The Paradox of Autotoxicity in Plants

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ABSTRACT: The production of self-inhibiting toxins by plants has been considered an evolutionary paradox. It has generally been assumed that autotoxicity is a nonadaptive consequence of toxic inhibition of interspecific competitors, predators or pathogens. However, autotoxicity may, in itself, be adaptive, improving escape from predators and pathogens, reducing intraspecific and intra-clone competition, increasing genetic diversity in some early successional populations, and reducing genetic contamination of highly r-selected populations by K-selected populations.

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Autotoxicity is a form of intraspecific competition resulting from liberation of self-inhibiting toxins. Autotoxins may be released from live plants by leaching, exudation or volatization, or from dead plant parts during decomposition. Autotoxicity generally causes reduced fitness or death in offspring. It has been demonstrated in successional annuals, biennials and herbaceous perennials (Abdul-Wahab and Rice 1967, Ballantyne 1962, Benedict 1941, Curtis and Cottam 1950, Guyot 1957, Jackson and Willemsen 1976, Keever 1950, Lodhi 1979, McNaughton 1968, Parenti and Rice 1969, Rice 1965, 1965a, 1975, Rasmussen and Rice 1971, Smith 1966, Wilson and Rice 1968), in shrub and tree species (del Moral and Muller 1969, Muller and Chou 1972, McPherson and Muller 1969, Webb et al. 1967), and in agricultural species (Bonner 1946, Bonner and Galston 1944, Borner 1956, 1959, Goodwin and Kavanagh 1949, Kimber 1973, Hirano and Morioka 1964, Martin 1950, Overland 1966, Patrick 1955, Pollock et al. 1954, Proebsting and Gilmore 1941, Tukey 1969, Welbank 1963). It is not possible to estimate relative abundance of autotoxic species in a given community, because extensive screening for autotoxicity has not been carried out.

Many studies suggest that toxin production can improve resistance to interspecific competition (Rice 1975), that it can confer protection against pathogens and herbivores (Bate-Smith 1972, Dethier 1970, Deverall 1972, Jacobson and Crosby 1971, Janzen 1969, 1970, Levin 1971, 1976), and that toxic compounds may be intermediates or waste products in metabolic pathways, apart from any role in pest resistance and competition (Jones 1979). A given toxin could have multiple functions (e.g., Davis 1928, and Gilbert et al. 1967). These studies suggest two general hypotheses concerning autotoxicity: (1) autotoxicity is an incidental correlate of metabolism and/or of defenses against pests and interspecific competitors, and is not itself of adaptive value; and (2) autotoxicity has adaptive value, although the toxins may have other functions as well. There is at present no basis for rejection of either hypothesis, because rigorous studies of population ecology, ecological genetics and biochemistry of autotoxic species are lacking. Nevertheless, the first hypothesis is often assumed to be correct: "Self-toxicity is an evolutionary paradox. One supposes that some selective advantage from production of toxic compounds outweighs the disadvantage of self-inhibition." (Whittaker and Feeney 1971, p. 750). The second hypothesis has

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received only two brief comments in the literature. Janzen (1970, p. 518) suggested that the self-inhibition demonstrated in an Australian rainforest species by Webb et al. (1967) could "serve as an efficient escape mechanism from very efficient predispersal seed predators that had great difficulty moving between adults." Jackson and Willemsen (1976) hypothesized that autotoxins produced by adults of Ambrosia artemisiifolia, an annual weed, may enforce seed dormancy. This species is largely restricted to very early stages of succession. Dormancy would increase the probability that viable Ambrosia seeds are present when a given site is disturbed and reopened. However, Lodhi (1979) found that autotoxins in Kochia scoparia, another early successional weed, did not induce seed dormancy. This paper presents additional hypotheses concerning the possible adaptive significance of autotoxicity in successional and climax species. Hypotheses 1-3 suggest that autotoxicity may arise through group selection (cf. Wade 1977, Wilson 1979); hypotheses 4-6 are based on individual selection. These hypotheses are presented not as a solution to the problem, but as a focus for discussion and research.

Hypotheses

1. Autotoxicity reduces predation and infection rates in annual weeds by causing rapid extinctions of local populations. Annual weeds may escape from predators and pathogens in both time and space by producing small, short-lived populations (cf. Dethier 1959, Harper 1977, Planck 1960). Efficient dispersal insures rapid colonization of newly opened sites. The more rapidly a local population reproduces, disperses its seeds and goes extinct, the lower the probability that predators or pathogens specific to it will be dispersed to that same site in time to establish a population. On a regional basis, rapid local extinction of plant populations could cause reduced predator and pathogen densities, further reducing the probability of their dispersal to newly established prey or host populations. Autotoxicity can cause more rapid local population decrease than would be predicted on the basis of interspecific competition alone (e.g., Keever 1950). Such rapid local extinctions due to self-inhibition might reduce the probability of regional or complete extinction due to widespread predator or pathogen outbreaks. Under such conditions, selection for autotoxicity could occur (cf. Van Valen 1971). Effective escape from pests could reduce selection for costly chemical defenses (Cates and Orians 1975, cf. Otte 1975). Local "extinction" in this case refers to elimination of the nondormant population; a population may persist on a given site as dormant seeds (Harper 1977).

2. Autotoxicity inhibits marginal, potentially "K-selected" populations on second-year sites, reducing gene flow to highly "r-selected" populations adapted to the environment of first-year sites. If a first-year annual species persisted in large numbers on a second-year site, high competition would favor K-selected genotypes. (cf. Gadgil and Solbrig 1972; Stearns 1976). Some of the seeds from this K-selected population would disperse to other second-year sites and to new first-year sites. A large proportion of the seed would remain dormant on the present site until a disturbance reestablishes first-year conditions (Harper 1977). On a regional basis, the relative frequency of K-selected genotypes would increase. In the long run, such a species might be displaced from first-year sites by a more r-selected, more autotoxic species.

3. Low levels of autotoxicity might increase the size of some first-year populations, thus increasing within-population genetic diversity. On those

first-year sites where population size may be primarily controlled by intraspecific competition, low-level autotoxic inhibition of growth rates in first-year plants might cause decreased between-plant resource competition. Slower growth rates would result in reduced per-plant resource utilization (cf. Parsons 1968) permitting larger population size, and thus, potentially, greater genetic diversity. High individual plant growth rates typically result in dominance of the population by a very few large individuals, with high mortality among the remaining suppressed plants (Harper 1977). Greater genetic diversity might increase the capacity of an established population to adapt to temporal and spatial heterogeneity in the environment, or to more effectively resist pest pressure (e.g. Planck 1960, Levin 1975). Many early successional species are inbreeders. However, genetic variability in inbreeding species is often high (Harper 1977). Slower growth rates could also result from endogenous chemical control (see Discussion).

4. Autotoxicity might reduce competition between offspring and parents, increasing reproductive value of the parent. High densities of offspring around an iteroparous parent might, as the result of intraspecific competition for resources, reduce reproductive value of the adult (cf. Smith 1979). The reproductive value of such juveniles might be very low, because juvenile mortality in plants is typically high and is often due to density independent as well as density dependent stress. If, despite resources obtained in competition with the adults, juvenile mortality remained high, then the decreased reproductive value of the adults due to competition with juveniles might not be balanced by an equivalent total increase in reproductive value of the juveniles. Thus, competition between parent and offspring would reduce parent fitness. In this case, mean fitness would be increased by toxic inhibition of the seedlings immediately surrounding the adults. There is evidence that adults of some perennial plants produce toxins which kill offspring but not adults (McPherson and Muller 1969, del Moral and Muller 1969). Potential subtle effects of these compounds on adult growth and reproduction have not been investigated. A juvenile at a greater distance from a parent tree probably has higher reproductive value than those in close proximity, due both to reduced intraspecific competition and to reduced attacks from species-specific predators and pathogens (Janzen 1971, Fox 1972, Whittaker and Levin 1977, cf. Smith 1979).

5. Autotoxic inhibition of the centers of tightly-packed perennial clones could accelerate vegetative expansion and seed production at clone margins, increasing colonization of new habitat. Curtis and Cottam (1950) described hollow-centered clones in the prairie sunflower which resulted from autotoxicity. The consequences of death of central shoots for subsequent growth and reproduction of periferal shoots in the same clone have apparently not been studied. However, it is probable that elimination of intra-clone neighbors results in greater growth and reproduction of remaining shoots. For example, rhizome initiation and, to a lesser extent, seed production are increased as intraspecific competition is decreased in Tussilago (Bakker 1969). Seed dispersal in most plants is strongly leptokurtic (Harper 1977), so that most seeds fall near the parent. Thus, continued seed production by shoots in the center of a large clone might have little impact on colonization of new sites. The same argument holds for new rhizomes produced by central shoots. The old clone center would receive heavy seed rain from periferal shoots, permitting recolonization of the site as toxins degrade and as the distance from old center to active clone margin increases.

A similar argument could be applied to dense, non-clonal aggregations consisting of offspring around parents (cf. Smith 1979). This hypothesis and Hypothesis 4 would be mutually exclusive. However, there is as yet no evidence for autotoxic inhibition of adult plants within such aggregations.

6. Autotoxicity in climax species might decrease densities of seedling predators and pathogens (cf. Janzen 1970). High seedling densities in the neighborhood of the parent tree could result in increased densities of species-specific seedling predators or pathogens (Burdon and Chilvers 1975, Planck 1963). Larger predator or pathogen population sizes would increase the probability that more isolated seedlings, further from the parent, would eventually be attacked (Janzen 1971). Low density of juveniles would be particularly beneficial if herbivores are polyphagous (e.g., Miller et al. 1970) and show a density-dependent response to food plant species (cf. Sloane and Clarke 1973). It is these more isolated seedlings which might generally have highest probability of survival, independent of the probability of predator or pathogen attack, due to reduced intraspecific competition. Autotoxic elimination of seedling aggregations near the parents might further increase survivorship of these isolated seedlings. Autotoxicity could reduce the density of aggregated stands of juveniles without eliminating the pattern of aggregation itself. Thus, the observation that juveniles often cluster around adults (e.g., Hubbell 1979) does not necessarily imply an absence of autotoxicity.

Discussion

It is apparent that autotoxicity could potentially affect rates of succession, and structure of successional and climax populations and communities. However, few studies have dealt with the phenomenon, and none have provided sufficient information to determine whether it is of adaptive significance. Large-scale screening studies will be needed to demonstrate its frequency along environmental gradients. Comparative data on demography, rates of selection, probability of extinction, seed dispersal rates, seed dormancy and gene flow will be required for autotoxic and non-autotoxic species. Such data can be used to develop realistic models for preliminary tests of hypotheses. Descriptive and experimental studies can then be designed for definitive tests.

Autotoxicity has so far been considered as a form of exogenous chemical control: the toxins affect growth and mortality after they are released into the environment. Endogenous control of growth and mortality has been more extensively studied (e.g., Noodén and Leopold 1977) and is relevant to the problem of autotoxicity. Experiments on soybeans (Leopold et al. 1959, Lindoo and Noodén 1976 and 1977, Noodén et al. 1978) suggest that monocarpic senescence results not because the developing fruit preempt resources directly, but because the fruit produce substances which actively induce leaf senescence. Similar effects have been described in other monocarpic crop species (Dale 1959, Eaton 1931, Hildebrand 1883, Lockhart and Gottschall 1961, Molisch 1938, Wareing and Seth 1967), but comparable studies are not available for non-crop species.

In soybean, the senescence-inducing substance acts primarily on leaves and does not directly affect roots (Lindoo and Noodén 1977). Induction of leaf senescence could therefore reduce competition between fruit and leaves for water, mineral nutrients and root-synthesized growth hormones, perhaps improving seed set. Extensive leaf senescence occurs after the greatest gain in whole-fruit dry weight, but before final seed maturation. The developing

fruit (pods) are photosynthetic, and so might provide some of the carbohydrates required for seed maturation.

If endogenous chemical induction of senescence occurs in non-agricultural species it could represent a strategy which guarantees that the parental site is rapidly vacated after reproduction, increasing the probability of offspring establishment (cf. Foster 1978).

Some monocarpic plants show prolonged post-reproductive survival (e.g., Schaffer and Schaffer 1977), potentially maintaining pest populations which would otherwise decrease. This could increase pest pressure on offspring. More rapid post-reproductive death could reduce such inter-generation infection (cf. Hypothesis 1).

Sequestering or excretion of toxic substances could reduce or eliminate endogenous autotoxicity; however, release of the sequestered toxins after plant death, or excretion from live plants, could result in exogenous autotoxicity. The two processes could therefore be linked, although the probability of such a relationship would vary greatly, depending on the types of toxins involved.

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