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Some notes on ecological theory.

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Guido Grandi]

In an interesting recent paper, H. KLOMP ('58) discusses the theories of host-parasite interactions. « Two important theories of host-parasite interaction » he says, « have been stated, those of THOMPSON (1922-24) and of NICHOLSON ».

These two theories according to KLOMP differ principally in that in NICHOLSON'S theory the reproductive rate of the parasite is related to and dependent on the density of the host while in THOMPSON'S theory it is not, being constant and limited only by the capacity to produce offspring. This description of the view of THOMPSON has also been published by G. C. VARLEY and R. L. EDWARDS ('57).

I propose in this paper to discuss these statements with particular reference to the remarks of KLOMP. To simplify the discussion, the theories will be referred to as those of NICHOLSON and THOMPSON.

In general, KLOMP'S criticism is that neither of the theories provides a basis for a « host-parasite system in fluctuating balance » which may be interpreted as the persistence in varying numbers of both populations in nature.

Nicholson's theory, in so far as it relates the reproductive rate of the parasite to the density of the host, provides a better starting point than the theory of Thompson. « In the former » says KLOMP, « any disturbance of the steady state by a changing specific or non-specific mortality factor included in the system, gives rise to regular oscillations about the steady state. In the latter every disturbance of the steady state by a specific or non-specific factor leads to unlimited increase or to extermination ». However, KLOMP does not give unqualified approval to Nicholson's theory. He suggests that « Nicholson's assumptions » may be « realized in nature ». Nevertheless he says, that the regular oscillations, « inherent in Nicholson's theory of parasite-host relationships, cannot exist under actual field conditions because of the changing environmental factors working within the system » and he states that « the main objection which may be levelled against his (NICHOLSON'S) theory is the ever-increasing amplitude of the oscillations, which has never been observed in nature ». He rejects a mechanism for the damping

of the oscillations suggested by NICHOLSON, saying that « In nature such a situation can not be ».

In another place, referring to Nicholson's oscillations, KLOMP says, « that according to the theory, an interaction with increased oscillation always leads to a peak, followed by a crash to a very low population density ». He mentions Nicholson's claim that « when such a situation has been reached the densities are maintained much below their steady values, the animals being distributed in small groups ». He objects « that under field conditions many host-parasite systems are not characterized by these phenomena » but he fails apparently, to note two points: first, that the population values in the « crash » fall below unity which may be interpreted as extermination, the final outcome being in fact as in the 1924 formulation by THOMPSON and depending on the fact that e^{-x} is never equal to zero; second, that the distribution in small groups is not a deduction from the theory but an extra-theoretical intercalation, similar to that made by THOMPSON when he said that annihilation, though produced by the theory is not, in fact to be expected. Klomp's misunderstanding is apparently shared by VARLEY and EDWARDS.

Klomp's view is that the theories of NICHOLSON and THOMPSON are not antagonistic but are applicable at different densities of the host. Both fail, however, since they do not account for the « fluctuating balance » observed in nature. I think it should be added that NICHOLSON has always regarded the «fluctuating balance » as the desirable theoretical result and has been inclined to suggest that his theory produces it. A departure from the steady state, said NICHOLSON and BAILEY, sets up a reaction « tending to cause a return to this density ». In fact, however, as KLOMP points out, and as I have already said, what happens after one or more oscillations, is a fall of host and parasite populations below unity. Figure 7B in NICHOLSON'S 1933 paper depicts this in a single cycle, at the end of which the host population, according to my calculation becomes 0.0162.

In Thompson's theory, according to KLOMP, a theoretical « steady state » or balance, is possible only with the special relation

$$\frac{p}{n} = \frac{h - s}{s}$$

where n = population of the host, p = population of the parasite, h = reproductive rate of the host, s = reproductive rate of the parasite with the proviso that « the number of eggs laid per parasite is extremely small » and in fact equal only to 1. Such a situation says KLOMP, « may be rejected as impossible because there are no parasites with so low a fecundity ». Furthermore, « the existence of a host attended by a single specific parasite and uninfluenced by any other mortality factor, as assumed by THOMPSON, is very unlikely to occur in nature ». This same objection can of course be made to Nicholson's initial formula. The existence of a parasite depositing only 1

egg is unlikely, but if a parasite population is stable, the effective reproductive rate cannot in the long run be greater than 1 in relation to the initial value. In the 1923 paper cited by KLOMP, a reduction of the reproductive rate to unity was given as a possible result of the attack of hyperparasites so that the process is quite different from the deposition of a single egg. (l.c. p. 11).

In some interesting sections of his paper, KLOMP has enquired whether the intervention of other mortality factors with an effect averaging around some particular level, would enable the stable condition determined by the relationship given above, to persist.

If there is a non-specific mortality (i.c. affecting host and parasite equally), operating at 95%, the effective reproductive rates, in a case where $n = 40$, $p = 10$, $h = 25$, $s = 20$, only 5% of the progeny of host and parasite survive, so that the next generation begins, as before with $n = 40$, $p = 10$. These with $h = 25$, $s = 20$, produce, as before, 800 hosts and 200 parasites. If the mortality then falls from 95% to 80% we get 4000 hosts and 800 parasites, of which 160 hosts and 40 parasites survive. If the mortality increases from 95% to 99%, we eventually get 8 hosts and 2 parasites. Thus stability is maintained but at a higher level in the first case and at a lower level in the second case. This point had already been noted by THOMPSON in a paper published in 1930 (1930, p. 60). « Suppose », said THOMPSON, « we have a population of one hundred female individuals per unit of area living in a region where the physical conditions are such that of the six offspring each individual can produce only one pair survive in each generation. If the intensity of the physical factors suddenly decreases, so that all the six offspring survive in every generation, in four generations there will be 64,800 female individuals per unit of area. If then, the physical factor returns to its former intensity, so that, as before, four out of six offspring are killed, the population will again become stable, but at a higher level than before. Similarly, if the population was depressed below the level of one hundred individuals per unit of area by an increase in the intensity of the physical factor, it would remain stabilized at the lower level in spite of the return to normal conditions.

The basic principle simply is, that the equation $\frac{p}{n} = \frac{h - s}{s}$ (where $s = 1$) can be satisfied provided the ratio between p and n remains equal *to* $(h - 1)$. In the other examples given by KLOMP, this ratio changes and the sequence of events therefore proceeds according to one or other of the basic equations given by THOMPSON in the 1922 and 1923 papers. Once the basic relationship has been altered stability can no longer exist at the same level and if the ratio $\frac{p}{n}$ has also been altered it cannot exist at any level. In the first example given by KLOMP he postulates a specific mortality of 75% acting on the host at the end of the first generation (where we had, $n = 40$, $p = 10$, $s = 1$ and $h = 5$ so that the effective reproductive rate of the host becomes 1.25, and therefore we get stability). KLOMP increases the host mortality in the pro-

geny to 80%, so that the effective reproductive rate of the host becomes 1. The number of hosts at the beginning of the next generation therefore falls to 30, with a reproductive rate restored to 1.25 as before. The position therefore corresponds to the equation (11) in THOMPSON'S 3d 1922 paper and we see that the host is exterminated in 6.21 generations, because the ratio of p to n is high. In the second example of this section, starting with the same conditions, KLOMP reduces the mortality at the end of the first generation from 75% to 70% so that the effective reproductive rate of the host is increased to 1.5. We therefore begin the next generation with 50 hosts and 10 parasites. This, again, corresponds to the conditions in equation (11) but extermination is then seen to be impossible. In fact, if we have $s < h$, the condition for a real finite value of t is

$$\frac{p}{n} > \frac{h - s}{s}$$

whereas in Klomp's example we have $\frac{p}{n} = 0.2$ and $\frac{h - s}{s} = 0.25$. Klomp's final example is with a non-specific mortality operating at 95% on the average and fluctuating at 92%, 97%, 93% and 98% in successive generations. He gives, $n = 40$, $p = 10$, $h = 25$ and $s = 20$. The equations in the THOMPSON papers show, that in the particular case where $\frac{p}{n} = \frac{h - s}{s}$, with $s > 1$ which is the position defined by KLOMP, the percentage of parasitism will be constant, but host and parasite will increase indefinitely. With 92% mortality KLOMP obtains reproductive rates of $h = 2$, $s = 1.6$; the second generation then starts with 1,280 hosts and 320 parasites, whose reproductive rates become $h = 0.75$ and $s = 0.6$ so that we get 960 hosts and 192 parasites. The next generation begins with 768 hosts and 192 parasites, and a 93% mortality reduces their reproductive rates to 1.75 and 1.4, so that the progeny are 1,344 hosts and 268 parasites. Finally we have 1,076 hosts and 268 parasites and a 98% mortality reduces their reproductive rates to 0.5 and 0.4 so that we end with 538 hosts and 107 parasites. There is therefore in the end, a decrease in hosts and parasites, though the percentage of parasitism does not change, (because the mortality is indiscriminate). The values at the beginning of each generation satisfy the equation $\frac{p}{n} = \frac{h - s}{s}$ so that the populations will increase (h=1.75, s=1.4) or decrease (h=0.75, s=0.6; h=0.5, s=0.4) though the parasite will never exterminate the host. It may be noted that in the final situation where n=430 and p=108, with effective reproductive rates restored to h=1.25 and s=1, the values satisfy the equation

$$\frac{p}{n} = \frac{h - s}{s}$$

where $s = 1$, so that stability has been restored, though at $n = 430$, $p = 108$ instead of $n = 800$, $p = 200$.

Nevertheless though the relation

$$\frac{p}{n} = \frac{h - s}{s}$$

with $s = 1$, not precisely « unstable » as KLOMP says, yet it seems too delicate and easily *upset* to explain, even theoretically, the co-existence of host and parasite in nature.

However, Klomp's review of the work of THOMPSON like the citation of VARLEY and EDWARDS is inadequate because he confines himself to these papers, though references to other papers occur in articles by MILNE (1957) and SOLOMON (1949) actually cited by KLOMP.

In fact in the 1924 paper on the effect of random distribution THOMPSON gives equations from which another stable system can be derived. The equations are

$$n_{t+1} = n_t\, he^{-\mu_t}, \qquad \text{where } \mu_t = \frac{P_t s}{n_t h}$$

$$P_{t+1} = ps^{t+1}.$$

The equations for stability are

$$p = nh \log h$$

$$s = 1$$

In this case it was assumed that though hosts may contain more than 1 parasite, all the parasites it contains emerge. A feature of this system is that random distribution leads, with higher host densities, to a waste of killing power by the parasite; for example, if a parasite deposits 10 eggs in 100 hosts 9.5 hosts are killed, if it deposits 100 eggs in 100 hosts, the kill is only 63.2. In a paper published in 1929, it was assumed that in cases of random distribution, only 1 parasite issues from each host attacked. Thus random distribution leads to a fall in reproductive efficiency. It is therefore misleading to say, as KLOMP does, that according to THOMPSON, the parasite lays a fixed number of eggs, in the sense that the power of increase is constant. The equations in this case are,

$$n_{t+1} = n_t\, he^{-\mu_t}$$

$$p_{t+1} = n_t\, h\,(1 - e^{-\mu_t})$$

and the equations for stability are

$$s = \frac{h \log h}{h - 1}$$

$$p = n\,(h - 1)\,.$$

A stable system would exist if we have $n = 50$, $h = 10$, $p = 450$, $s = 2.56$. Applying Klomp's method and supposing that at the end of each generation there is an indiscriminate mortality of 20%, 60% and 40%, averaging

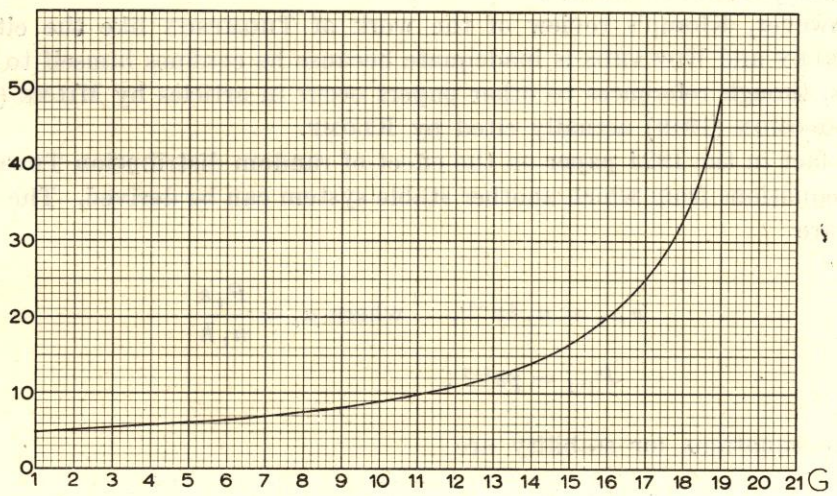

FIG. I.

Curve of % of parasitism given by the equations, $n_t = n(hi)^t$, $p_t = nh^t i^{t-1} a$, where $n = 40$, $p = 2$, $h = 2 = s$, $i = 0.5 = a$. Ordinates = % parasitism, abcissae = generations.

40%, with survivals of.8, .4 and .6, we find, as in his example, that the population ratios remain constant at $n = 1$, $p = 9$. The values of n are, 40, 16, 9.8 and of p, 360, 144 and 85.8; at the end of this series of generations, we have $n = 10.4$ and $p = 87.5$. In every case we have an equation of stability. In the original system with the final values of $n = 10.4$, $h = 10$, $p = 87.5$, $s = 2.56$, the system would not be stable (see 1939, p. 367); since we have $s < h$ and $\frac{p}{n} > \frac{h - s}{s}$, extermination would occur. It appears therefore that in the system we are now discussing stability would recur, though at varying values; that is, the values at the beginning of each generation would be reproduced in that generation. The remarks of KLOMP do not therefore apply perfectly to this theoretical development.

However, this system does not provide a satisfactory solution of the problem of « fluctuating balance », considered by KLOMP as the desirable objective.

In another (1929) paper by THOMPSON, not cited by KLOMP an approach to a solution may be found. This paper concerns the part played by parasites in the control of insects living in « protected situations ». This solution was suggested in the 1923 paper (section V, p. 11). The situation studied in the 1929 paper was that where a proportion of the host population is inaccessible to the parasite. The first examples given were those of the European Corn Borer, the Codling Moth and the Mediterranean Fruit-Fly; but in the last paragraph it was pointed out that « in order to be inaccessible to an enemy, the host need not necessarily live in what are ordinarily called protected situations, such as tunnels in the substance of plants or beneath the surface of the soil. The mere fact that it can live in zones in which the parasite and predator cannot survive, or to which it is not attracted, or maybe present at times when its enemies are not in a stage when they can attack it, consistutes an equally efficacious protection ». « Inaccessibility » was thus very broadly defined and as so defined, includes the inaccessibility resulting from a fall in host density; since this is precisely a case in which a given host is in a zone to which its parasite is not attracted.

It was shown in the paper cited, that if the number of hosts $= n$, the proportion of inaccessible hosts $= i$, and the proportion of accessible hosts $= a$: $(i + a = 1)$, the hosts and parasites issuing in the generation are

$$n_t = n\, h^t\, i^t$$

$$p_t = n\, h^t\, i^{t-1}\, a\,.$$

Up to the point when the parasite attacks all the available hosts, the cause of events will be as outlined in the original (1922) system. After this point the conditions exemplified in the formula will take effect. It should be noted that in this presentation, one parasite is assumed to issue from each host attacked, but random distribution is not expressly postulated.

It is clear that when we have $hi = 1$, $n(hi)^t = n$ so that the population is stabilized. If we have $hi > 1$ host and parasite will continue to increase. If we have $hi < 1$, then $n(hi)^t$ will tend to 0 as the value of t increases so that extermination of host and parasite will result.

In figure 1 a typical curve of the percentage of parasitism is shown, where the initial values are $n = 40$, $p = 2$, the reproductive rates of host and the same and 2, the proportion of inaccessible and accessible hosts, also the same, and $= 0.5$. Since $hi = 1$ and $a = 0.5$, the populations of host and parasite will become stabilized when the parasitism reaches 50%. The generation t in which this will occur is given by the equation

$$\alpha = \frac{100\, p}{n - p\,(t-1)}$$

which, when $\alpha = 50$ gives $t = 19$. The numbers of hosts and parasites at the end of this generation are given by the equations,

$$n_t = h^t\{n - p\,(t-1)\}$$

$$p_t = ps^t$$

and are found to be

$$n = 2{,}097.152$$

$$p = 1{,}048.576$$

so that the parasitism has reached 50% .

The curve of this case from the origin at $n = 40, p = 2$ is shown in figure 1. Judging from what we know, it is characteristic ot the events which sometimes follow the introduction of a small parasite population into a large host population. If the host population continued to increase as it has done in the case of introduced pests having a large suitable area available, the populations might increase according to the above equations for a considerable time.

In fact, the pest population may rise to a peak where it is overtaken by the parasite and suddenly suffers a catastrophic reduction, followed by that of the parasite — as in the first equations published by THOMPSON. Eventually, however the pest may become stabilized and at that level a certain proportion will be accessible, a certain proportion inaccessible to the parasite. It is conceivable that these proportions may change and the parasitism may then rise or fall. Mortality factors may also intervene. For example, we might have an indiscriminate mortality of 20% where the values were $n = 50$, $p = 25$, $h = 2 = s$ and $i = 0.5 = a$. Other values remaining the same, the host population would stabilize at 40 and the parasite population at 25. If the parasite population alone suffered a decline from 25 to 15, while the host merely maintained its position we would have a sequence of events as follows:

$G1$	$N1 = 50 \times 2 = 100$ of which	50 accessible
	$P1 = 15 \times 2 =$	30
	Hosts surviving	$20 + 50 = 70$
$G2$	$N2 = 70 \times 2 = 140$ of which	70 accessible
	$P2 = 30 \times 2 =$	60
	Hosts surviving	$10 + 70 = 80$

$G3$ $N3 = 80 \times 2 = 160$ of which 80 accessible

$P3 = 60 \times 2 =$ 120 reduced to 80 by superparasitism

$G4$ $N4 = 80 \times 2 = 160$ of which 80 accessible

$P4 = 80 \times 2 =$ 160 reduced to 80 by superparasitism

and thus in a few generations stability would be restored.

If the host population suffered a decline from 50 to 30, other factors remaining the same, the sequence of events would be

$G1$ $N1 = 30 \times 2 =$ 60 of which 30 accessible

$P1 = 25 \times 2 =$ 50 reduced to 30 by superparasitism

$N2 = 30 \times 2 =$ 60 of which 30 accessible

$P2 = 30 \times 2 =$ 60 reduced to 30 by superparasitism

so that stability would again be restored.

If a parasite colony is introduced into a large host population, which has already become stabilized, its progress might be represented by the equation

$$P_t = ps^t$$

to the point where it has come to equal the accessible population. It is possible, however, as THOMPSON explicitly recognized on several occasions, that the factor i might be increased by the rarefaction of the host population produced by the parasite itself. If as a result hi became $= 1$, then the host would increase and this would eventually produce more individuals accessible to the parasite so the populations would again, eventually, level off.

It is important to note that in this system, the populations during the greater part of their existence, *may not be in a true balance.* They may be moving toward a balance they never reach or they may be receding from it. This theoretical conclusion agrees with the views of MILNE ('57) and to some extent with those of SOLOMON ('57). Theoretically, the movement toward balance depends on the possibility that we have $hi = 1$ rather than $hi > 1$ or $hi < 1$. The condition $hi > 1$ means an « outbreak » and outbreaks are exceptional. The condition $hi < 1$ means extermination and though extermination or near-extermination must occasionally occur and perhaps more often than we realize, it seems also to be exceptional at least for an entire species. The intrinsic limitations of specific beings, the diversity and fragmentation of habitats and the change in the character of habitats from moment to moment, working against the natural reproductive power of the organisms tend in general, it seems, to bring $hi = 1$. This principle has therefore a broader application than to the interrelation of a host and its parasite.

A rather more elegant formulation of the ideas developed above was published by THOMPSON in 1939. There it was assumed that in the situation where a proportion of the host population is inaccessible, the parasite distributes its progeny among the accessible hosts at random. Using the same symbols as before, this leads to the equations

$$N_{t+1} = N_t h\left(i + ae^{-\frac{p_t s_t}{n_t ha}}\right)$$

$$P_{t+1} = N_t ha\left(1 - e^{-\frac{p_t s}{n_t ha}}\right)$$

Commenting on these equations, (1939, p. 372), THOMPSON said. If $h = 1$, N_{t+1} and P_{t+1}, tend asymptotically to 0; if we have $h > 1$, N_{t+1} tends asymptotically to the value of $N_t hi$ while P_{t+1} tends to the value of $N_t ha$; if we have $hi =$ or > 1 then the value of N_t increases. If we have

$$s = \frac{nha}{p} \log\left(\frac{ah}{1 - hi}\right)$$

$$p = nhae^{-\frac{ps}{nha}}$$

and $hi < 1$ the populations remain stable. If hi were $>$ or $= 1$ then the population of inaccessible hosts would be $> n$ or $= h$. Even if it were only n it is clear that the host population could not remain stable because the accessible host population would always have a positive value, unless the index of e were indefinitely large, which would require an infinitely large parasite population.

The investigation of this case is more difficult than that of the preceding system.

If we have $n = 10$, $p = 8$, $h = 4$, $s = 11$ (approx.), $i = 0.2$, $a = 0.8$, so that $s > h$, while $hi = 0.8$ and is, therefore < 1, the equations give

$$N2 = 10.04$$

$$P2 = 8.03$$

so that the populations are immediately stable. However, if we have $hi = 1$ and $h > 1$ with $s =$ or $> h$ the populations rise, as stated above, *but eventually become stable.* The point at which stability is reached depends on the constants. If we have $n = 5$, $p = 1$, $h = s = 10$, $i = 0.1$, $a = 0.9$ the populations become stable at a high level in 15 generations with a parasitism of 90% which, of course is $= a$. If we have $n = 10$, $p = 9$, $h = s = 10$, $i = 0.1$, $a = 0.9$, the populations become stable at a low level in six generations, with the same parasitism. If we have $hi = 1$, with $s = h$, the parasitism remains constant

but the populations rise; if we have $hi > 1$ with $h > s$, both populations rise but parasitism falls; if we have $hi < 1$, $h = s$, $h > 1$ the populations steadily decrease so that both eventually disappear. If we have $s < h$, even if we have $hi = 1$ the populations both rise, but parasitism falls.

Thus the populations tend to stability when we have, $hi = 1$, $h > 1$, and $s =$ or $> h$. If $h = 1$ only, the equations for stability become insoluble. It is clear that stability cannot then occur since the value on n constantly falls.

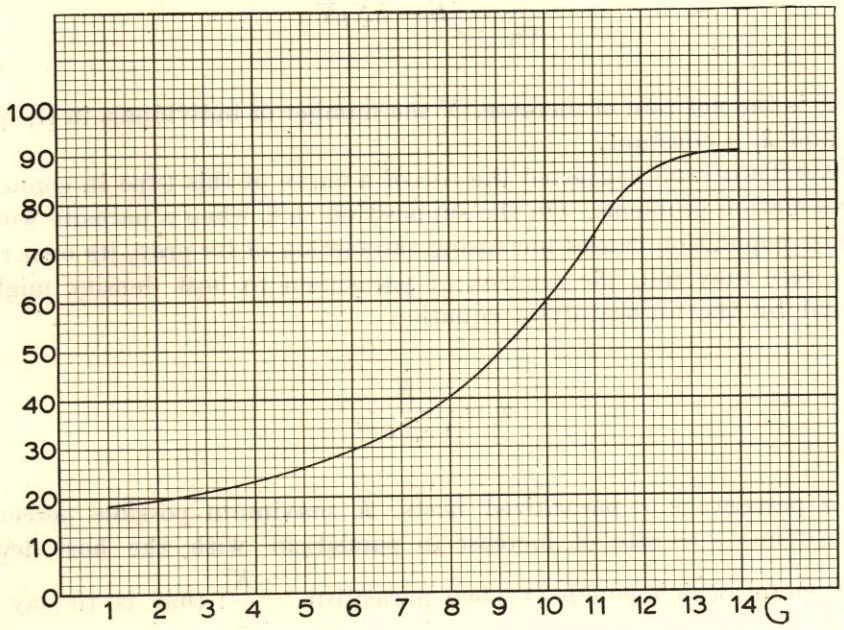

FIG. II.

Curve of % of parasitism given by the equations, $n_{t+1} = n_t h\left(i + ae^{-\frac{p_t s}{n_t h a}}\right)$, $p_{t+1} = n_t h a\left(I - e^{-\frac{p_t s}{n_t h a}}\right)$, where $n = 5$, $p = 1$, $h = 10 = s$, $1 = 0.1$, $a = 0.9$.

These conditions seem not intrinsically unlikely. If we have $h = 1$, then the inroads of a mortality factor might eventually release more favourable situations, so that h would return to a value greater than 1. If the reproductive rate of the parasite is less than that of the host, and the host increases, then the accessible hosts will become more numerous and the value of s will rise.

Figure 2 shows the graph of parasitism in a case where $n = 5$, $p = 1$, $h = s = 10$, $i = 0.1$ and $a = 0.9$. Parasitism levels off at 90% but more gradually than in the preceding system (1).

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(1) Host and parasite curves in a case of this type were published in 1947 (see Bibliography).

It is clear from what has preceded, that in this system, increases or decreases in the values of n and p, whether specific or indiscriminate, will not prevent the trend toward stability.

It will be noted that the curves for percentage of parasitism, in both of the preceding systems recall the sigmoid curve so commonly used in population theory and developed as an integral of the differential equation

$$\frac{dN}{dt} = (E - \lambda N)N$$

where E is the net rate of increase, N the number of individuals in the population and λ, a constant.

In 1930 THOMPSON suggested the use of a curve of this type in connection with population problems. On the supposition that when a parasite working on a host population causes increasing rarefaction of its prey, its own reproductive rate may fall, the relation of parasitism to host density might be expressed by the autocatalytic equation

$$\alpha = \frac{K}{1 + e^{-rt}}$$

where α percentage of parasitized hosts, K maximum possible percentage of parasitism, r = rate of increase in parasitism with the host density, and t a factor which equals O when parasitism $= \frac{K}{2}$: that is to say with a maximum possible parasitism of 100%, would $= 0$ when $\alpha = 50$, in which case the equation becomes

$$\alpha = \frac{100}{1 + e^{-t}} = 50$$

The use of this equation need not be restricted to the relation of a parasite to its host; it can just as easily be extended the general problem of the effect of variation in accessible or inaccessible situations on the mortality of an organism.

The first step is the construction of the curve for α. Taking the value of K as 100 and putting $r = 1$, we see that at $t = 0$, $\alpha = 50$ and this is the point from which calculations are made, giving t positive values to the right of $t = 0$ and negative to its left (Fig. 3).

If the value of K is set below 100 for example, at $K = 50$ the value of α at $t = 0$ will be 25% and on its right hand section, the curve will tend asymptotically to this value.

Within the limits of the general mathematical law of the curve the relationship between density and mortality can be varied. The general law accepted is, that at both low and high levels, variations in density have slight effects while at intermediate levels, the effects are well marked. The parasite would not die out completely at low densities, nor would it exterminate the host completely at high densities. One might say also that a free-living animal would always find some favourable environments even if they are widely scattered but would not exhaust them even when they are abundant. If we have $r > 1$, the curve for α rises more steeply than for $r = 1$; if we have $r < 1$ it rises less steeply. An interpretation of this might be, that when $r = 1$ mortality is proportional to numbers, if $r > 1$ it is more than proportional, if $r < 1$, numerical changes have relatively little effect. The following table will give an idea of the differences thus produced. For $r = 2$, values beyond $t = 5$ have not been calculated

t	−6	−5	−4	−3	−2	−1	0	+1	+2	+3	+4	+5	+6
α at $r=0.2$ ·	23	27	31	35	40	45	50	55	60	64	68	73.0	77.0
α at $r=1.0$ ·	0.25	0.67	1.79	4.7	11	26	50	73	88	95	98	99.3	99.75
α at $r=2.0$	—	0.045	0.33	3	10	12	50	88	90	99.7	99.9	99.99	—

The curve having been plotted in one of its various forms, a set of regularly increasing density values is made to correspond with the values, the density values increasing from the extreme $-t$ value to the extreme $+t$ value. These density values may be numbers but they may also be the ordinary logarithms of numbers. Without altering the curve, we can space the densities so as to investigate hypothetical cases where small increases in density produce large increases in mortality, or where large increases in density are required for this. Thus with the values for the curve given above where $r = 1$ we can set 1 for log density opposite $t = -3$ which means a 4.5% mortality at a density of 10 and the log density of 2.5 opposite $t = 0$ which means a 50% mortality at a density of 170 approximately. Using a number scale, we can set a density of 10 at $t = -2$ which means a 12% mortality, with a density of 210 opposite $t = 0$ with a 50% mortality. Using the same number scale, mortality of 50% at density 10 would give a mortality of about 86.75% at a density of 200. Mortalities for host densities are read off from the curve and rather accurate readings are required, so it is best to draw the curve once and for all on a large scale. This is particularly important at high densities.

Even with the curve drawn on a large scale, it is difficult, at the high levels, to read the percentage mortality accurately. More precise figures can be obtained by calculating the increment or decrement of t as proportional to the increment of host density. For example, if at $t = 4$ the population has

been set at 100 and at $t=5$, has been set at 200, we know that a population value of 150 corresponds to $t=4.5$ or, in general that an increase of 10 in the population corresponds to an increase of 0.1 in t. A population of 176 will thus correspond to 4.76 and inserting this in the equation the exact percentage of parasitism can be calculated, either from a table for exponential functions or with a good slide rule.

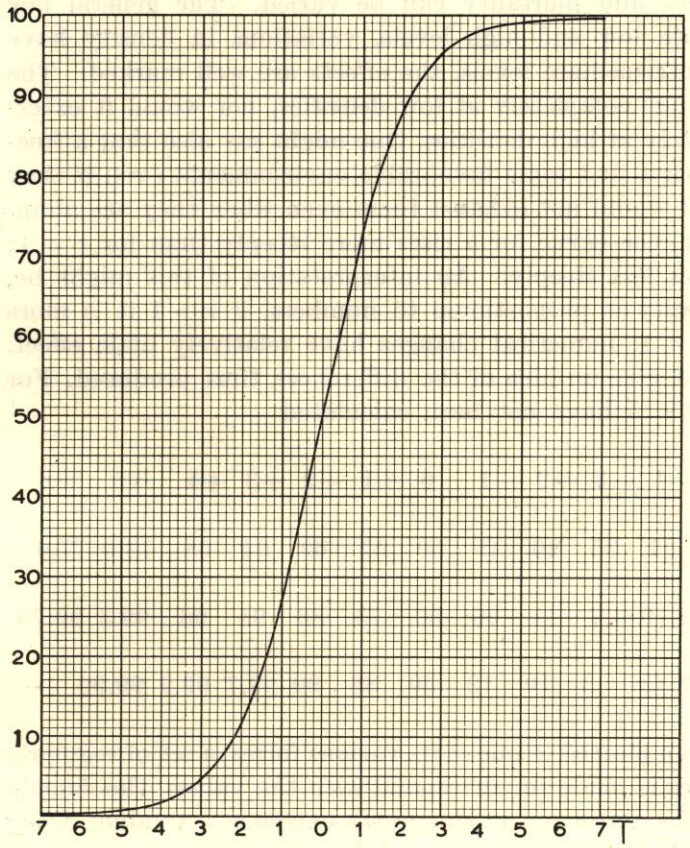

FIG. III.

Curve of the equation, $\alpha = \frac{K}{1 + e^{-rt}}$, where $K=100$ and $r=1$. Ordinates = % mortality, abcissae = values of t, which are + to the right of 0 and — to the left of 0.

To illustrate the use of the curve, a few examples may be given. Suppose that we begin with a population of 10 individuals with a reproductive rate of 50 and use a scale where the log of host density changes by 0.5 for each change of one unit in the value of t, and set the density scale at 1 (for log density) opposite $t = -4$. The course of events is then as follows:

*G*1:	$10\times50=$	$500\cdot\log_{10}$ *H.D.* 2.699	: $\alpha=35\%$	Number surviving = 325
*G*2:	$325\times50=16{,}250\cdot$	» 4.2108	: $\alpha=91.6\%$	» =1365
*G*3:	$1365\times50=68.250\cdot$	» 4.8388	: $\alpha=97.95\%$	» =1706
*G*4:	$1706\times50=85.300\cdot$	» 4.9309	: $\alpha=97.95\%$	» =1748
*G*5:	$1748\times50=87.400\cdot$	» 4.9415	: $\alpha=97.98\%$	» =1765
*G*6:	$1765\times50=88.250\cdot$	» 4.9455	: $\alpha=98.0\%$	» =1765

Thus the population has risen from 10 to 1765 and has become stabilized there; with a 98% mortality.

If we have 400 individuals with a reproductive rate of 3, the population rises in the 6th generation to 775 and becomes stable at about 780, with a mortality of about 65%.

If we start with a population of 10 and a reproductive rate of 3 the population requires 10 generations to level off and does so at a value of about 782. The population curve here is distinctly sigmoid (Fig. 5).

If we construct the curve for α with $r = 1.05$ and begin with a population of 10 and a reproductive rate of 3 (setting a density of 10 opposite $t = -3$)

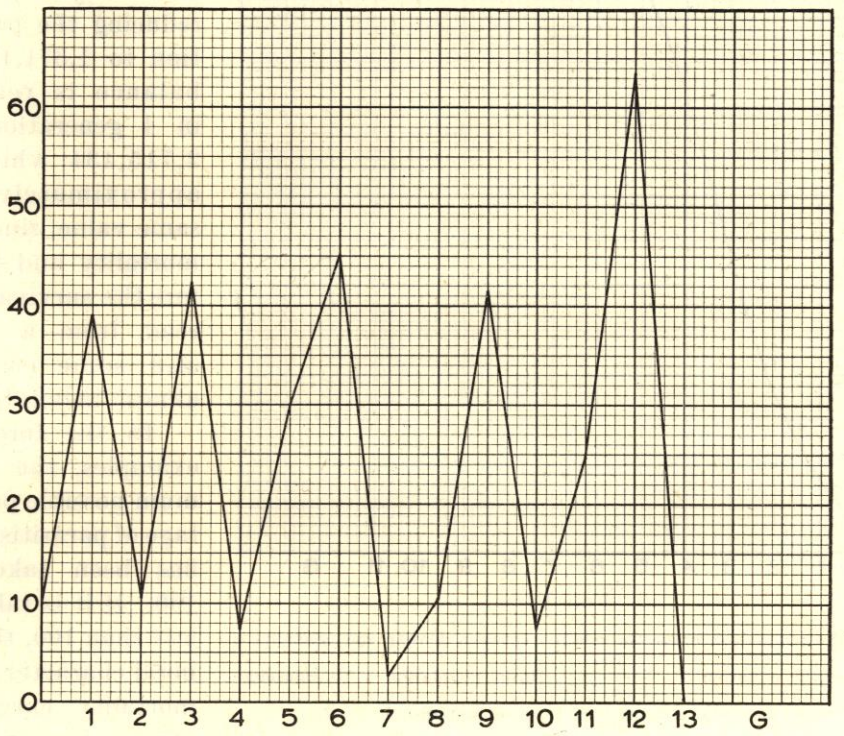

FIG. IV.

Curve of a population where the relation between density and mortality is given by the equation of Figure 3 ($r = 1.05$) with $H = 10$, $h = 4$; values set are a density of 10 at $t = -5$ with an increase of density of 20 for 1 of t.

we get a series of oscillations such that the surviving population in the 14th generation is about the same as the surviving population in the 1st generation (Fig. 6). Changing the reproductive rate from 3 to 4 produces cycles of greater amplitude and a drop below unity in the 13th generation (Fig. 4). With a reproductive rate of 5, we get in the 6th generation, a return to the value in the 2nd generation, after which the cycles are perfectly regular (Fig. 7). It would probably be hard to infer from an inspection of the population values, that they arise from a combination of constant mathematical laws.

If, in a case where a balanced position is reached, an independent mortality factor intervenes, the population proceeds again to a condition of balance. For example, using the value $r=1.05$ with 20 organisms and a reproductive rate of 20, balance is reached, according to my calculations, at 2,742,865 in 10 generations. If then there is a 50% mortality, reducing the population to 1,371,432, a balance is reached in 4 generations at 2,715,434 which is approximately the same value, since the mortality and therefore the survival, was read from a curve and some error is almost inevitable.

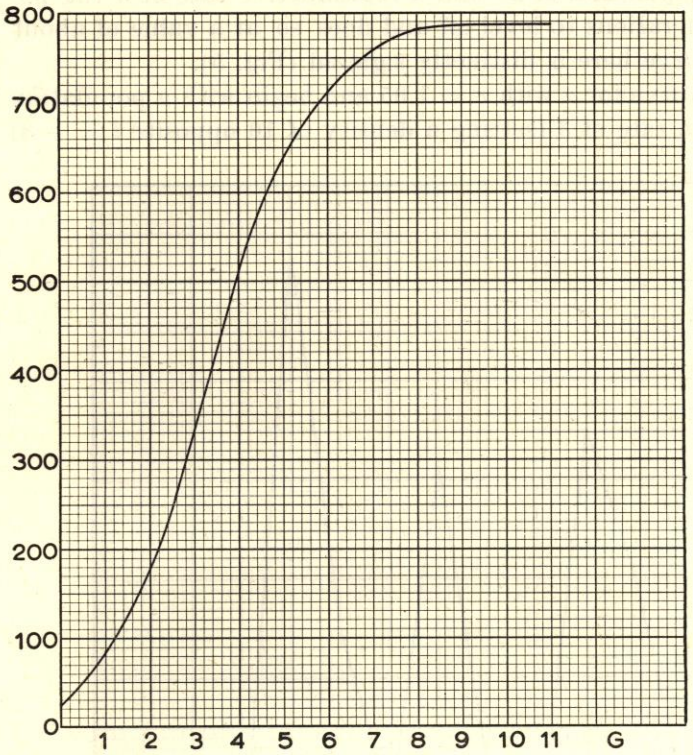

FIG. V.

Curve of a population where the relation between density and mortality is given by the equation $a = \frac{100}{1+e^{-1\cdot05}}$. Initial population = 10, reproductive rate = 3; a density of 10 is set opposite $t = -3$.

In the foregoing examples, the maximum possible percentage of parasitism (K) has been taken as 100. If it is taken at less than 100, the general character of the mortality curve remains the same and tends asymptotically to the value of K. The population will then reach a balance only when we have a relation between reproductive rate (h) and K such that

$$\frac{h-1}{h} \leqq K .$$

Thus if $K = 20$, h must be equal to or less than 1.25, for eventual balance. Taking it as 1.25, with an initial population of 50, balance is reached in about 20 generations with a population of 143 and a parasitism of 19.77.

An interpretation of such cases might be that other factors keep the effective reproductive rate down to a constant level which would allow continuous increase, but the addition of the parasitic factor produces eventual balance.

It is possible to envisage the mathematical process as the interaction of a parasite and a host. If we assume the existence of enough parasites to produce, in the first generations, the parasitism given by the curve, the parasites issuing in each generation can immediately be calculated. For exam-

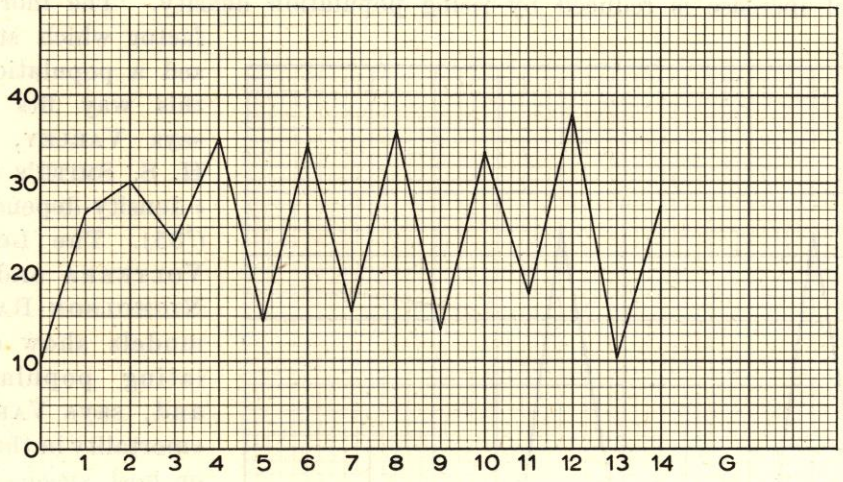

FIG. VI.

Curve of a population where the relation between density and mortality is derived from the equation $a = \frac{100}{1 + e^{-t}}$. Initial population = 10, reproductive rate = 3; Ordinates = population values, abcissae = = generations. The values set are = 4.5% mortality for $H = 30$, = 37 % for $H = 540$.

ple, where we have $H = 10$, $h = 3$, $r = 1.05$ with a density of 10 set opposite $t = -3$ and an increase in density of 20 for each unit of t, we get:

Generation	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Hosts	30	80	90	70	105	44.1	104	46.8	108	40.5	99.6	52.2	114	30.9
Parasites	3.3	50	66.6	35	90.3	9.3	88.4	10.8	94.5	7.3	82.2	14.2	103.7	3.4
Hosts Emerging	26.7	30	23.4	35	14.7	34.8	15.6	36.0	13.5	33.2	17.4	38.0	10.3	27.5

This is an oscillating curve and it will be noted that the reproductive rate of the parasite is related to the changes in host density for which it is itself responsible.

Thus, the simple mathematical theory outlined above produces, at least as a mathematical conclusion either a steady increase to a state of balance, an oscillating increase to a state of balance, or a series of oscillations whose character differs according to the values inserted in the equations.

This point seems of interest in connection with the recent discussion between MILNE, SOLOMON and VARLEY about of the use of the term «density-

dependent ». VARLEY, in his contribution ('35) stressed the difference between the two models used to explain the way in which animal populations are stabilized: the VERHULST-PEARL model and the LOTKA-VOLTERRA mode, used in a special form by NICHOLSON and BAILEY. VARLEY says that the VERHULST-PEARL model considers one species only and assumes that its relative rate of increase is reduced by rising population density. The mortality factor which stabilized a population in this way fits best, says VARLEY, with H. S. SMITH'S term, « density-dependent » ('35). The LOTKA-VOLTERRA and the NICHOLSON-BAILEY models show oscillating populations and, says VARLEY, «mortality in the prey or host species does not show a simple relationship to its own population density ». He adds, that « the maximum mortality lags a quarter of a cycle behind the population density ». Hence VARLEY introduced the term: « delayed density-dependent factor », justified also, he thinks by the fact that « the maximum rate of increase of the predator or parasite is a quarter of a cycle after its own population minimum, so that the effect is delayed, being density-dependent for two quarters of the cycle and inverse for the other two ». A genuinely « density-dependent » mortality factor, for VARLEY and also for SOLOMON ('58), is one whose intensity increases with the numerical value of the population in which it acts, becoming more intense as the population increases and less intense as it decreases. An inverse or inversely density-dependent factor, is one whose effect increases with a decrease in the population on which it acts, and decreases as this population increases.

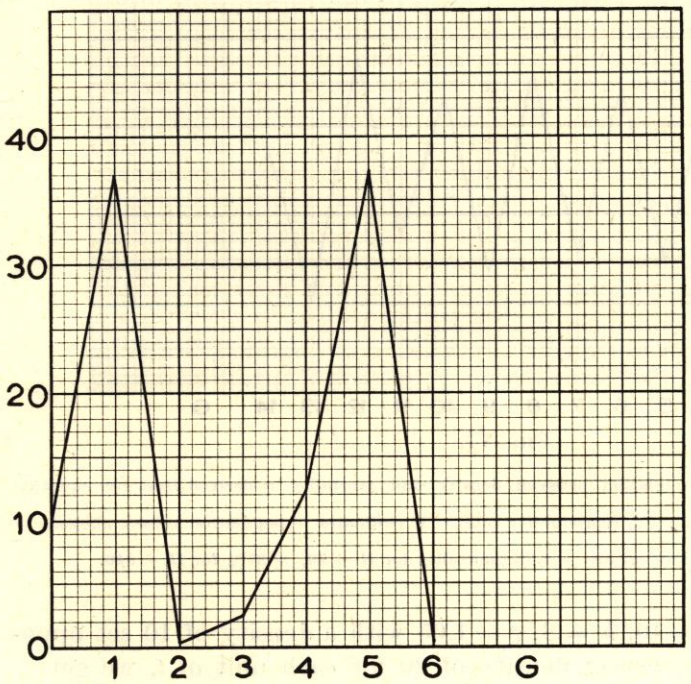

FIG. VII.

Curve of a population where the relation between density and mortality given by the equation of Figure 6 ($r = 1.05$) with $H = 10$, $h = 5$; a density of 10 is set opposite $t = -3$.

I do not feel able to agree entirely with the contentions of VARLEY. In the equation of LOTKA and VOLTERRA and in the NICHOLSON-BAILEY equa-

tions also, the reproductive rate of the *individual* predator rises when the population of the prey rises and falls when the population of the prey falls. I am satisfied, from my personal discussions with H. S. SMITH that this is how he understood the term « density-dependent ». That the maximum *mortality* produced by the predator lags behind the population density and that the *maximum reproductive rate* of the predator is a quarter of a cycle behind its own population minimum, does not fundamentally alter the position. It does not seem correct to describe the predator as an inverse factor. Furthermore, as has been shown above, a curve of the VERHULST-PEARL type may be derived from cases of the interaction of a host and its parasite. Here, however, the density-dependence of the reproductive rate of the individual predator, though it always exists, may during certain long mathematical intervals, be inconspicuous.

VARLEY considers that when NICHOLSON says that « for the production of population balance, it is essential that the controlling factor should act more severely against an average individual when the population density is high, and less severely when the population density is low », he is speaking in terms of the VERHULST-PEARL model, although he « then proceeds to elaborate his theory of population balance by parasites, which is a form of model two. NICHOLSON does not point out, that according to his theory, the host's mortality due to parasitism reaches its maximum not when host density is maximal but when it is half way down to ist minimum again. Hence parasites, according to his theory, do not fit his definition of controlling factors which I have quoted ». This criticism seems to be justified but I suspect the confusion to which it refers is partly due to the fact that NICHOLSON tends to transfer to the population the attributes of the individual. The reproductive rate and therefore the killing power of the individual may be strictly density-dependent but of course, if the parasite population is very small with respect to the host population it will not be able to respond adequately to the host's high density. On the other hand, a larger number of parasites may produce a large host mortality when the host population is well below its maximum. The individual parasite, according to the assumptions adopted is still, nevertheless, strictly density-dependent in its reproductive rate.

I must agree with SOLOMON ('58) that in the thought of H.S. SMITH the term « density-dependence » referred to the relation between the individual reproductive rate of a parasite or predator and the density of its prey. The fact that the density-dependence in a series of generations may be delayed if we use the LOTKA-VOLTERRA or NICHOLSON-BAILEY equations has, so far as I can see, no real bearing on the matter. The numerical sequences obtained with these equations could not, indeed, be obtained but for the fact that the concept of the density-dependence of the individual reproductive rate is incorporated in them as a basic assumption.

As I have already said, the mathematical models discussed in this paper need not be interpreted as models of host-parasite interaction. They may

be regarded simply as depicting a relationship between mortality from unspecified causes and the density of a population and the variations in the mortality may be regarded as due simply to the inaccessibility to the organism of the materials necessary for its existence.

Hovever, we need not claim that any one of the special mathematical formulations so far produced fits all the facts. THOMPSON'S original « model », except for the fact that it gives complete annihilation corresponds quite well to the course of events in certain successful cases of biological control, and this applies also to certain applications of the formula of NICHOLSON. This formula, with other values, gives oscillations increasing in amplitude with an eventual fall to a level below unity. This phenomenon does not appear to have been observed in nature and certainly is not general though it may occur exceptionally. On the other hand, it has been produced in experimental studies by T. BURNETT and by NICHOLSON himself. The rise of a population to a level at which it becomes more or less stabilized or a more or less regular oscillations are much more frequently observed. This is what we expect and find with many introduced insects and the oscillations we find in such cases are also fairly similar to those produced by the mathematical formulations in the preceding sections. The sigmoid curve has also been found in many experimental studies, such as those of PEARL and in relation to field populations, such as the sheep population of Australia studied by DAVIDSON. It does appear that it is much easier to find in nature phenomena in agreement with the laws expressed by this curve than to fit them into the theoretical picture developed by NICHOLSON since the curves produced by his formula cannot really be interpreted as indicating a balance, but rather a lack of balance, ending in a catastrophe.

I have not attempted, in this short article, to enquire whether a reasonable theory of the « natural control » of organisms can be constructed on the postulate of « density-dependence ». I propose to return to this later. In this paper I have limited myself to the suggestion, that in spite of the statements of certain authors, there are among the various « mathematical models » proposed for study by THOMPSON, several which produce the « fluctuating balance » regarded as theoretically desirable by ecologists without producing results that can perhaps be reproduced in laboratory experiments, but do not seem to correspond to the events we observe in the field. K. E. F. WATT ('59) and following him, C. S. HOLLING ('59), have recently used one these models (THOMPSON, 1930a, p. 647) but have not referred to the fact that they were proposed long ago by the present writer. For this reason and also in order to explore certain aspects of special interest to the writer this brief review is being published.

I should like to offer my grateful thanks to my old friend Professor GUIDO GRANDI for accepting the paper for publication in the Bulletin of his Institute; and also to the National Research Council of Canada for covering some of expenses entailed by the production of the paper.

RIASSUNTO

Nel presente lavoro l'A. discute le affermazioni di KLOMP secondo le quali la teoria di NICHOLSON e quella di THOMPSON, sulle relazioni intercorrenti tra ospite e parassita, differiscono principalmente per il fatto che, mentre nella prima il tasso di riproduzione del parassita è correlato e dipendente dalla densità dell'ospite, nella seconda è costante e limitato soltanto dalla capacità riproduttiva propria della specie. Egli mette in evidenza che tra i vari « modelli matematici » che ha proposto, parecchi giungono all' « equilibrio fluttuante » considerato come teoricamente desiderabile dagli ecologi.

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