a background of neutrinos and antineutrinos would make our universe symmetric with respect to matter and antimatter except for "small fluctuations" like the matter observed in our galaxy. Note, in this connection, that reaction 5, $Cl^{37}(\nu, e^-)Ar^{37}$, can occur only with neutrinos, but that reaction 7, neutrino-electron scattering, can occur both with neutrinos and with antineutrinos. Thus, if a detectably large background of neutrinos (or antineutrinos) exists, one can determine the ratio of matter (neutrinos) to antimatter (antineutrinos) in the cosmic signal. This kind of observation—which distinguishes matter from antimatter at astronomical distances—cannot be made with electromagnetic waves because the light from antiatoms is identical with the light from ordinary atoms (*28*).

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28. Developments in the field of neutrino astronomy that have occurred since 1 September 1964 are described in the article "Observational Neutrino Astronomy: a ν-Review," by J. N. Bahcall, which will appear in the Proceedings of the Second Texas Conference on Relativistic Astrophysics (to be published by University of Chicago Press).

Dynamics of Epidemics of Plant Disease

Population bursts of fungi, bacteria, or viruses in field and forest make an interesting dynamical study.

J. E. Van der Plank

The potato blight fungus survives the winter in diseased tubers. At the end of winter the potato stores are opened and diseased tubers are dumped outside in cull piles. Here the tubers sprout. The fungus invades the sprouts and in due course spreads from the diseased sprouts to young potato fields in the neighborhood. Alternatively, diseased tubers taken from the winter stores are planted as "seed." Shoots emerging from the seed become diseased, and from these primary diseased shoots the fungus spreads throughout the field, and from field to field. It has been found that in a very susceptible variety of potato there is about one primary diseased shoot per square kilometer of potato fields (*1*). To spread from the primary diseased shoots and destroy all the fields, the fungus must increase about a billionfold. In favorable conditions it can do this in less than 90 days.

That is a description of an epidemic process. Details of the process vary with the different blights, rusts, mildews, blasts, and other diseases that afflict our gardens, fields, orchards, forests, and plantations. But all epidemics have in common a dynamic process of increase of the pathogen—of the fungus, bacterium, or virus that causes the disease.

This dynamic process of increase is my topic, especially the rate of increase and the factors that govern the rate.

Originally the rate was studied largely with the practical aim of determining the best strategy for controlling the various diseases (*2*). But here I barely touch on practical problems of disease control and, instead, study the general pattern of disease increase, in the belief that understanding of the epidemiology of plant diseases can do much to illumine the wide problems of population dynamics.

In this study it is unnecessary to distinguish between an increase of disease in a population of plants and an increase of the population of the pathogen in these plants.

A Relative Infection Rate

To follow the increase of disease with time, we define an infection rate r as

$$\frac{dx}{dt} = rx(1-x) \qquad (1)$$

where x is the proportion of susceptible tissue infected at time t. The rate dx/dt is related both to the proportion x of

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tissue already infected and to the proportion $1 - x$ of tissue still healthy and available for infection; and r is measured experimentally as the regression coefficient of $\log_e[x/(1-x)]$ with time.

Figure 1 shows the progress of blight in potato fields during an epidemic (*3*). The top graph records the proportion x of tissue infected at various dates. The curve is sigmoid. The lower graph presents the same data, with $\log_e[x/(1-x)]$ plotted instead of x. A straight line is fitted, and the regression coefficient with time in days is 0.22. Hence $r = 0.22$ per day. This is no more than an estimate of the average value over the whole period. For illustration we chose data for which r stays roughly constant over the period of observations. But there is no reason why r should stay even roughly constant. Weather governs potato blight epidemics; r is inherently as variable as the weather (*4*) and is best estimated over short intervals of time.

For potato blight and the rust diseases of cereals, r is commonly between 0.1 and 0.7 per day. Interpreted approximately for the early stage of an epidemic when x is small and the proportion of healthy tissue available for infection is still large, the expression $r = 0.1$ per day means that disease doubles in 7 days, and $r = 0.7$ per day means that disease doubles in 1 day. Years, not days, are often the natural units of time for the spread of plant diseases, especially for diseases of perennial plants. In five epidemics of swollen shoot disease of cacao (*5*), r varied from 0.4 to 0.9 per year. (The progress of these swollen shoot epidemics is recorded in Fig. 2.) A more rapid increase, as measured in years, occurred in the case of chestnut blight in the United States. The disease is native to the Orient. In 1904 it was found attacking a few American chestnut trees in the New York Zoological Garden in Bronx Park. During the next 40 years it spread throughout the range of the American chestnut in the eastern United States—from southern New England and the Middle Atlantic States to northern Georgia, Alabama, and Mississippi in the south and to Indiana in the west—killing most of the trees. In any particular area there was about a sevenfold increase of the disease each year during the early stages of an attack (*6*); this is equivalent to $r = 1.9$ per year.

The rate r is a sensitive index. It reflects in a single figure all the conditions governing infection. A high rate —say, 0.7 per day—means that conditions favor infection: the host plants are susceptible; the pathogen is virulent; the weather is "disease weather"; and other environmental conditions are also conducive to disease. A lower-than-usual rate shows some conditions to be adverse to infection: the host plant is perhaps of a resistant variety, the weather is too dry for a disease that needs moisture, and so on. Every factor that promotes or retards infection—irrespective of whether it comes from the host, the pathogen, or the environment—affects r. That is why r is such an informative parameter.

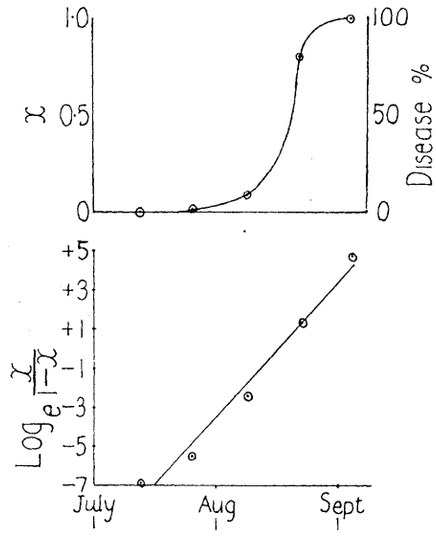

Fig. 1. Progress of an epidemic of potato blight.

Period of Latency and Period of Infectiousness

Equation 1 relates r to the proportion of infected tissue. But *infected* is a broad adjective, and infected tissue is of three sorts: (i) infected tissue that has not yet become infectious; (ii) infectious tissue; and (iii) tissue that was infectious but has ceased to be so. Newly infected tissue takes a period p to become infectious (for example, to start forming spores which disperse to propagate the fungus anew); this is called the period of latency. Thereafter it remains infectious only for a period i, after which it is "removed" (to use the customary word) from the epidemic.

Suppose $x(t)$ is the proportion of susceptible tissue infected at time t. Then $x(t-p)$ represents the proportion infected at time $t-p$, and, at time t, the proportion that has passed through the period of latency, p, and is either infectious or removed. Similarly $x(t-i-p)$ represents the proportion infected at time $t-p-i$, and, at time t, the proportion that has been removed. Hence $x(t-p) - x(t-i-p)$ is the proportion infectious at time t.

A new infection rate R, based specifically on infectious tissue and not just infected tissue, is defined by

$$\frac{dx(t)}{dt} = R[x(t-p) - x(t-i-p)][1 - x(t)] \qquad (2)$$

The definition means that R is proportional both to the infectiousness of the infectious tissue (for example, to the abundance of spores produced per unit area of spore-forming leaf tissue) and to the susceptibility of healthy tissue to infection (as shown, for example, by the proportion of spores which, after falling on healthy leaves, germinate and initiate new lesions).

We want a relation between r, R, i, and p. To get it, we simplify the problem by first confining attention to the early stage of an epidemic when $x(t)$ is small and $1 - x(t)$ is near enough to 1 to be assigned a value of unity for all practical purposes. At this stage, if conditions stay constant, r settles down to a value which is independent of t and x and is related only to R, i, and p (*2*). The relation is

$$r_l = \frac{R(e^{ir_l} - 1)}{e^{(i+p)r_l}} \qquad (3)$$

This relation is for logarithmic increase—the form of increase found only early in an epidemic. To show this we add the subscript l, and have r_l. By definition, a quantity increases logarithmically when the rate of increase is proportional to the quantity itself. Early in an epidemic—when $1 - x(t)$ is near enough to 1 to be negligible—the *absolute* rate of increase dx/dt is proportional to x and therefore logarithmic. It follows that the *relative* rate $dx/(x dt)$, which is r_l, is independent of x.

A practically constant value of r_l from day to day or year to year—a value practically independent of t and x —is not uncommon in epidemics; two examples are given in the next section.

Equation 3 is the simplest link between the infection rate and the factors that affect the rate: the weather, fungicides, resistance of the host plants to infection, and so on. The factors that affect r_l do so through R, i, or p irrespective of whether the factor comes from the host, the pathogen, or the environment. For example, resistance of the host plants to infection reduces r_l by reducing R or i or by increasing

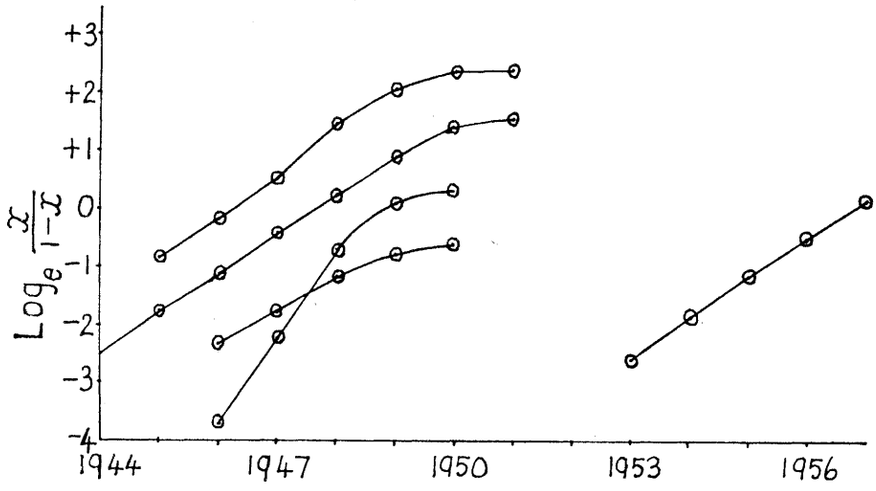

Fig. 2. Progress of five epidemics of swollen shoot disease of cacao.

p. All three types of resistance are known to occur, often in combination. So, too, weather favorable to disease acts by increasing R or i or by reducing p.

The effect of a factor may sometimes be on R, i, or p singly. But even then the effect can be evaluated only if we know all three. For example, chemical fungicides are used to protect plants from infection. Spores falling on the protected plants have to break through the fungicide barrier before they can infect. The fungicide reduces the proportion of spores that can infect: it reduces R. But the effect on r_t is determined by i and p as well. The same degree of fungicidal action —that is, the same proportion of spores stopped from infecting the host plants, and the same relative reduction of R —may be adequate for controlling one disease but not for controlling another. The rust diseases of cereals commonly have values of R, i, and p that make control by fungicides difficult. Commonly, i exceeds 10 days, p exceeds 7 days, and R exceeds 20 per day in unprotected fields. This combination of values is one of the several reasons why fungicides have been little used against cereal rusts.

Equation 3 defines the threshold condition for an epidemic. It shows that $r_t > 0$ only if $iR > 1$. In terms of systemic disease—disease that permeates the whole plant system, in contrast to disease, such as potato blight, that occurs in localized lesions—this means that an epidemic will develop, in the long run, only if each infected plant (while it is infectious) infects, on an average, more than one healthy plant. Note that the period of latency, p, does not enter into the threshold condition.

At the other extreme, Eq. 3 shows that there is an upper limit to an epidemic's explosiveness, and it roughly fixes that limit. Because removals reduce the upper limit, we can logically ignore them and write

$$pr_t = pRe^{-pr_t}$$

As pR increases, pr_t increases, rapidly at first but later more and more slowly. I know of no recorded epidemic in which pR exceeded 250. This value corresponds roughly to $pr_t = 4$. Even if, to be on the safe side, we assume that pR could be as great as 2500, this is equivalent only to $pr_t = 6$. It seems that pr_t probably has a maximum value of from 4 to 6; thus, if we know p we can fix the probable maximum of r_t. Note that the upper limit of explosiveness is determined by p alone.

We have been ignoring inevitable biological variation. We have also been ignoring the fact that some lesions, such as those of potato blight so carefully studied by Lapwood (*7*), grow markedly in size as they grow older; the period of latency measured from the time of initial infection is longer in tissue near the periphery than near the center. We can correct as follows. If $a_1, \ldots a_j, \ldots a_n$ are the proportions of tissue with periods of latency $p_1, \ldots p_j, \ldots p_n$, we replace the constant p in Eq. 3 by a weighted value $\acute{p}$, where

$$e^{-\acute{p}r_t} = \sum_{j=1}^{n} a_j e^{-p_j r_t}$$

A corresponding equation, with p replaced by $i + p$, gives weighted estimates of $i + p$ or of i to substitute in Eq. 3. These equations can also readily be modified to express changes of infectiousness with time, as when infected tissue starts to form spores sparsely at first but abundantly some days later, or when infectious tissue gradually loses its capacity to form spores. For the particular purpose of applying the threshold condition, no weighting is needed (because the need vanishes as r_t approaches zero). For R we estimate unweighted means.

The Logarithmic Stage and After

We have been considering the logarithmic stage of epidemics in order to find the simplest relations between r, R, i, and p. What happens beyond this stage? The logarithmic stage is the stage in which the progress of the epidemic is unhampered by lack of susceptible, healthy tissue that can be infected—the stage in which disease can increase as if the scope for increase were unlimited. As the epidemic progresses beyond the logarithmic stage, less and less healthy tissue is available for infection until, when all plants are 100 percent diseased, there can be no further infection.

To follow the progress of an epidemic beyond the logarithmic stage is to follow the effect of decreasing proportions of healthy tissue—of a shrinking supply of food for the fungus or other pathogen. And in order to concentrate on the effect of decreasing proportions of healthy tissue, with a minimum of complications from variable weather, we choose diseases of perennial plants in the tropics, where changes from year to year are less, on an average, than the day-to-day changes in annual crops in the temperate zones.

Let us start with the logarithmic stage. The prediction is that, if conditions stay constant from year to year, the epidemic will settle down (*8*) and the infection rate will remain constant. Clove trees succumb to a fungus disease called "sudden death." Among the 2.5 million trees on the island of Pemba, in the Indian Ocean, losses were relatively small at the time Nutman and Sheffield (*9*) studied the disease there, and the epidemic was practically in the logarithmic stage. The annual rate of increase of disease stayed constant, with $r_t = 0.13$ per year (*2*). Fusarium wilt of banana plants—a fungus disease—was discovered in Jamaica in 1911, and legislation was passed requiring infected plants to be destroyed. Inspectors recorded the number of plants de-

stroyed (see *10*) from 1912, when 625 were destroyed, to 1939, by which date over 4 million plants had been destroyed. The records have been tabulated (*11*). Changes in the infection rate accompanied changes in and relaxation of the inspection regulations. But over periods during which the regulations were unchanged, the rate remained practically constant. For example, from 1920, when the regulations were relaxed, until 1929, when the parish having the worst infection was excluded from the campaign, the number of infected plants destroyed by the inspectors increased from 2400 to 241,000 at the rate $r_i = 0.51$ per year (*2*). These data support the theoretical expectation of a constant rate under constant conditions during the logarithmic stage.

Now let us consider increase after the logarithmic stage is over. In Zanzibar, sudden-death disease destroyed more clove trees than it destroyed in Pemba. By 1946 half the clove trees had been killed (*9*), and from indirect evidence concerning the number killed annually from 1937 to 1946 it was estimated that r stayed fairly constant at roughly 0.42 per year (*2*). Swollen shoot disease of cacao is a systemic virus disease. Figure 2 is based on records of five epidemics, in Ghana, Nigeria, and Trinidad (*5*). $\text{Log}_e [x/(1-x)]$ is plotted against time, in years. At first the lines are nearly straight—that is, r is nearly constant for each separate epidemic. This finding links up with the evidence for the logarithmic stage. But later, as the epidemic proceeds, r begins to decrease markedly in four of the five epidemics.

What has theory to say about the progress of an epidemic under constant and uniform conditions? We interpret "constant conditions" to mean constant i, p, and R.

In Fig. 3 we use p as the unit of time. The straight line A is for $pr = 1.386$. Curve B, with $pR = 5.544$, is for an epidemic without removals. Curve C, with $i = p/3$ and $pR = 14.98$, is for an epidemic with removals. The curves were obtained (*12*) by integrating Eq. 2. The curves are made to coincide during the logarithmic stage, and $pr_i = 1.386$ for all of them. This means that Fig. 3 represents diseases which, in the logarithmic stage of the epidemic, increase fourfold during one period of latency, p (because $e^{1.386} = 4$). Figure 3 is probably as representative as any single graph can ever be of slow (but not fast) epidemics of potato

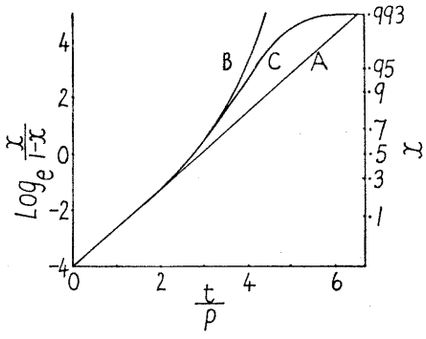

Fig. 3. Progress of disease in three models: in A, r is constant; in B, p and R are constant and there are no removals; in C, i, p, and R are constant.

blight and the cereal rust diseases, and of swollen shoot disease of cacao. [The evidence for swollen shoot disease (*13*) is scanty.]

Curve A shows r constant. Curve B (for an epidemic without removals) follows curve A at the start but then diverges, with r increasing and continuing to increase for the rest of the epidemic. Whereas curve A is based on infected tissue of all sorts, curve B is based only on that fraction which has passed the period of latency, p, and, in the absence of removals, is infectious. Thus it is p that causes r to increase. Quantitatively, as the epidemic runs from start to finish—as x increases from 0 to 1—r increases e^{pr_i} times, if p and R are constant.

Curve C (for an epidemic with removals) follows curve B at first. There is the same increase of r. Later, removals begin strongly to influence the form of the curve, and r starts to decrease. But the decrease comes late in the epidemic, and in our model r does not fall back to its initial value r_i until x exceeds 0.98. We can change the model and alter this detail. But in no model of the sort of epidemic we have been considering will removals fully offset the increase of r with time that p causes.

In epidemics of swollen shoot disease of cacao, r decreases with time. In models for epidemics under constant and uniform conditions r increases with time, at least until near the end of the epidemic. Where is the difference?

Our models, which curves B and C describe, require that conditions be both constant and uniform. The epidemics of swollen shoot disease proceeded under conditions that were probably reasonably constant from year to year, but in populations that were far from uniform. The cacao trees were grown from normally diverse and heterozygous seedlings and were of variable size, growing in plantations having variable environment from tree to tree.

An epidemic proceeds as healthy tissue succumbs to infection. That is why the proportion of healthy tissue enters our equations. But there is an implication: the equations hold only if all healthy tissue is uniformly susceptible and uniformly vulnerable to infection. Any departure from uniformity, whatever its source, necessarily causes r to decrease with time. Consider a systemic disease such as the swollen shoot disease of cacao. To give a hypothetical example, if 80 percent of the trees were susceptible and 20 percent were immune from infection, r (defined by Eq. 1) would decrease as x approached 0.8 and would finally become zero when $x = 0.8$. For one reason or another, we cannot picture any natural epidemic of plant disease proceeding under uniform conditions, and there is always an inherent tendency for r to decrease because of this departure from uniformity. The tendency may be masked by other factors, but it is there.

Two opposing tendencies exist in epidemics. The period of latency, p, causes r to increase with time. Departure from uniformity causes r to decrease with time. Both tendencies are weak when an epidemic starts but strong later. We can divide an epidemic into three arbitrary stages. First, there is the logarithmic stage, when neither p nor lack of uniformity can stop r from being constant with time, if conditions stay constant. (In making this statement I ignore sampling errors; they are not being discussed.) Secondly, beyond the logarithmic stage there is a stage that lasts until x is from 0.15 to 0.5, depending on circumstances and the accuracy desired in interpretations. During this stage neither p nor lack of uniformity markedly affects the constancy of r, and, as in the logarithmic stage, if any large change of r is observed it is likely to be caused by a change of R, i, or p, as when, for example, the weather changes and affects a weather-sensitive disease. Finally, especially after x exceeds 0.5, there is a stage when both p and lack of uniformity can cause r to change markedly with time. During this stage r is still a valuable parameter because it indicates the rate of progress of the epidemic, but, with our present knowledge, any attempt to analyze the effect of R, i, or p on r cannot be justified.

The End of an Epidemic

In annual crops (*14*) an epidemic may end because of a change in the environment—as when dry weather curbs a moisture-loving fungus—or because the host plants become resistant. But often it ends because the crop ripens. With a few exceptions, practical measures of disease control are aimed at reducing the amount of disease that can develop before the crop ripens. Thus, we can reduce the time available for the epidemic to develop by planting early-maturing varieties or, if the epidemic normally occurs late in the season, by planting early. Or we can reduce the initial inoculum from which the epidemic starts through sanitation (such measures include chemical eradication, crop rotation to kill the pathogen in the soil, sowing seed known to be healthy, isolation from outside sources of the pathogen, and so on) or through planting varieties with vertical resistance (*15*) to the common strains of the pathogen. Or we can reduce the rate at which the epidemic develops—that is, we can reduce r—by applying protectant fungicides, by manipulating the environment so as to make it less favorable to disease, or by planting horizontally resistant varieties. Peel off the empiricism of control methods and you will often find a dynamics problem beneath.

In some diseases of annual plants the pathogen passes through only one generation before the season ends. These are "simple-interest" diseases (simple interest on money does not itself earn interest). Most of the pathogens of simple-interest diseases survive well from year to year (in the soil, on or in seed, and so on), and although in a single season the epidemic ends after an increase of disease at simple interest, the increase over the years—as when disease caused by a soil-borne pathogen builds up when the crop is sown year after year in the same soil—is compound.

In perennial plants—to turn now to them—an epidemic can usually run its course, and we can see an epidemic as a whole process. The end differs according to whether infectious tissue is or is not removed.

Curve B of Fig. 3 represents an epidemic without removals. In an unlimited population of plants the epidemic can continue indefinitely, the proportion of surviving plants growing less and less. In a limited population, such as we ordinarily deal with, the epidemic continues until all plants are infected. Tristeza disease of oranges—a virus disease—is an example. On tolerant rootstocks oranges carry the virus without apparent symptoms on the tree and (so far as we know) without serious effect on yield. Infected trees are not removed, and in countries where the virus exists together with an abundant, efficient insect vector, all orange trees of the old commercially valuable varieties seem to be infected.

Curve C of Fig. 3 shows what happens when there are removals. The epidemic stops short of 100 percent infection (the final percentage being determined by i, p, and R). Chestnut blight in the eastern United States is an example. Blight swept through the American chestnut trees, destroying most of them but leaving some survivors. It is hoped (*16*) that resistant trees will be found among these survivors. This hope may well be justified, because, if susceptibility varied in the original population of chestnut trees, the surviving trees would indeed have a higher average level of resistance than the original population had. But even with uniform susceptibility a few trees could be expected to have survived the initial epidemic, because of removals.

Summary

In the context of this discussion an epidemic is defined as an increase of disease in a field, forest, or other population of host plants. The susceptibility of the host plants, the virulence of the fungus or other pathogen, and the weather and other environmental conditions all affect the relative rate of increase. They do so by affecting the time it takes newly infected tissue to become infectious, the time tissue remains infectious, the infectiousness of infectious tissue, and the susceptibility of healthy tissue to infection. These factors operate throughout the epidemic. Two other factors become increasingly important as the epidemic proceeds: the proportion of healthy susceptible tissue remaining available for infection, and the degree of uniformity of the population of host plants and of their environment.

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8. If an epidemic starts abruptly—for example, after artificial inoculation—the infection rate first varies with time even under constant external conditions and only later settles down to a constant value (for details, see J. E. Van der Plank, *2*). In the natural epidemics we are considering there had been plenty of time for the rate to settle down before observations started.
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12. Without removals and with p as the unit of time

$$x(t) = 1 - (1 - a) \exp \left[- \int_0^t pRx\,(t - 1)\,dt \right]$$

where a is the initial value of x. At first, the relative rate of increase of x—that is, the rate r—varies cyclically with time. These variations are irrelevant to our story now, and we make a very small in order to get rid of them largely before the graph starts. The starting point of Fig. 3 is not the point where $x = a$; instead, Fig. 3 starts quite arbitrarily but more conveniently where $\log_e [x/(1 - x)] = -4$.
13. From Thresh's table (see *5*) in which data for coppiced cacao trees are recorded, it seems that the incubation period for swollen shoot disease is from 1 to 2 years under plantation conditions. Very roughly, the incubation period (the time needed by symptoms to develop) indicates the period of latency.
14. This section pertains also to annual new growth of perennial plants.
15. According to current definition, "immunity" controls disease absolutely; "resistance" gives only partial control; "vertical resistance" gives control of some strains of the pathogen but not of others; "horizontal resistance" is spread against all strains. Commonly, both vertical and horizontal resistance occur together in one variety.
16. J. D. Diller, quoted by T. R. Peace in *Pathology of Trees and Shrubs* (Oxford Univ. Press, London, 1962), p. 403.