Results and discussion. The records of figure 1 show that the DRPs evoked by stimulation of posterior tibial nerve vary in amplitude in consecutive segments of the cord. Ipsi-potentials are large in cranial segments of the studied part of the cord (L6–S1) and become smaller in more caudal segments (S2–Ca1). Contralateral depolarizations are increased in records taken in 2 lowermost segments of the cord.

The mean sizes of the DRPs elicited by stimulation of posterior tibial and superficial peroneal nerves are illustrated in figure 2, A and B. These 2 nerves enter the cord through lower lumbar dorsal roots. It may be seen that in both instances ipsi- DRPs attain maximum amplitude in L7 segment. In neighboring segments (L6 and S1) potentials are decreased to approximately the same level. Their most rapid reduction occurs between S1 and S2 segments. Potentials in S2 and S3 segments are significantly lower than those in L7 dorsal roots ($p < 0.05$). In lower sacral segments the size of potentials ceases to diminish and in Ca1 dorsal root some enhancement of the DRPs is observed. Contralateral DRPs are small in the 2 last lumbar and the 1st sacral segments. Then they increase, but differences between the smallest and the largest potentials do not attain the level of statistical significance ($P > 0.05$). Figure 2, C and D, shows amplitudes of the DRPs evoked by volleys in caudal femoral cutaneous and pudendal nerves which enter the cord via sacral dorsal roots. In both cases the maximum sizes of ipsi- DRPs are in S1 dorsal root and are thus shifted one segment caudally as compared with those seen in figure 2, A and B. In neighboring segments a marked decrease of potentials is observed. It is smaller in caudal than in cranial parts of the portion of the cord studied. Only ipsi- DRPs to volleys in the caudal femoral cutaneous nerve are significantly higher in the S1 segment than in the L6 segment of the cord ($p < 0.05$). Contralateral potentials are very small in lower lumbar segments and increase markedly in lower sacral and Ca1 segments, the differences between depolarizations in L6 and Ca1 dorsal roots being statistically significant ($p < 0.05$). It should be noted that in all compared instances the largest ipsilateral DRPs are significantly higher than contralateral potentials in the same segments of the cord ($p < 0.05$).

The extent of spread of ipsi- DRPs to volleys in the superficial peroneal nerve cannot be directly compared with the findings of Carpenter et al.[5] who evoked potentials with lower stimulus strengths and recorded them in close proximity to the entry zone of this nerve. Differences between the present findings and our previous studies[4] are most probably due to the fact that in the latter instance the DRPs were elicited by stimulation of the dorsal roots in spinal animals. Our present results show that for each nerve the peak amplitudes of ipsi- DRPs correspond to the largest field potentials which in general conform to the levels of entry of afferent volleys into the cord[2,5–9]. In contrast to these data the distribution of contralateral DRPs does not show any relationship with the entry levels of afferent volleys. The enhancement of contralateral depolarization in S2–Ca1 segments which occurs irrespective of nerves employed for their elicitation indicates a very fixed pattern of depolarization in successive segments of these parts of the cord. There is evidence that the DRPs spread with different velocities on both sides of the cord and that temporal facilitation of potentials traveling caudally is larger on the contralateral than on ipsilateral side of the cord[4,10]. It has also been postulated that ipsilateral DRPs which spread in the caudal direction are transmitted to the contralateral side in several segments below the zone of entry of afferent volleys. It is conceivable that these factors may increase the size of the DRPs elicited by single volleys and spreading to the lower sacral and upper caudal segments of the spinal cord. The distribution of bilateral DRPs and hence of presynaptic inhibition may have functional significance. Sensory information received by L6–S1 segments originates from paired structures displaying antagonistic activity (hind legs) while that reaching S2–Ca1 segments comes mainly from non-symmetric organs (urethra, anal canal etc.). We suggest that differences between the level of ipsilateral and contralateral presynaptic inhibition in L6–S1 segments, together with similar inhibition on both sides of S2–Ca1 segments, are required for proper regulation of the activity of the structures innervated by both parts of the cord.

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Are plant toxins aimed at decomposers?

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Summary. Secondary plant substances may regulate the rate of decomposition. Evidence from many quarters suggests that this may be one of their primary ecological roles.

The widespread distribution of secondary plant substances in natural systems is well known. These are almost always interpreted as being defences by plants against herbivores and pathogens[1–7]. However, they are also effective against decomposers[3,8–11], and since the flow of organic matter through the decomposition subsystem is many times larger than that through the herbivore subsystem, in almost all ecosystems[8], the greatest quantitative effect of plant toxins is against decomposition. Such an effect on decomposition has been interpreted as secondary fallout from the battle

between plants and their predators. In the present discussion I suggest that the effect of secondary plant substances on decomposition may be one of their primary ecological roles.

The simplest scheme of nutrients in an ecological system would be something like that of figure 1. This arrangement has perfect dynamical stability according to the technique of Loop Analysis[12]. However, any real system of this form will collapse as soon as the finite store of nutrients is all transferred to the external sink. Call this a problem of mass stability (after Lotka[13]). This problem can be ameliorated by changing the system to the form of figure 2. However, the system now involves a dynamically destabilizing mutualistic loop among P, D, and N, and will tend to collapse. Dynamical stability and mass stability can be reconciled only if there is some sort of negative influence acting on or within the decomposition compartment. Plant toxins can play this role since the concentration of toxins in plant tissues entering the litter is tightly controlled by the metabolic homeostatic mechanisms of the plant. The system of figure 3 can be dynamically stable if the magnitudes of links involving toxins are sufficiently large compared to other links in the system (specifically, if $a_{TT}a_{NP} > a_{DP}a_{ND}$ and $a_{TP}a_{DT} > a_{TT}a_{DP}$ where a_{ij} is the magnitude of the direct effect of component j on component i).

In practice, the situation is more complicated. In almost every realistic loop diagram of an ecosystem, stability is not qualitatively guaranteed by the arrangement of links and compartments, but rather depends on the relative quantitative magnitudes of stabilizing and destabilizing loops. Many factors in addition to plant toxins can be relevant to this balance. Furthermore, real systems in fluctuating, stochastic environments need not have formal dynamic stability in order to persist. An adequately slow rate of collapse relative to the frequency of redemptive perturbations is all that is required. Thus plant toxins acting on decomposers can contribute to the persistence of a real ecological system even if they are not sufficient in themselves to ensure formal dynamic stability. Any magnitude of effect can be significant.

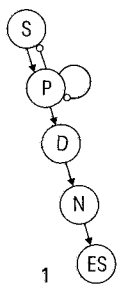
Similarly, the inhibitory effect of plant toxins on decomposers can assist the mass stability of the system. In the three-compartment flow model of figure 4, the largest accumulation of matter will be in the compartment preceding the slowest proportional rate of flow[13]. In a real system, nutrients can be lost most easily from the free-nutrients box. This danger of loss can thus be minimized by making the proportional flow from free nutrients to plants relatively large (efficient uptake of nutrients by plants), and by making the proportional flows from plants to decomposers and from decomposers to free nutrients relatively small. Plant toxins are an effective way of accomplishing the latter, and so again contribute to the persistence of the ecosystem. Note that by this strategy plants gain some control over all three transfer processes.

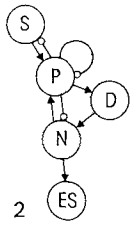
Toxic chemicals slow the rate of decomposition and lead to a direct increase in the rate of accumulation of organic matter in the soil[8]. This is especially important in oligotrophic systems on nutrient-poor soils[14]. Such soil organic matter is also an important feature for buffering the impact of external perturbations such as fire, since it is much less prone to giving up its constituent and adsorbed nutrients than is the living above-ground biomass[8,15,16]. Similarly, nitrification, which tends to lead to loss of scarce fixed nitrogen, is inhibited by plant chemicals in a variety of mature ecosystems[8,14,17,18].

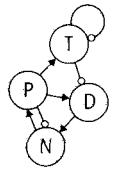
An individual plant interacts almost entirely with those decomposers directly beneath it. Thus, selection for successful toxic manipulation of decomposers would be straightforward and unproblematical. Furthermore, the requirements for a successful chemical strategy of this sort are not stringent, since only a moderate inhibition (rather than complete prohibition) of decomposition is aimed at. Any number of widely different chemicals would work equally well. Thus, this hypothesis would predict a great variety of chemical toxins among and within plant species, in accordance with observation[1-6]. Consumers reacting to these various different toxins would tend to become arranged in converging food chains with high specificity at the herbivore level diminishing among secondary consumers. This is often observed[19,20], but the hypothesis used here is simpler than the evolutionary cat-and-mouse game normally invoked[2,6] (but compare Gilbert[20]).

It is widely reported that plant toxins are most common in mature systems with a high biomass, on intrinsically poor soils, or where nutrients are otherwise limiting[4,5,8,21]. This is exactly what would be expected by the hypothesis discussed here, for in such situations attention to careful husbandry of nutrients is most critical, and would justify the metabolic costs of synthesizing toxins.

The resistence imparted by plant toxins is rarely absolute. It can be overcome by a process of active detoxification[2,6,8]. The time necessary for detoxification is often short compared to the life spans and even growing seasons of adult plants. Thus there is a problem in understanding how such chemicals can have a significant impact on the rate of herbivory (except in seeds where a short delay in predation may be critical in allowing other seeds to escape via dispersal[2]). By contrast, this 'soft' character of many secondary plant substances is exactly right for moderating rates of decomposition where complete protection of tissues would be inappropriate. More recalcitrant 'hard' compounds can also lead to this result by temporarily blocking access to more vulnerable structural compounds and so







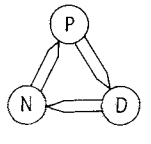


Figure 1. Mass instability, dynamical stability.

Figure 2. Potential mass stability, dynamical instability.

Figure 3. Potential mass stability and potential dynamical stability.

Figure 4. For any compartment n, $dS_n/dt = F_n(S_{n-1}, S_n, S_{n+1}) = g_{n-1}S_{n-1} - g_nS_n$, where S is the quantity of matter and $g_n = F'_n(S_{n-1}, S_n, S_{n+1})$. At steady state, S_{n-1}: S_n: $S_{n+1} = 1/g_{n-1}$: $1/g_n$: $1/g_{n+1}$ (after Lotka[13]).

P, plants; D, decomposers; N, free nutrients; S, source; ES, external sink; T, toxins; →, direct positive effect; —○, direct negative effect; ⇒, flow of matter.

delaying their rate of decomposition[8]. The residue of recalcitrant compounds contributes to colloidal soil organic matter[8].

It is clear that secondary plant chemicals also have a role to play in deterring herbivores and pathogens in many situations. This role is not inconsistent with that of manipulating decomposers, and the evolution of one will also involve the other. Sometimes the dividing line between these two is not clear – a good example of ambiguity is the role of bark toxins in preserving the dead heart wood of a living tropical tree[3]. In other cases the linkage may involve a sequence in evolutionary time. Just as toxicity to vertebrates may originate as an incidental consequence of toxicity to invertebrate seed-predators[2], so also may toxicity to predators originate as an incidental consequence of toxic manipulation of decomposers.

The anti-predator interpretation of plant toxins is sometimes supported by citing the lack of evidence for any other direct metabolic importance to the adult plant[2,6]. However, there is evidence that toxic manipulation of decomposition is of direct ecological significance to the adult plant in its ecosystem. Consideration of all the evidence will, I think, change our estimation of the importance of anti-predator and anti-decomposer influences in driving the evolution of plant toxins, and assign a greater, and perhaps primary role to the effect on decomposition.

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Pulmonary edema in mice infected with *Plasmodium berghei*. Involvement of catecholamines[1]

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Summary. Mice inoculated with *Plasmodium berghei* developed a drastic and significant pulmonary edema. Treatment of animals with phenoxybenzamine rendered mice hyporeactive to this physiopathological alteration.

We have previously described that the lungs of mice inoculated with *Plasmodium berghei* showed dramatic edema beginning on the 4th day of infection[3]. Pulmonary edema in malaria has been reported in animal species other than rodents[4], and was described in humans more than 70 years ago[5]. Hyperactivity of the sympathetic nervous system has been observed in malaria[6], and it is known that catecholamines injected i.v. cause pulmonary edema in mice and rats[7]. Since it was shown that the phenomenon is inhibited by alpha-adrenergic receptor antagonists in rats[8], the present investigations were designed to examine the existence of any causative relation between catecholamines and pulmonary edema associated with malaria in mice.

Material and methods. Male mice of the Swiss-44 strain, weighing 18–20 g, were used for the study. The strain of *Plasmodium berghei*[9] used was the Pasteur strain[3]. The strain was maintained by successive inoculations, every 5 or 6 days, in Swiss-44 mice, by the i.p. route, with oxalated blood containing about 5×10^6 parasitized erythrocytes. The laboratory temperature was kept constant at 27 °C.

This work was conducted using 4 experimental groups: group I, normal animals injected with saline; group II, animals inoculated with *P. berghei* only (5×10^6 parasitized erythrocytes, i.p.); group III, animals injected with phenoxybenzamine in 3 separate doses of 1.0; 3.0 and 3.0 mg/kg at 1-h intervals, i.p., and group IV, animals injected with phenoxybenzamine as above and inoculated with the parasite 1h after the last dose of alpha-sympatholytic drug. Additional doses of 3.0 mg/kg of phenoxybenzamine were given each day from the 1st to 7th (the last day of infection) day to groups III and IV.

Each day, 5 animals of each group described above were anesthetized with sodium pentobarbital (40 mg/kg, i.p.) and killed by exsanguination through the aorta and inferior vena cava. The lungs were dissected free from trachea and weighed. Significant changes in lung wet weight/body weight ratios were considered to reflect pulmonary edema[10].

To verify if phenoxybenzamine could be interfering with the parasitemia of *P. berghei*-infected animals, thin blood films were obtained from infected animals only and phenoxybenzamine-treated and infected animals.

The thin blood films were stained with May-Grünwald-Giemsa and parasitemia was expressed as:

$$\text{Percentage of parasitemia} = \frac{\text{number of parasitized erythrocytes}}{\text{number of total erythrocytes}} \times 100$$

Statistical analysis was performed using Student's t-test.