis, i.e., one of population growth is always highly unreliable.

To sum up, no satisfactory way of making forecasts for a population has been found. Hence, in no single case can we have any certainty that growth would not have taken place if there had been no experimental interference. The thesis that growth was the consequence of experimental interference must,

therefore, be substantiated by various circumstantial evidence, mostly by quantitative differences between the results as of experiments and controls, and by comparisons of phenomena (fecundity, survival) before experimental interference with those after it.

In this category of evidence belongs analysis of the time interval between a recorded event and onset of growth. This analysis – let us recall – demonstrated that a control date was not really an event in a population's life: growths were distributed fairly evenly over the entire period after the control date (Table II, Fig. 2). Not so after experimental interference; growths were clearly concentrated in the first ten weeks – i.e., in a period when the effects of experimental interference may be presumed to have been still in operation; this suggests that the higher incidence of population growths was due to experimental interference.

3.2. Probability of growth

3.2.1. Analysis of result uniformity

Within experiments particular series were compared in the percentage of positive responses. Series *Z* were used for the analyses, and series *A* and *B*, as less numerous, served merely for additional comparisons.

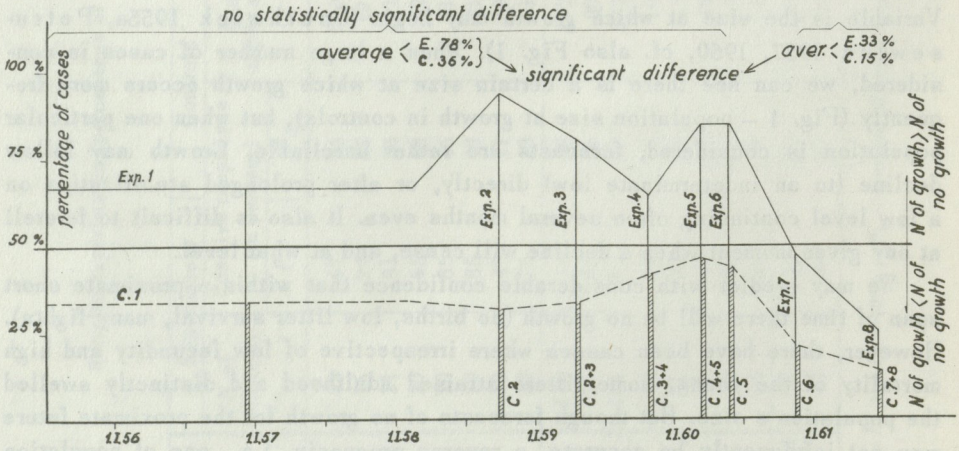

Fig. 3. Percentage of positive responses in populations *Z*.

The differences between series 1 – 6 in the percentage positive responses – invariably larger than 50 – were smaller (statistically not significant); not so in series 7 and 8, which differs from series 1 – 6 and from each other; percentage of positive responses was in experiments invariably higher than in controls

In the first six experimental series of type *Z* the results were found (Tab. III; Fig. 3) to be uniform, viz., 67–94 percent the differences between then being within the limits of chance[4]. Series *Z*7 and *Z*8 (47 percent and 25 percent), however, differed significantly from the first six.

It should be added that in each of the first six series the result is outside the limits of chance (Tab. III), since the percentage of positive responses exceeded negative ones in a statistically significant manner. In series *Z*7 and *Z*8, on the other hand, the percentage of negative responses was higher than that of positive, the difference being in series *Z*8 statistically significant.

Wery similar results were obtained for series *A* (Tab. III). In the first two series the percentage of positive responses exceeded that of negative ones in a statistically significant manner, whereas in the last three series the responses were prevalently negative.

The conclusions that may be drawn from the analysis are: growth-response to experimental interference is not in all populations uniform; the percentage of responses varied with time; in the first series of experiments response was uniform and positive, whereas in the final series response was either indeterminate (about 50 percent of growths) or negative (growths distinctly fewer than "no growth").

32.2. Experiments and controls compared

The results obtained in the final series (either within the limits of chance or negative) may mean either that the reply to the question in point is negative (i.e., that the population does not respond to experimental interference by growth), or that the number of positive responses depends on the condition of the population, and that when in a certain condition the population responds by growth not at all or less readily. Which of the two alternatives obtains is shown by a comparison of experiments with controls in the number of positive responses (the controls were so arranged as to coincide more or less with the experiments – Tab. I). The analysis warrants the following conclusions (Fig. 3 and Tab. III);

1. Negative responses exceeded positive ones in all controls, mostly by a statistically significant margin (Tab. III).

2. In controls the percentage of positive responses in cages *Z* and *A* varied with time in much the same way as in experiments (Fig. 3). In the first five

[4] Here, as elsewhere, the likelihood of agreement (or disagreement) of the percentages was compared with the aid of the criterion $t = \frac{P_1 - P_2}{\sigma}$, where P_1 and P_2 stand for the percentage compared, and σ for the standard error, and $\sigma^2 = \frac{P_1(100-P_1)}{n_1} + \frac{P_2(100-P_2)}{n_2}$, n being the number of observations. The likelihood of agreement between P_1 and P_2 was read from tables with the aid of t.

control series of type *Z* coreesponding to series *Z*1 – *Z*6 the percentage of positive responses was 32 – 46, average 36 percent (differences between series were not statistically significant), and in series *Z*6–*Z*8 only 15 percent (in experimental series accordingly 78 percent versus 33 percent – Fig. 3).

3. In all 13 series (types *Z* and *A* jointly) the percentage of positive responses in experimental series exceeded that in controls by a margin statistically significant in either type separately as well as in the majority of particular series, the exceptions being series *Z*7, *Z*8, *A*3 and *A*5 (Tab. III and Fig. 3).

The analysis suggests that even in series where response was prevalently negative, the question whether a population responds to interference by growth is answered in the affirmative. Positive response of a population to experimental interference is a regularity, but the likelihood of growth (expressed as the percentage of interference followed by growth within ten weeks) varies with time. It is greater in periods where spontaneous growths are more frequent (the first six series of type *Z*, serie *B*, and *A* 1–2), and diminishes with the likelihood of spontaneous growth.

Yet another fact should be noted: in type – *A* cages there is usually only one spontaneous growth; it occurs after the establishment of the population, which then gradually dies out. In the present investigations, concerning 40 populations, further spontaneous growth in addition to the first one occurred only 12 times. In the work on growth induced by change of cages (Petrusewicz 1957), there was not a single case of a second spontaneous growth in cages *A*. Experimental interference, on the other hand, provoked growth in nearly all the 40 populations, in some even more than once.

Additional analysis concerned the percentage of cases where growth was preceded by some decline (Tab. III). This percentage was invariably in the experimental series higher than in controls, and always by a statistically significant margin. Abrupt and often unexpected change of shape in a population curve may mean that after experimental interference the population's development is in character unlike after the random-chosen control dates, i.e., that after experimental interference there are processes at work that differ from those in normal undisturbed development.

32.3. The likelihood of a population's extinction after experimental interference

Discussing the nonuniformity of responses to experimental interference we would call attention to extinction. In *Z* and *B* cages, populations can continue indefinitely. And yet, within the total of 2225 months of life of 80 populations, death of a population (due to extinction of one sex) was recorded 46 times. A very characteristic fact should be noted: populations *Z* were observed from 1954 onwards; now, by April 1, 1960, i.e., in almost 6.5 years and a total of

1733 months of life of 80 populations, 23 populations died out. From June 1960 to June 1961, i.e. in one year – during which the remaining 57 populations *Z* lived jointly 492 months – also 23 populations died out. This concentration of population deaths coincides in time precisely with the period in which the number of spontaneous growths clearly declined, as also did positive responses to experimental interference. This was the time of experimental series 7 and 8, and control series 6 and 8 (Fig. 3).

The reasons of this attenuation of the population (greater mortality, reduced likelihood of growth) could not be reliably ascertained. It could hardly be blamed on "external contitions", such as temperature, feeding, and moisture, since: (1) living conditions, though not fully uniform (if only for diurnal temperature variations), were the same throughout the time of observation as far as ascertainable by the experimenter; and (2) populations bred for different purposes, as well as mice in breeders, exhibited neither a change in "behaviour" nor a greater tendency to die out than in previous years. The only surmise that suggests itself is that excessive frequency and number of experimental interferences brought the experimental populations into a condition in which these were little if at all capable of growth (spontaneous or induced), and prone to depressions; and during a strong depression there is always a greater likelihood of one, usually the male sex dying out, which of course means extinction of the population.

This seems to be confirmed by yet another observation: strikingly, population deaths (extinction of a sex within 26 weeks after experimental interference or control date) were after experimental interference more frequent than after control dates in populations *Z* as well as *A* (series *B* comparised too few populations to be considered) (Tab. II). The explanation may be the following. As already mentioned, experimental interference was frequently, clearly more often than control dates, followed by some numerical decline (Tab. III) (a similar phenomenon was noted when growth was induced by a change of cage – Petrusewicz 1957). Now, it happened that the usually slight decline – averaging 4.1 mice in series *Z*1–*Z*8 – became prolonged and led to a deep depression and ultimate death of the population. The material is not ample enough and we cannot tell, but if the phenomenon were sufficiently frequent to exclude some mere coincidence, experimental interference would appear have two alternative effects: usually (in 65 percent of experiments) it would induce population growth and occasionally (in 11.7 percent of experiments) it would cause population death.

32.4. Relation between results and the phase of the population

An analysis was made of the relation between percentage of positive responses and the phase of the population cycle. With some approximation, the following phases were distinguished in a population's life:

1) Peak – clear symptoms of overcrowding (*Mx*).

2) Decline – decrease by not less than two adults within a month (↘).

3) Stabilization – when growth or decline did not exceed one adult a month (→).

4) Growth – when numerical rise was not less than two adults a month and totalled at least five (↗).

Calculations made for populations *Z*1 to *Z*6 (Tab. IV) warrant the following statements:

1) The percentage of experiments at particular phases in the life of populations matched that of control dates fairly well. This means that at the time of experimental interference the natural probability growth was neither greater nor smaller than at the time of control dates.

2) Irrespective of the phase, positive responses was after experimental interference much more frequent than after control dates.

3) The frequency of growth after experimental interference, expressed as the percentage of positive responses, differed some what between particular phases. It was less at peaks and declines, and greater at the time of either stabilization or growth. The range of similar differences in controls was much greater, viz., 13–98 percent versus 71–92 percent in experiments (Tab. IV). Furthermore, the frequency of positive responses in experiments exceeded that in controls much more conspicuously in phases of peaks and declines. viz., 3.2 times and 5.7 times respectively, the average calculated from all four phases being only 2.2 times (cf. Tab. IV).

In experiments, therefore, a relatively large proportion of population growths may be concluded to have been due to experimental interference especially at times when there was little likelihood of spontaneous growth (at peaks and declines).

32.5. Summing up

Summing up considerations on growth likelihood, reflected in the percentage of positive responses, we may say that:

1) Growth probability was in all series after experimental interference higher than after control dates.

2) The percentage of positive responses was in experiments roughly constant (uniform) at times when it was so in controls. But when it showed a downward trend in controls, it did the same in experiments. Hence, the likelihood of growth after experimental interference depended on the general condition of the population.

Number of recorded events and positive responses in particular population phases in series Z 1–6
(in percentage of sum of experimental interferences and control dates separately)

Tab.IV

Phase	Number of		Percentage of observation in:		P	Percentage of growths		P	% experiment
	experiment	control	experiments	controls		experiment	control		% control
Mx	32	61	25	22	.3734	71	22	.0000	3.2
↘	51	131	39	45	−.2461	74	13	.0000	5.7
→	16	38	12	13	− 7642	81	55	.0404	1.5
↗	25	51	19	17	.6242	92	98	−.6093	0.9
?	6	11	5	3	–	–	–	–	–
Total or average	130	292	100	100	–	78	36	–	2.2

Average population size (number of mice) at the time of: recorded events, growth onset and peak
(All means ave weighted means)

Tab. V

Series No	Recorded events			Onset of growth			Peak		Growth in percent	
	experiment	control	*P*	experiment	control	*P*	experiment	control	experiment	control
E.1/C.1	24.4	25.0	−.7642	19.1	16.6	.1096	36.4	2[illegible]	190.6	176.5
E.2/C.2	33.6	36.7	−.5485	29.7	23.0	.1336	46.7	3[illegible]	157.2	152.2
E.3/C.2+3	36.3	33.7	.3681	32.6	21.5	.0001	43.0	34.5	131.9	160.5
E.4/C.3+4	35.2	31.2	.3173	24.9	21.2	.1336	36.1	34.8	144.9	164.1
E.5/C.4+5	29.5	30.5	−.9203	28.3	22.2	.0891	42.4	34.8	149.8	156.7
E.6/C.5	35.6	29.2	.2301	37.4	21.9	.0001	51.2	34.1	136.9	155.7
E.1–6/C.1–5	31.7	26.9	.0574	28.4	17.9	.0000	42.2	30.8	148.6	172.1
E.7/C.6	34.8	34.8	1.0000	24.1	20.5	.5485	38.5	31.7	159.7	154.6
E.8/C.7+8	28.4	27.5	.8414	18.6	19.2	−.7642	28.7	25.5	154.3	132.8
E.7+8/C.6–8	31.6	29.4	.7699	21.4	18.4	.3681	33.6	28.4	157.0	154.3
E.1–8/C.1–8	31.6	27.5	.0681	27.4	18.0	.0001	40.4	30.7	147.4	170.5
E.1*A*/C.1*A*	10.3	8.0		10.1	9.1		16.2	14.1	160.4	154.9
E.2*A*/C.2*A*	17.0	19.2		16.8	16.9		27.7	27.1	164.9	160.3
E.3*A*/C.3*A*	21.5	21.3		20.4	18.4		26.6	27.6	130.4	150.0
E.4*A*/C.4*A*	19.0	18.2		16.2	–		21.7	–	133.9	–
E.5*A*/C.5*A*	11.7	12.3		14.0	–		21.0	–	150.0	–
E.1–5*A*/C.1–5*A*	15.4	14.4		15.1	14.4		23.1	20.1	153.0	139.6
E.*B*/C.*B*	36.7	40.3		33.2	29.2		52.5	51.3	158.1	175.7

3.3. Size of population

33.1. Size of population at the time of recorded events

Comparisons were made concerning the size of populations as at the time of recorded events. This was done to see whether the size was not in experiments smaller than in controls, which could mean that the experiments were made at a time when the populations involved were more apt to grow than were those in controls (growth is less likely when a population's numerical status is high).

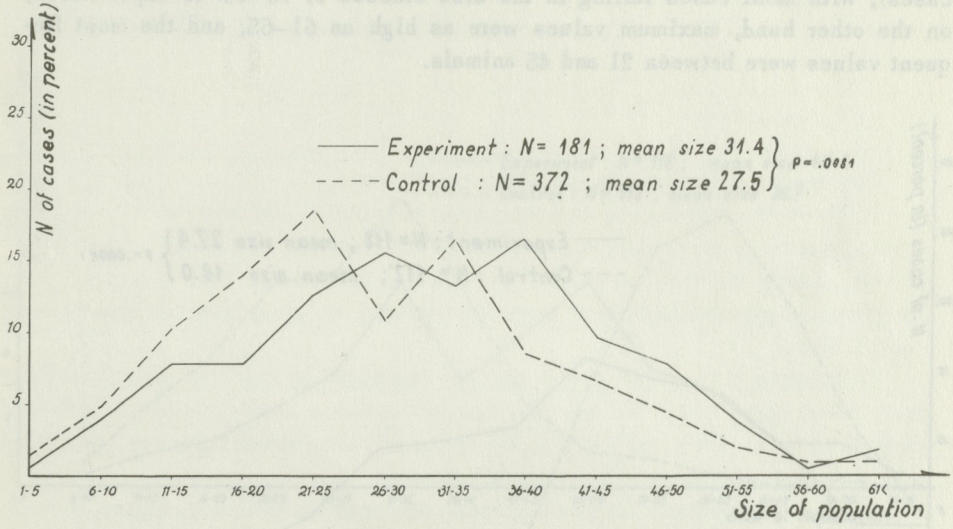

Fig. 4. Size of population (type Z) at the time of the recorded event.
The curves as from experiments and controls are alike

Analysis of size averages in particular series (Tab. V) and size distribution for all experiments of type Z (Fig. 4) show there were no distinct differences in population size between experiments and controls at the relevant moments. The size was larger sometimes in experimental series, and sometimes in control series, but the difference was statistically significant neither between the particular series nor for the for the material as a whole (checked with Student's test). This means that the size was in experiments roughly the same as in controls and that, therefore, the experimental populations were in no way naturally more predestined to grow.

33.2. Population size at the beginning of growth

Next, experiments and controls were compared in population size at the beginning of the positive response. Only series Z were compared, since in

two of control series *A* there never was either induced nor spontaneous growth, and in the remaining series *A*, control and experimental alike, the number of positive responses was small (2–12).

The results are completely different from those obtained on comparison of experiments and controls in population size at the moment of recorded events. Population size at the beginning of positive response was in experiments distinctly greater than in controls. This is borne out by the following.

1) Distribution of the population sizes recorded at the beginning of growth (Fig. 5). In experiments the size values were shifted to the right, i.e., towards the higher values. In controls, maximum values were 31–35 (in 1.1. percent of cases), with most cases falling in the size classes of 16–25. In experiments, on the other hand, maximum values were as high as 61–65, and the most frequent values were between 21 and 45 animals.

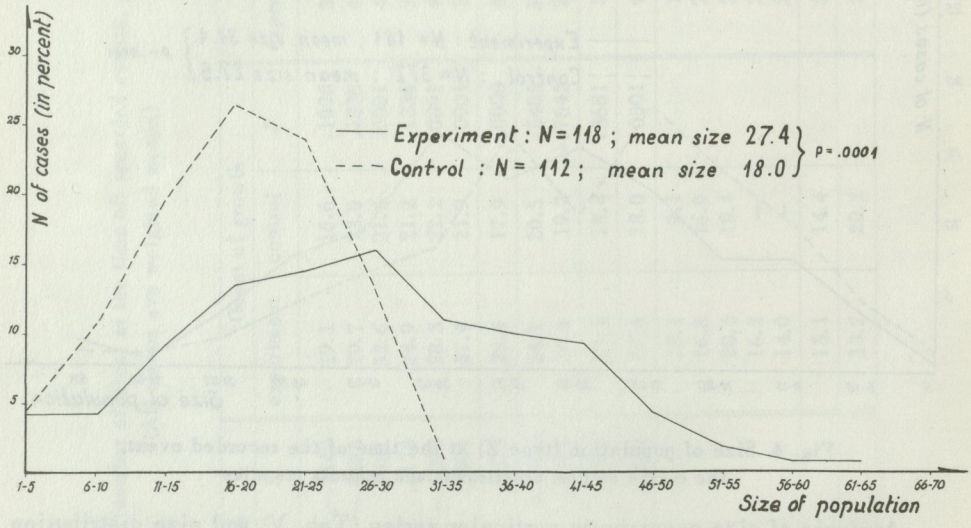

Fig. 5. Size of population (type *Z*) at the time of the beginning growth onset (which begins to 10 weeks from recorded events).
As opposed to controls, growth-starting levels were in experiments much higher

2) Comparison of experiments and controls in population size at the beginning of growths. Throughout all except the 8th series the starting size was in experiments higher than in controls. For all *Z* series jointly, the difference was statistically significant (Student's test, $P = .0000$; Tab. V).

3) Experimental interference was able to stimulate growth in populations at a size level higher than that at which spontaneous growth was ever noted to occur. The latter was 32 at the most (in cages of type *Z*), whereas experimental interference started growth from levels higher than 35 in 27 percent of cases, and from a maximum as high as 62.

It should be noted that this was very much like in growth stimulation by change of cage (P e t r u s e w i c z 1957).

33.3. Population size at peaks

Experiments were compared with controls in the maxima, or peaks, attained during positive responses. In all of *Z* series positive response rose in experiments higher than in controls, although the difference was statistically significant only for all the series jointly. The distribution of the particular values recorded (Fig. 6) also suggests that the peaks were higher in experiments

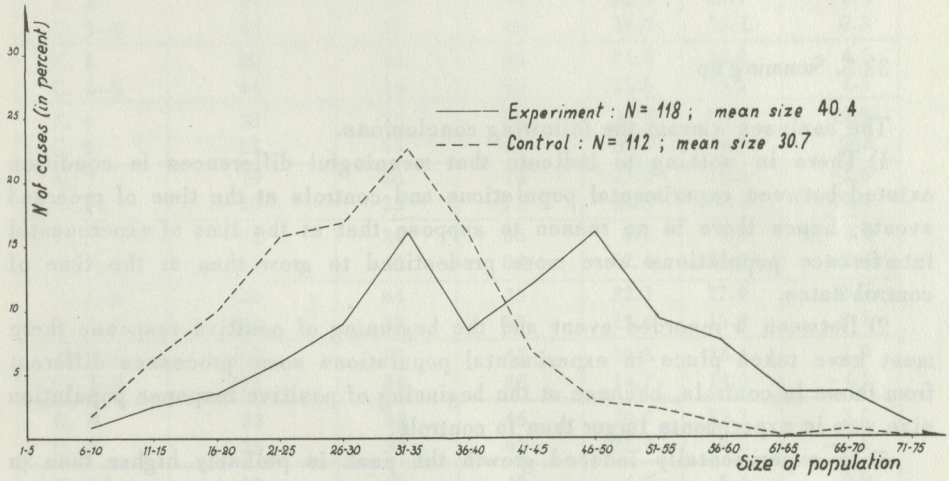

Fig. 6. Population levels at experimentally induced peaks

rather. In series *A* and *B*, though, this is not so conspicuous (Tab. V). The peaks were in experiments so little higher than in controls that the difference was statistically significant neither for particular series nor for all the series 6 jointly; in series *A*3 the peak was actually higher in controls. An explanation of this may be that in series *A* peak averages were calculated from a very small number of replications – 2–12 in experiments, and only 5 in controls. This small number of replications may have had the accidental effect of raising the average for controls. The conclusion is that we cannot claim definitively that population peaks induced by experimental interference to exceed those in spontaneous growth, even though this seems likely, as having been noted in all *Z* – i.e., long – series.

33.4. Growth magnitude

Experiments do not seem to differ substantially from controls in growth magnitude, which is the peak in percents of the starting level $(\frac{\text{peak level}}{\text{starting level}} \cdot 100)$ (Tab. V). The average in series Z and B jointly is indeed larger for spontaneous growth, but the difference is not statistically significant. At first sight this appears puzzling, considering that induced peaks were higher than spontaneous ones, but is readily accounted for by the fact that induced growth started from higher levels (cf. Tab. V). In populations Z the ratio of starting levels as in induced and spontaneous growth was 27.4 : 18.0 = 1.5, whereas the ratio of peaks was 40.4 : 30.7 = 1.3, i.e., smaller, though still statistically significant.

33.5. Summing up

The analyses warrant the following conclusions.

1) There is nothing to indicate that meaningful differences in condition existed between experimental populations and controls at the time of recorded events; hence there is no reason to suppose that at the time of experimental interference populations were more predestined to grow than at the time of control dates.

2) Between a recorded event and the beginning of positive response there must have taken place in experimental populations some processes different from those in controls, because at the beginning of positive response population size was in experiments larger than in controls.

3) In experimentally induced growth the peak is probably higher than in spontaneous growth.

3.4. Population fecundity and survival of litters

34.1. Fecundity before and after recorded events

Fecundity of a population was defined as the number of individuals born in one month, and was determined as for the month:

1) preceding a recorded event (F), and

2) following a recorded event (F').

The percentages as of cases where fecundity was after the recorded event greater than before it ($F' > F$) and where it was not ($F' \leqslant F$) were calculated separately for each series. Compiled in Table VI, the data show that:

1) The percentage of cases where fecundity was after a recorded event greater ($F' > F$) was in experiments higher than in controls in all 14 series

Fecundity before (F) and after (F') a recorded events

Tab. VI

Series	N of replication	Percent of replication where		Average size of:		
		$F' > F$	$F' \leqslant F$	$\overline{F}$	$\overline{F'}$	$\overline{F'} - \overline{F}$
E. 1	33	55	45	21.8	23.8	2.0
C. 1	210	33	67	11.2	9.5	−1.7
E. 2	16	75	25	25.4	35.2	9.8
C. 2	19	53	47	16.9	18.4	1.5
E. 3	21	67	33	22.8	25.7	2.9
C. 2–3	41	51	49	17.7	16.6	−1.1
E. 4	20	65	35	22.3	25.7	3.4
C. 3–4	42	52	48	14.8	15.1	0.3
E. 5	20	55	45	21.3	25.8	4.5
C. 4–5	41	44	56	15.4	16.6	1.2
E. 6	20	75	25	19.3	31.2	11.9
C. 5	21	33	67	20.5	20.7	0.2
E. 7	19	26	74	17.5	15.5	−2.0
C. 6	19	25	75	18.5	8.1	−10.4
E. 8	32	34	66	5.7	4.3	−1.4
C. 7–8	61	16	84	12.4	10.3	−2.1
E. 1–6	130	64	36	22.1	27.9	5.7
C. 1–5	292	37	63	15.6	15.8	0.1
E. 1–8	181	55	45	19.5	23.4	3.9
C. 1–8	372	34	66	14.7	14.0	−1.2
E. *B*	13	54	46	19.7	22.7	3.0
C. *B*	35	37	63	27.1	19.5	−7.6
E. 1*A*	10	60	40	4.5	5.4	0.9
C. 1*A*	10	30	70	2.1	1.1	−1.0
E. 2*A*	15	67	33	10.6	13.3	2.7
C. 2*A*	15	13	87	16.3	9.9	−6.4
E. 3*A*	15	47	53	9.7	5.9	−3.8
C. 3*A*	15	20	80	8.5	3.1	−5.4
E. 4*A*	15	47	53	1.3	3.5	2.2
C. 4*A*	15	13	87	1.3	1.1	−0.2
E. 5*A*	15	20	80	0.4	1.3	0.9
C. 5*A*	14	14	86	1.0	1.6	0.6
E. 1–5*A*	70	47	53	5.3	5.9	0.6
C. 1–5*A*	69	17	83	5.8	3.4	−2.5

(the difference is statistically significant for all series in cages either Z or A, or B – Tab. VI).

2) In experiments: the percentage of cases where fecundity was after experimental interference greater $F' > F$ than before $F' \leqslant F$ was distinctly higher,

and therefore clearly better than 50. This was so in all except the last two series (*Z*7 and *Z*8). The difference was statistically significant for all series *Z* jointly ($P = .0000$).

3) In controls the reverse was true: the percentage of cases where fecundity was after the control data greater than before ($F' > F$) was smaller, and, therefore, less than 50; the difference, however, was not statistically significant (cf. 34.5).

34.2. Average fecundity before and after recorded events

Average fecundity as before ($\bar{F}$) and after ($\bar{F}'$) a recorded event was calculated for each series.

Analysis of the averages warrants the following conclusions (Tab. VI).

1) In most experimental series, except *Z*7, *Z*8, and *A*3, average fecundity was before a recorded event greater than after; the difference was statistically significant for all series *Z* jointly ($P = .0000$); for series *A*, $P = .0676$.

2) In controls the difference was virtually nil; average fecundity was indeed after recorded events smaller than before in all series *Z* jointly, and so it was in series *A* and *B* ($\bar{F}' < \bar{F}$), but the difference was statistically significant only in series *B* (cf. also 34.5).

3) In all 14 series the difference $\bar{F}' - \bar{F}$ was in experiments greater than in controls by a margin statistically significant in all series *Z* jointly and in serie *B*. The distribution of the values $\bar{F}' - \bar{F}$ (Fig. 7) as obtained for particular

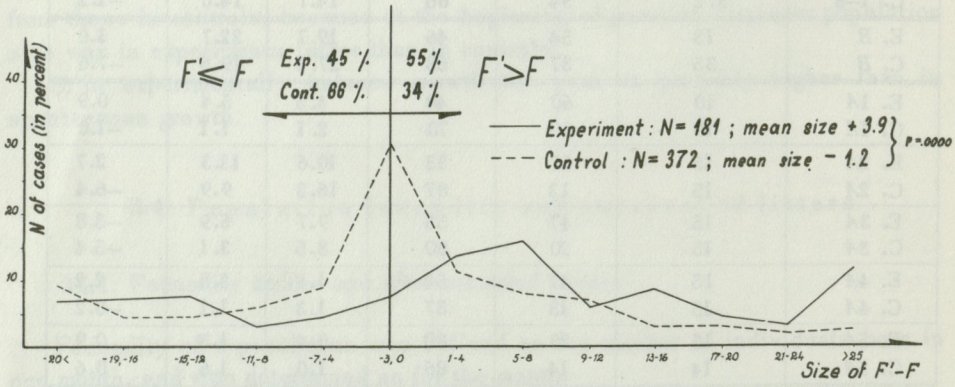

Fig. 7. Differences between fecundity as after (F') and before (F) recorded events

recorded events also shows that the values were in experiments predominantly positive, as also was the overall average, whereas in controls the opposite was true (the grand mean – as for experiments versus controls – was for *Z* + 3.9 versus −1.2; for series *A* + 0.6 versus −2.5; and series *B* + 3.0 versus −7.6).

General conclusion: after experimental interference fecundity is higher than before; on the other hand, fecundity is alike before and after control dates.

34.3. Survival of litters before and after recorded events

Survival of litters was defined as the percentage of the young that survived three weeks, i.e., became self-dependent components of the population.

It was calculated for the last two months preceding a recorded event (S), and the first two months after it (S').

The percentage of cases where survival was after a recorded event higher than before ($S' > S$) was compared with that of cases where it was not ($S' \leqslant S$) for each series separately The results, recorded in Table VII, show the following:

1) In all 14 series the percentage of cases where $S' > S$ was in experiments higher than in controls by a margin statistically significant for all series jointly within the particular types Z, A, and B considered separately.

2) The percentage of cases where $S' > S$ exceeded that of cases where $S' \leqslant S$ in most experimental series except $Z7$, $Z8$, and $A3$–5, the margin being statistically significant in series Z as well as A.

3) In controls, the percentage of cases where $S' \leqslant S$ exceeded that of cases where $S' > S$ in all series (i.e., more often than not survival was before a control date larger than after it); the difference was statistically significant except C. 3–4.

34.4. Levels of survival before and after recorded events

In addition to the percentage of cases where survival was before a recorded event greater than after it, or not, the level of survival also was investigated.

Distribution of the values $S' - S$ obtained from particular experiments (Fig. 8) shows that these values were in experiments such higher than in controls. This is confirmed by an analysis of serial averages of survival as before ($\overline{S}$) and after ($\overline{S}'$) recorded events, which gave the following results:

1) In experiments, the value $\overline{S}' - \overline{S}$ was positive in 13 out of 14 series, i.e., survival of litters was after experimental interference on a higher level than before. The difference was statistically significant for series Z, A, and B.

2) In controls, average survival was in most series (viz., in 10 out of 14) greater before than after control dates (i.e. the difference $\overline{S}' - \overline{S}$ is negative) but the difference was not statistically significant (Student's test).

Survival before (S) and after (S') a recorded events

Tab. VII

Series	N of replication	Percent of replication where		Average size of:		
		$S' > S$	$S' \leqslant S$	$\bar{S}$	$\bar{S}'$	$\bar{S}' - \bar{S}$
E. 1	33	64	36	9.9	22.5	12.6
C. 1	210	22	78	13.6	9.9	−3.7
E. 2	16	69	31	22.5	29.5	7.0
C. 2	19	21	79	6.7	8.4	1.7
E. 3	21	71	29	1.6	23.1	21.5
C. 2–3	41	34	66	8.1	12.9	4.8
E. 4	20	65	35	11.0	23.6	12.6
C. 3–4	42	45	55	22.0	19.8	−2.2
E. 5	20	60	40	22.6	32.5	9.9
C. 4–5	41	37	63	29.7	25.9	−3.8
E. 6	20	65	35	35.2	41.1	5.9
C. 5	21	29	71	38.4	27.4	−11.0
E. 7	19	47	53	12.4	18.9	6.5
C. 6	19	21	79	19.7	13.7	−6.0
E. 8	32	22	78	7.4	12.3	4.9
C. 7–8	61	12	88	8.0	6.8	−1.2
E. 1–6	130	65	35	17.1	28.7	11.6
C. 1–5	292	26	74	18.5	17.1	−1.5
E. 1–8	181	56	44	15.3	25.4	10.1
C. 1–8	372	24	76	16.2	14.1	−2.0
E. *B*	13	54	46	13.0	21.7	8.7
C. *B*	35	31	69	17.7	14.1	−3.6
E. 1*A*	10	80	20	21.8	38.1	16.3
C. 1*A*	10	10	90	5.0	5.0	0.0
E. 2*A*	15	60	40	35.2	32.1	−3.1
C. 2*A*	15	33	67	37.3	18.1	−19.2
E. 3*A*	15	47	53	21.9	22.3	0.4
C. 3*A*	15	20	80	29.5	10.4	−19.1
E. 4*A*	15	33	67	3.4	17.5	14.1
C. 4*A*	15	7	93	0.0	6.7	6.7
E. 5*A*	15	13	87	3.3	10.3	7.0
C. 5*A*	14	0	100	7.1	0.0	−7.1
E. 1–5	70	44	56	17.1	24.1	6.9
C. 1–5	69	14	86	15.8	8.0	−7.7

34.5. Discussion of control results

It has already been said that both fecundity and survival were before the random-chosen control dates greater than after. This was evident in both the

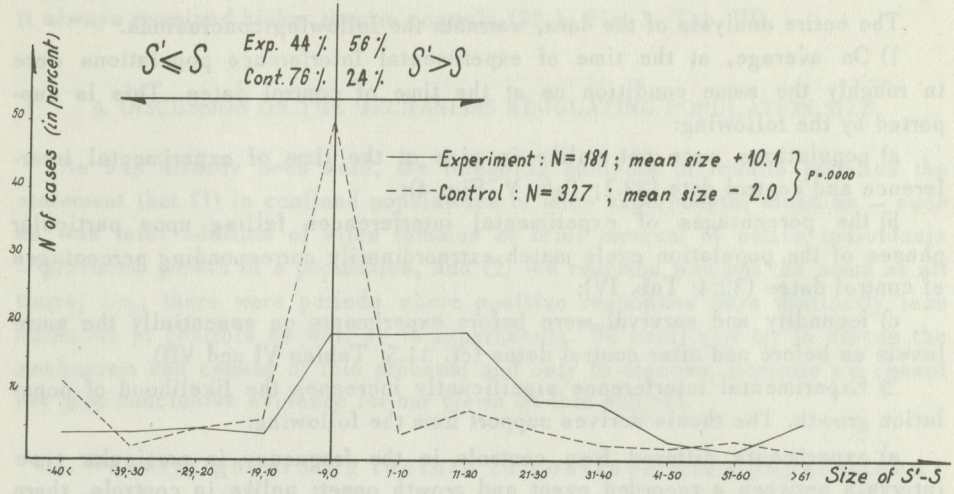

Fig. 8. Differences between suckling survival as after (*S'*) and before (*S*) recorded events

average values and the percentage of cases where either fecundity or survival was so. The difference was not always statistically significant, to be sure, but the fact that it existed in all control series invites attention. Why fecundity was after control dates – chosen in great numbers and at random – unlike before may perhaps be explained in the following way. Decline in usually much more prolonged than growth (population curves slant to the right over the most part); hence, there is always more likelihood of a control data falling upon decline rather than on growth, and during decline fecundity and survival usually have a downward trend. Therefore, more often than not fecundity and survival are after random-chosen time points lower than before. Consequently, the fact than after experimental interference fecundity and survival not only fail to diminish but even rise takes on still greater significance. This cannot be explained except as an effect of experimental shock.

34.6. Summing up

The results of fecundity and survival analyses may be summed up as folows.

1) Compared with what they had been before a control date, fecundity and survival were after it the same or slightly smaller (cf. 34.5).

2) Fecundity and survival as before experimental interference differed little, if at all, from those before and after control dates.

3) After experimental interference fecundity and survival were higher than before it, and higher than before and after control dates.

3.5. Summing up of the results of analysis

The entire analysis of the data, warrants the following conclusions.

1) On average, at the time of experimental interference populations were in roughly the same condition as at the time of control dates. This is supported by the following:

a) populations were not unlike in size at the time of experimental interference and control date (33.1; Tab. V, Fig. 4);

b) the percentages of experimental interferences falling upon particular phases of the population cycle match extraordinarily corresponding percentages of control dates (32.4; Tab. IV);

c) fecundity and survival were before experiments on essentially the same levels as before and after control dates (cf. 34.5; Tables VI and VII).

2) Experimental interference significantly increases the likelihood of population growth. The thesis derives support from the following:

a) experiments differed from controls in the frequency in particular time intervals between a recorded event and growth onset; unlike in controls, there was in experiments conspicuous concentration of growth oncets, and it fell upon the first ten weeks (2. Tab. II, Fig. 2);

b) the percentage of cases where positive response (growth) followed a recorded event was in experiments greater than in controls (32.2; Fig. 3).

c) the percentage of cases where experimental interference was followed by growth (positive response) was greater than that of cases where it was not (32.2; Tab. III; Fig. 3).

d) the percentage of cases where growth (positive response) was preceded by some decline was in experiments clearly greater than in controls, which suggests a change in the population curve due to experimental interference (32.2; Tab. III);

e) experiments differed from controls in the percentage of positive responses that fell upon particular phases; in experiments the percentage was for peaks and declines (i.e., for phases in which the likelihood of spontaneous growth is small) greater than would follow from the probability of spontaneous growth in these phases (3.2; Tab. IV);

f) in experiments, positive response could start from higher population levels than in controls (than could spontaneous growth) (33.2; Fig. 5; Tab. V);

g) in induced growth the peak is probably higher than in spontaneous growth (33.3; Fig. 6; Tab. V).

h) fecundity and survival were after experimental interference greater than before it and greater than before and after control dates (3.4; Tables VI and VII; Fig. 7 and 3).

3) The results of experiments were not uniform; they depended on the phase upon which a recorded event fell and on the population's general predisposition for growth. At times where the likelihood of spontaneous growth was diminish

ing, so also was in experiments the number of positive responses, although it always remained higher than in controls (32.1; Fig. 3; Tab. III).

4. DISCUSSION ON THE MECHANISMS REGULATING POPULATION SIZE

As has already been said, the foregoing analysis of results justifies the statement that (1) in confined populations of mice experimental stimulus – such as was brief addition of alien females or brief removal of native individuals – provoked growth of a population, and (2) the reaction was not the same at all times, i.e., there were periods where positive responses were distinctly less numerous in controls as well as in experiments. We shall now try to discus the mechanism and causes of this process; and only to discuss, because we cannot yet give conclusive evidence for our views on this subject.

4.1. Density governing factors in confined mice populations

As has already been said, a population of laboratory mice can live indefinitely in a cage of type *B* (160 × 80 cm) and *Z* (80 × 80 cm). In cages *Z*, the populations was observed averaged a life-time of about 28 months (80 populations lived a grand total of 2225 months). However, this figure is not conclusive, since when observation was discontinued 34 of the 80 populations were still and would have lived on and raised that figure in future, postexperimental time. The longest period of observation in *Z* cages was 8 years (populations Z2, ZI, ZXI, and ZXII). Some 20 populations lived each 4–5 years.

Even though widely varying between populations and variable for single populations, size curves showed certain regularities. So it was possible to note the most frequent level attained by peaks (although its range was fairly wide, viz., 26–46 individuals; Fig. 5, Petrusewicz 1957, 1960). Growth-starting levels were confined within narrower limits: usually growth commenced at levels of 16 to 25 individuals, the full range (Fig. 4) being 4–32 individuals (and 4–30 in Petrusewicz 1957, 1960). Growth usually lasted shorter (0.5 to 2 months at the most) than did decline, which not infrequently continued 12 months and more (probably this also is why fecundity was after control dates usually lower than before; Tab. VI; 3.4). Many other regularities might still be added, but these will be the subject of a separate paper because at the moment it suffices to say that some regularities in the dynamics of confined populations of mice did exist.

Let us recapitulate the following findings: even with excess of food, confined populations of mice do not multiply indefinitely; peaks usually attain levels of which we know that in "abstract terms" they do not exhoust the habitat's ecological capacity (usual peaks are 26–46, whereas the maximum observed under exactly the same conditions was 69); the populations in point

could have lived indefinitely, varying in size in keeping with certain regularities. All these findings suggest certain mechanisms controlling the size of confined populations of mice.

By such regulatory mechanisms are meant processes that tend automatically to confine population size within certain limits by restricting excessive growth as well as decline. Apparently such processes must promote growth when size diminishes or has diminished below a certain level, and must inhibit growth when size rise or has risen above some ceiling. The nature of these processes is well rendered by the term "feedback", now in vogue to the interest aroused by cybernetics, but long-know in ecology.

Where do these regulatory processes operate? All processes that restrict or increase population size (consequently also regulatory processes) are nowadays attributed in ecology to the effects exerted on organisms by: environment, biocoenosis, or population itself. In our experiments, environmental conditions were more or less constant and the same for all populations and could not, therefore, regulate the size of populations. Especially, it is hard to imagine constant conditions as restricting population size to different levels. It also is difficult to imagine biocoenotic conditions as responsible for size regulation in the experiments, since the biocoenosis here in point was very poor and incomplete. Food, as one of the most important factors, was always overabundant. There were no carnivores only bacteria and other germs can be considered, as well as a few ectoparasites, against which the populations were protected as much as possible, and rather effectively. In practical respects, therefore, the populations lived outside any biocoenosis.

Consequently, the regulatory mechanisms cannot be imagined except within populations and must be so-called self-regulatory mechanisms. In other words, within the populations themselves processes must be at work that are capable of automatically restricting their excessive growth as well as excessive decline.

It would appear simplest to regard density as responsible for regulatory processes and to seek density-dependent factors and processes. It would seem natural precisely for density to regulate size through fenomena attendant upon overcrowding. Yet there is much to refulte this. Turning points in the life of populations occurred at widely varying size levels (hence also densities, since the area was constant). Spontaneous growth began at size levels of 4–32 individuals. Peaks (end of growth and beginning of decline) were 16–69 individuals. Thus, growth may start from levels as low as a few individuals at one time, and as high as 30 at another. Similarly, decline may continue to anything between these levels. Moreover, these levels, vary not only between but also within populations. Hence, the situation shown in the hypothetical population curve in Fig. 9 can, and not infrequently does occur. At the level of for instance, 20 individuals, i.e., at a definite density, we have at *A* growth, at *B* a trough (beginning of growth), and at *C* decline; at *D*, on the other hand, i.e., at a den-

sity less than at *A* and *B*, where we had growth and a trough respectively, we have a peak, i.e., symptoms of overcrowding and beginning of decline. A case like this hypothetical example is oft-seen in nature, and may be noted repeatedly in the curves for populations ZVIII and Z 24 (Fig. 1).

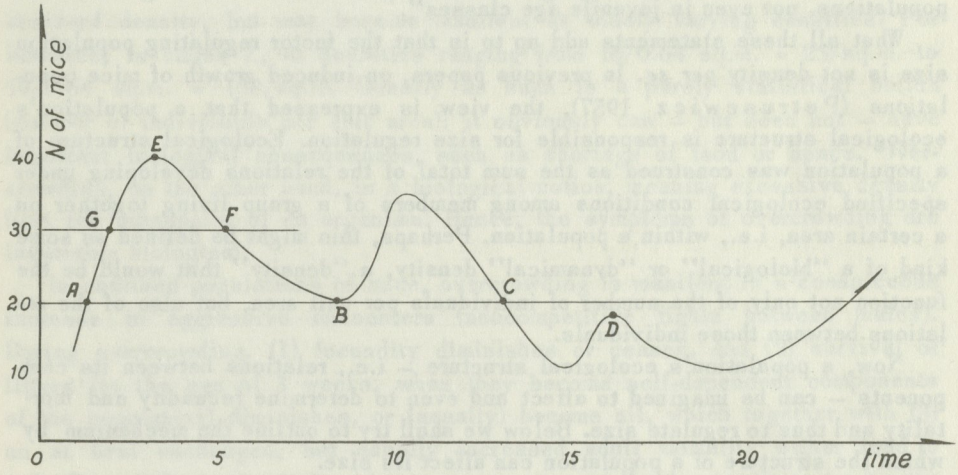

Fig. 9. Hypothetical population curve (explanations in the text)

The foregoing clearly shows that density as such is not the factor that regulates size, at least not in confined populations. Incidentally, this idea is not new in ecology. While reserving our views on the fundamental question – still not settled in discussions between ecologists – of whether it is useful and at all practicable to distinguish between density-dependent and density-independent factors, we shall refer to some papers that show population size to be related not to density, or not only to density, or not directly to density.

For instance, from studies on confined populations of mice with a limited and constant supply of food, Strecker and Emlen (1953) concluded that fecundity diminishes as the population grows not only owing to want of food (i.e., overcrowding) but also owing to social factors. In discussing the factors restricting the size of confined populations, Southwick (1955a) said they "were related to crowding and confinement, but not to density per se"; and in a later work (1953) he concluded that "Mortality rates of the populations revealed no dramatic or conspicuous change throughout the density classes studied". Calhoun (1956) believes population growth to be affected by differences in social behaviour, noted between verious strains of investigated mice. From three-year observations of four outdoor confined populations of *Microtus arvalis*, Wijngarden (1960) concluded that there are ecological processes highly significant in a population's life that are not directly related to density. He wrote: "The factor which was to become restrictive in our case

was – besides a slight decrease in the rate of increase in connection with the population density – the space in which they could make a sufficient number of burrows". At the same time he believes "There is no indication that the rate of decrease of the males was larger than that of the females" and concludes "In general I may say that mortality was not density-dependent in these confined populations, not even in juvenile age classes".

What all these statements add up to is that the factor regulating population size is not density *per se*. In previous papers, on induced growth of mice populations (Petrusewicz 1957), the view is expressed that a population's ecological structure is responsible for size regulation. Ecological structure of a population was construed as the sum total of the relations developing under specified ecological conditions among members of a group living together on a certain area, i.e., within a population. Perhaps, this might be defined as some kind of a "biological" or "dynamical" density, a "density" that would be the function not only of the number of individuals per unit area, but also of the relations between those individuals.

Now, a population's ecological structure – i.e., relations between its components – can be imagined to affect and even to determine fecundity and mortality and thus to regulate size. Below we shall try to outline the mechanism by which the structure of a population can affect its size.

4.2. Regulatory mechanisms in confined populations

Density has above been shown not to be the factor directly controlling the size of confined populations of mice. The latter (consequently also density) has been suggested to depend on the population's structure. Let us now consider by what ways can structure regulate the size of a population, what are the mechanisms involved. Here it must be understood from the beginning that the hypotheses will apply only to confined populations of mice bred in rather small space, since the mechanisms suggested will be based chiefly on hierarchy and noncompetitive fights between males. Possibly, and even probably, altogether different structural moments will be decisive in free-living populations. Without any attempt to exhaust this problem, I shall merely indicate some selected differences. Migration is known to affect vitally, often decisively, population dynamics and structure (Naumov 1956; Andrewartha 1961), wheras in confined populations this moment does not exist. Relations between individuals obviously develop differently when spatial relations can evolve. Also, the density attained in cage Z populations 48/ sq.m!) is incomparably higher than in any free-living population (Evans 1949, regards as little as 3 mice/sq.m. as a very high density, and even overcrowding, and Tupikova reports 10–18/100 sq.m. as the highst density known).

We believe the essential mechanism of regulation to be the following. Normal and healthy mice, usually the majority in a population, are always potenti-

ally capable to multiply and increase their numbers. Hence, after a preliminary period of organization, characterized by aggressive relations between males, a certain hierarchy becomes established, which is based on mutual discrimination of particular individuals. Next, numerical increase leads eventually to overcrowding. This overcrowding cannot be gauged by any fixed reference or standard density, but may become manifest at widely varying densities. For instance, in cages Z, at densities ranging from 16/0.64 sq.m. = 25/sq.m. to 69/0.64 sq.m. = 108/sq.m. Density as such is a purely statistical notion (number of individuals per unit area); it obviously can – but need not – have important biological consequences, such as shortage of food or space. Overcrowding, on the other hand, is a biological notion, meaning excessive density from the standpoint of an organism. Hence, the symptoms of overcrowding are invariably biological.

In confined populations of mice, overcrowding is manifest in a conspicuous increase in aggressive encounters (noncompetitive fights between males). During overcrowding, (1) fecundity diminishes or ceases, and (2) survival of litters (to the age of 3 weeks, when they become self-dependent components of the population) diminishes, or (usually) become nil, which together with (3) an at best unchanged, but usually increased adult mortality either lead to a decline, or keep population size at some constant level, sometimes over many months. Hence, the quantitative dynamics of a confined population of mice rests on the periodical release of mechanisms that inhibit proliferation and promote (or unaffected) adult mortality.

The simplest is the mechanism that abolishes survival of litters. It is released by noncompetitive fights among males, which become eminently numerous during peaks, i.e., during overcrowding period (Brown 1953, Southwick 1955b, Petrusewicz 1960). The increased number of fights among males triggers, as it were, a number of processes: fights between females, lack of care for the young (which are often seen outside the nest), trampling (and crushing) of litters, and finally general cannibalism (Brown 1953, Southwick 1955b, Petrusewicz 1957, 1960, and present observations). Cannibalism is in males rather regular. But normally the females defend the nest, even though they need not do so permanently. Once expelled, a male will not return to the nest soon. But when fights increase, females protect the nests less carefully or not at all. Devouring of the young often starts all of a sudden. Some male begins it and in next to no time all the other mice, females included, throw themselves upon the litters as if at a signal and devour them in a matter of several seconds (Brown 1953, Petrusewicz and Wilska 1959). Once this has happened, the habit continues a long time, often for months on end. So long as it does, the population fails to grow and either declines or remains on a constant level.

More complicated is the mechanism of fecundity failure or diminution. During overcrowding, per capits food intake diminishes as a rule, even though

food is overabundant.This is probably due to mutual interference during feeding, and the excitement caused by continual fighting. Southwick (1955b) reports that at peaks average daily per capita food intake drops from 4.2 g to 2.4 g. In the present investigations the average dropped from 4.0 g to 2.8 g, and even to 2.2 g at exceptionally high peaks. This was average food intake, and individuals low in hierarchy must be presumed to have eaten considerably less. In starved females the oestrous cycle is known not to run the complete course, and the females are not ready to be fertilized. Southwick (1955b) confirmed this mechanism as operative during overcrowding. To these another factor must be added: inadequate copulation. Normally mice mate about 11 seconds. At peak periods, however, only 1.5 seconds (Southwick 1955b), since the female is surrounded by other males that interfere with one another and chase one another of the female. Hence, even in spits of excessive copulation there is often, perhaps even usually, no fertilization. Finally, resorption of the foetus – rather common in rodents – may be presumed to be at peak periode more frequent than at others.

These processes adequately account for the decline or failure of both fecundity and survival of litters, which eventually arrest population growth.

At peak (overcrowding) periods, there is often – though not invariably – a rise in adult mortality. This is rarely due to outright killing. The mice are simply found dead, with bacteriological and virological investigations remaining negative (Petrusewicz 1960). Wijngaarden (1960) noted similar facts in a confined population of *Microtus arvalis*. When adult mortality increases, the population is about to decline, when at a peak, it does not, the population continues on a constant size level, or declines mildly through normal mortality.

The usual pattern is the following. At a peak growth ceases (failure or decline of fertility, cannibalism), and adult mortality rises: the population diminishes. Next, adult mortality returns to normal and growth remains absent: the population continues on a constant low level.

Yet another phenomenon is worth noting: the fairly high permanence and a certain "inertia" of the population structure responsible for growth inhibition. Increased fighting among males lasts short, a few or several days. But infecundity and litter mortality often continue for months on end. All sign of overcrowding has vanished, and the population still fails to grow.

We cannot say why growth is resumed (i.e., why the growth-inhibiting processes cease to operate or become ineffectual). The following is the usual, though not invariable pattern. Fecundity rises, but cannibalism still inhibits growth. It even happens that relatively brief increased fecundity subsides again without having caused growth, or, alternalively, continues even some months but is offset by the devouring of all litters. Eventually, mortality of litters diminishes or becomes nil, and than the population resumes growth.

What mechanism suppresses suckling mortality, chiefly due to cannibalism, it is difficult to say. In three cases growth was reasumed only after the last male had died (females were pregnant). Growth can be imagined as the consequence of the death of some specific male, or female. But on analysis, the data show that suckling survival is restored after the death of an adult as often as it is at precisely the same composition at which all litters were regularly devoured before. On several occasions nests were seen to be defended by females, usually primiparae; but no defence was noted as often, and yet survival of litters increased. All in all we have to admit that we cannot identify the mechanism that disinhibits population growth. All we can say is that such disinhibiting processes exist, since in far the most cases the decline that follows a peak does not wipe out the population; it become arrested, and growth is resumed (in *Z* and *B* cages).

Resumed growth means that inhibition has been abolished. And the inhibition – as we have sought to demonstrate – was conditioned by some specific relations between the members of a population, i.e., by the population's structure. Hence, removal of the inhibition may be supposed to have connexion with a change in population structure. This surmise may be made even though we do not know the exact mechanisms responsible for disinhibition.

Some light is shed on the process by the experiments already described, and by the studies on growth induced by a change of cage (Petrusewicz 1957). In either the population was subjected to an experimental shock (change of cage, and removal or addition for a time of a few individuals). In consequence or the shock, the growth-inhibiting relations that have become established between population members vanish; the natural upward trend is resumed and the population grows. But what essentially the mechanis n is that removes the inhibition we cannot say; our only surmise is that the cause varies between cases.

To sum up: underlying the mechanism of growth regulation in confined populations of mice is a permanent capacity for multiplication and its periodical inhibition (at population peaks) by partial or complete failure of fecundity and increased suckling mortality. The inhibitory factors, such as undernourishment (failure of oestrus), probably resorption, defective copulation, and devouring of sucklings, are released by more frequent noncompetitive fights among males. These inhibitory factors may be operative over an appreciable period of time. Usually fecundity is restored first, and only then subside cannibalism and mortality of suckings. The mechanisms responsible for fecundity restoration and suppression of suckling mortality remain obscure

The author is sincerely grateful to M.sc. T. Wierzbowska of the Institute of Ecology for helping him with the statistical analysis.

REFERENCES

1. Andrewartha, H.G. 1961 – Introduction to the study of animal population – Chicago, pp. XVII + 281.
2. Brown, R.Z. 1953 – Social behaviour, reproduction, and population changes in the House mouse (Mus musculus L.) – Ecol. Mon. 23, pp. 217–240.
3. Calhoun, J.B. 1936 – A comparative study of the social behaviour of two inbred strains of the House Mice – Ecol. Mon. 26, pp. 81–103.
4. Evans, F.C. 1949 – A population study of house mice following a period of local abundance – Jour. Mamm. 30; pp. 351–363.
5. Naumov, N.P. 1953 – Ekologia zivotnych – Moskva, pp. 532.
6. Petrusewicz, K. 1957 – Investigation of experimentaly induced population growth – Ekol. Pol. A, 5, pp. 281–309.
7. Petrusewicz, K., Wilska, T. 1959 – Investigation of the influence of inter-population relations on the result of fights between male mice – Ekol. Pol. A, 7, pp. 357–390.
8. Petrusewicz, K. 1960 – Some regularities in male and female numerical dynamic in mice population – Acta theriol. 4, pp. 103–137.
9. Petrusewicz, K. 1960 – An increase in mice population induced by disturbance of the ecological structure of the population – Bull. Acad. pol. Sci., 8, pp. 301–304.
10. Southwick, C.H. 1955a – The population dynamics of confined house mice supplied with unlimited food – Ecology 36, pp. 212–224.
11. Southwick, C.H. 1955b – Regulatory mechanisms of house mouse populations: social behaviour affecting litter survival – Ecology 36, pp. 627–634.
12. Southwick, C.H. 1958 – Population characteristics of house mice living in english corn ricks: density relationships – Proc. zool. Soc., London 131, pp. 163–175.
13. Strecker, R.L. and Emlen, J.T. 1953 – Regulatory mechanisms in house-mouse populations: the effect of limited food supply on a confined population – Ecology 34, pp. 375–386.
14. Tupikova, N.V. 1947 – Ekologija domovoj myši srednej polosy SSSR – Materiały po gryzunam, 2, Fauna i ekologija gryzunow, red. A.N. Formozov – Moskva, pp. 5–67.
15. Wijngaarden, A. van 1960 – The population dynamic of four confined population of the continental vole Microtus arvalis – Meded. R.J.V.O.N., 84.

WZROST POPULACJI INDUKOWANY PRZEZ ZAKŁÓCENIE EKOLOGICZNEJ STRUKTURY POPULACJI

Streszczenie

W jednej z poprzednich prac (Petrusewicz 1957) wykazano, że przeniesienie populacji białych laboratoryjnych myszy do innej klatki (większej, takiej samej lub nawet mniejszej) wywoływało wzrost liczebności populacji. Wyrażono też przypuszczenie, że procesem odpowiedzialnym jest tu zaburzenie ekologicznej struktury populacji. W prezentowanej obecnie pracy dokonano próby zakłócenia struktury populacji przez dodawanie lub wyjmowanie na pewien czas kilku osobników.

Myszy były hodowane w klatkach typu *Z* (80×80 cm) typu *B* (160×80×80 cm) oraz *A* (37×15 cm). Do każdej klatki wkładano pewną ilość myszy (najczęściej 3 ♂♂ i 8 ♀♀) i pozwalano im mnożyć się swobodnie. Woda i żywność były zawsze w nadmiarze. Ogółem hodowano 80 populacji typu *Z*, które żyły łącznie 2225 miesięcy, 40 populacji *A*

379 miesięcy i 7 populacji *B* 284 miesięce; z tymi populacjami wykonano 264 razy (tab. I) następujący eksperyment:

Wariant 1. Do klatki z populacją wkładano kilka (4–7) dziewiczych samic. Po upływie ok. 1 tygodnia te obce samice wyjmowano (ciąża myszy trwa 20 dni).

Wariant 2. Z klatki wyjmowano kilka 4–7 samic (nie wysoko ciężarnych i niekarmiących) i 2–10 samców. Wyjęte samce i samice trzymano rozdzielnie. Po upływie ok. tygodnia wyjęte myszy zwracano do ich populacji. Eksperyment był więc prowadzony w ten sposób, że do wolnorozmnażającej się i samoregulującej swoją liczebność populacji nigdy nie dodawano nowej „krwi" z zewnątrz.

Jako kontrolę zanalizowano liczebność, prawdopodobieństwo zaistnienia wzrostu populacji, płodność i przeżywalność młodych po 476 losowo wybranych momentach w życiu populacji. Czas wykonania, połączenie w serie itp. daje tab. I. W trakcie eksperymentu (po dodaniu obcych lub wyjęciu własnych myszy) lub po zakończeniu eksperymentalnych wpływów na populację (po zwróceniu swoich lub wyjęciu obcych myszy) dał się zaobserwować lekki i krótkotrwały (1–5 dni) wzrost liczby walk, oraz spadek liczebności populacji (w 80 przypadkach na 264 eksperymentów). Po czym następował zwykle dość gwałtowny wzrost liczebności populacji (przykłady – fig. 1).

Do analizy obliczono:

1. Liczbę odpowiedzi pozytywnych – wzrost po eksperymencie/kontroli nastąpił, przy czym zaczął się nie później niż 10 tygodni po eksperymencie/kontroli.

2. Wielkość populacji (liczba osobników) w momencie

a) eksperymentu/kontroli, b) początku wzrostu, c) szczycie będącym „odpowiedzią pozytywną".

3. Wielkość wzrostu (procent wzrostu od punktu wyjściowego);

4. Płodność przed (*F*) i po (*F'*) eksperymencie/kontroli tzn. liczbę zrodzonych w ciągu miesiąca przed i po;

5. Przeżywalność młodych przed (*S*) i po (*S'*) eksperymencie/kontroli (procent urodzonych, które dożyły 3 tygodni w ciągu 2-ch miesięcy przed i po eksperymencie/kontroli).

Z analizy obliczonych wielkości, można wyciągnąć następujące wnioski:

1. Populacje w momentach gdy dokonywano eksperymentów nie różniły się od przeciętnego stanu jaki stwierdzono dla momentów losowanych jako kontrolne. Za tą tezą przemawiają następujące stwierdzenia:

a. Wielkość populacji w momentach wykonywania eksperymentu i wielkość populacji uznanych za kontrolne nie różniły się (33,1, tab. V, fig. 4),

b. Liczby procentowe eksperymentów i momentów kontrolnych przypadających na różne fazy cyklu populacyjnego wykazują zadziwiającą zgodność (32.4, tab. IV).

c. Wielkość płodności i przeżywalności przed eksperymentem nie różniła się od analogicznych wielkości przed i po kontroli (por. 34.5, tab. VI i VII).

2. W wyniku szoku eksperymentalnego prawdopodobieństwo zaistnienia wzrostu liczebności populacji rośnie w sposób istotny. Za tą tezą przemawiają następujące stwierdzenia:

a. Rozkłady czasokresów od momentów eksperymentu i kontroli do początku wzrostu są różne; po eksperymencie mamy wyraźne nagromadzenie liczby początków wzrostów (do 10 tygodni), czego nie ma po kontroli (2, tab. II, fig. 2);

b. W ogromnej większości serii procentowe liczby powtórzeń, gdy wzrost nastąpił, są większe od liczby powtórzeń gdy wzrostu nie było (tzn. procent pozytywnych odpowiedzi jest wyższy niż 50% – tab. III, fig. 3);

c. We wszystkich seriach procentowe liczby odpowiedzi pozytywnych są po eksperymencie większe niż po kontroli (fig. 3); wielkości te są statystycznie istotne dla całości materiału i dla 10 z 14 serii;

d. Liczba przypadków (w procentach) gdy wzrost był poprzedzony pewnym spadkiem jest wyraźnie większa po eksperymencie niż po kontroli (różnica statystycznie istotna), co wskazywałoby na zmianę biegu krzywej populacyjnej pod wpływem szoku eksperymentalnego (tab. III);

e. Częstość wzrostów przypadających na poszczególne fazy jest inna w eksperymencie niż w kontroli; w eksperymencie wyraźnie wyższy jest procent wzrostów w szczycie i w spadku (tzn. w fazach, w których małe jest prawdopodobieństwo spontanicznego wzrostu), niż prawdopodobieństwo samorzutnego wzrostu w tychże fazach (tab. IV);

f. Wzrost poeksperymentalny mógł następować przy istotnie większej liczebności populacji niż wzrost samorzutny (fig. 5, tab. V);

g. Jest prawdopodobne, że szczyt poeksperymentalny jest wyższy niż spontaniczny (jest on większy w większości serii, różnica jednak nie jest statystycznie istotna) — fig. 6, tab. V;

h. Płodność i przeżywalność jest większa po eksperymencie niż przed eksperymentem i niż przed i po punktach kontrolnych (tab. VI i VII, fig. 7 i 8). Różnice te są statystycznie istotne zarówno odnośnie do częstości przypadków jak i dla wartości średnich.

3. Wyniki eksperymentów nie są jednorodne; zależą od fazy życia populacji na jaką przypada eksperyment (punkt kontrolny), oraz od ogólnej predyspozycji populacji do wzrostu. W okresie gdy rezultaty (procenty odpowiedzi pozytywnych) serii kontrolnych były jednorodne, również jednorodne były rezultaty serii eksperymentalnych. W czasie gdy prawdopodobieństwo wzrostu spontanicznego maleje, maleje również liczba wzrostów indukowanych eksperymentem, pozostaje jednak wciąż wyższa niż liczba wzrostów spontanicznych (fig. 3, tab. III).

Prawdopodobne wyjaśnienie zaobserwowanych prawidłowości może być następujące: w każdej populacji wytwarza się określona i swoista dla niej struktura populacyjna, tzn. określone stosunki między osobnikami, hierarchia socjalna, sposób budowania gniazda, sposób i charakter opieki nad młodymi, stosunki terytorialne, liczba i nasilenie walk między myszami, a w szczególności między samcami, presja kopulacyjna itp. Po osiągnięciu pewnego (różnego przy różnych strukturach populacyjnych) zagęszczenia występują objawy przegęszczenia: walki niekonkurencyjne samców, za nimi walki samic, brak opieki (lub niedostateczna opieka) nad młodymi, zmniejszenie spożycia na głowę (mimo nadmiaru pokarmu) i związany z tym nieprawidłowy cykl płciowy samic, prawdopodobnie zwiększona resorbcja płodów, wreszcie kanibalizm młodych. W rezultacie tego wszystkiego płodność i przeżywalność maleją lub zanikają, populacja przestaje wzrastać, utrzymuje się na poziomie lub — jeśli zwiększa się śmiertelność dorosłych — maleje. Jednym słowem wytwarzają się między osobnikami populacji takie stosunki i zależności (struktura ekologiczna populacji) które przeciwdziałają wzrostowi i sprzyjają spadkowi liczebności populacji.

Struktura taka wskazuje pewien bezwład, może trwać nawet przez szereg miesięcy, mimo iż walki samców — proces wyzwalający wszystkie dalsze, hamujące procesy — już dawno ustały. Struktura taka trwa nieraz do czasu gdy populacja osiągnie bardzo niski poziom liczebności (nieraz poniżej 10 osobników, zwykle około 15 osobników). Populacja nie wzrasta, mimo że nie ma przegęszczenia, ani jego objawów. Przez bodziec jakim jest dodanie lub odjęcie na parę dni kilku osobników obcych, narusza się istniejącą strukturę populacji, po czym myszy zachowują się mniej więcej jak nowo założona populacja. W nowo założonej populacji obserwujemy zaś zawsze tendencje do wzrostu.

Mechanizm regulacji w zamkniętych populacjach myszy polegałby więc na stałej potencjalnej zdolności do rozmnażania się oraz na okresowo uruchamianych procesach hamujących rozród (płodność młodych i przeżywalność). Procesem wyzwalającym hamowanie rozrodu są głównie niekonkurencyjne walki samców. Procesu wygaszającego hamowanie rozrodu w normalnych warunkach nie umiemy jeszcze wskazać; wiadomo jedynie, że w ogromnej większości wypadków zaczynają wzrastać – tzn. procesy hamujące ustają.

ADDRESS OF THE AUTHOR:
Prof. Dr Kazimierz Petrusewicz,
Institute of Ecology, Warszawa,
Nowy Swiat 72.