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Population Processes in Plants and the Evolution of Resistance to Gaseous Air Pollutants

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Introduction

Human activities since the industrial revolution have resulted in considerable pollution of the earth's air, water, and soil, particularly in technologically advanced countries of the world. Pollution is manifested in many ways including alterations in the chemical composition of the atmosphere, and contamination of air, water, and soils by heavy metals, pesticide residues, and toxic chemicals associated with industrial activities and agriculture. The most obvious ecological effect of pollution stress is alteration of the species composition of biological communities through elimination of sensitive species or individuals (e.g., Hutchinson and Meema 1987). There is also evidence that populations of some species have responded to the ecological challenges presented by pollution by evolving the ability to grow and reproduce in contaminated environments (Bradshaw and McNeilly 1981). The best evidence for the evolution by natural selection of plant populations resistant to anthropogenic pollutants involves plants growing in heavy metal contaminated soils (Antonovics et al. 1971) and those that are tolerant to herbicides (LeBaron and Gressel 1982). There are fewer examples of evolutionary responses of plant populations to the effects of gaseous air pollutants, however, the widespread occurrence of genetic variation in sensitivity to air pollutants suggests that a potential for adaptive responses exists in many species (Roose et al. 1982; Hutchinson 1984; Pitelka 1988; Scholz et al. 1989). The purpose of this chapter is to identify features of the population biology of plants that may be important in determining whether evolutionary responses to gaseous air pollutants are likely to occur.

It is often tacitly assumed that observable differences in phenotype that distinguish populations and species are the products of natural selection. Demonstrating this, however, is not a trivial issue because natural selection is a complex process that is notoriously difficult to quantify directly, particularly in long-lived organisms (Endler 1986). Microevolutionary forces largely occur within populations and gradually convert individual genetic differences in fitness to variation among populations, races, and ultimately species. As a result, studies of natural selection require a population-level approach, involving ecological and genetic investigations. Although demonstrating that species level genetic variation exists for a particular trait (e.g., resistance to a pollutant) is of value if the goal is to breed for increased resistance, from an evolutionary perspective, it may tell us little about the potential for evolutionary response in contemporary populations. To do this it is necessary to demonstrate that heritable variation occurs *within* natural populations and that the variation can be transformed into fitness differences among genotypes over generations. If this occurs, natural selection of the trait exhibiting genetic variation will inevitably occur resulting in increased adaptedness of the population.

The nature of evolutionary change depends on the amounts and kinds of genetic variation within natural populations as well as the intensity and type of natural selection that occurs. In addition, however, patterns of evolutionary diversification are also influenced by the intrinsic features of the population biology of organisms. Plant populations exhibit an enormous diversity of life history and of demographic and reproductive characteristics (reviewed by Harper 1977; Solbrig 1980). This variation plays an important role in controlling the organization of genetic variation within and among populations and hence the type of selective responses that occur. Simple selection models developed for many animal populations that assume diploid, unisexual, outbreeding populations of large effective size with nonoverlapping generations are largely invalid for most plant species because of their distinctive features. Restricted recombination systems involving high degrees of inbreeding, limited gene flow, small effective population size, large dormant seed banks, clonal propagation, and high phenotypic plasticity make it difficult to generalize about the outcome of selection in plant populations (Levin 1978; Crawford 1984). Features of some plant populations such as a small effective size and the large environmental component to fitness variation can reduce the intensity of selection and increase the likelihood that genetic drift will be a dominant force in influencing population genetic structure. Although random processes are unlikely to retard the progress of selection where intensities are very high (e.g., in heavy metal contaminated sites), they may be of more significance where selection pressures are weaker and intermittent in nature such as in the case of some gaseous air pollutants.

The main objective of this chapter is to address two general questions that pertain to population-level processes governing the evolutionary responses of plants to gaseous air pollutants. First, how might variation in the life history attributes and reproductive systems of populations facilitate or constrain selective responses to pollution stress? Second, what features of population genetic structure are likely to be important in determining whether resistance to air pollution will evolve and what genetic consequences might result from such a change? While empirical data demonstrating the selective effects of air pollutants are limited, wherever possible the largely intuitive arguments presented here will be buttressed with information gleaned from case studies from the air pollution literature. Since the harmful ecological effects of air pollution on forest communities have been of major concern in recent years, particular attention is given in the discussion to the ecological genetics of tree species and how the population biology of long-lived organisms is likely to effect genetic responses to air pollution.

Life History Characteristics

The interaction of life history variation with anthropogenic pollutants has perhaps no greater relevance than with respect to air pollution stress. Atmospheric pollution is so widespread that no habitat may be entirely protected and consequently all plant life forms are likely to risk exposure to air pollution. This contrasts with the exposure of plants to heavy metal contamination, in which case the contaminated habitat is often discrete, being confined to the boundaries of mining areas or other point sources of metal contaminants. Only a limited number of species can survive on soils contaminated with heavy metals, and the vast majority of cases discovered to date have involved herbaceous perennial plants. An even more restricted subset of plants has evolved herbicide resistance (e.g., weeds of annuals crops) and their life histories represent only a small sample of the existing diversity.

The range of variation in plant life histories adds considerable complexity to attempts to assess the potential for evolution of resistance to air pollution. Longevity in plants covers the spectrum from short-lived annuals to perennials that may live many hundreds of years. This variation creates practical problems in assessing the effects of air pollution stress on life time fitness and makes comparisons among groups with contrasting longevities particularly difficult. A further level of complexity is introduced by variation in the frequency of sexual versus asexual reproduction, within and among plant populations. This character is itself often very plastic but is critical for assessing rates of evolution in populations. Furthermore, there is an array of mechanisms for achieving asexual propagation, by corms, bulbs, rhizomes, and stolons and by fragmentation of stems, runners, and tillers. It seems likely that these contrasting clonal regenerative strategies will vary in the extent to which they are susceptible to air pollution stress, just as we might expect differences among other ecological and life history groupings. However, predicting which sets of life history traits are more likely to confer resistance is a formidable task without comparative experimental work or surveys of the response of plant communities to pollution-induced stresses.

Given the diversity of life history traits in plants it is relevant to ask whether Grime's classification of adaptive plant strategies (Grime 1979) can be employed when considering plant responses to air pollution stress. Grime's initial divisions placed plants into one of three primary categories: ruderals, competitors, and stress tolerators. Further subdivisions into secondary strategies include plants adapted to environments at the interface of each of these three extremes. The importance of Grime's work in the context of responses of plants to air pollution stress is that each strategic category contains plants united by common features of morphology, life history, and physiology. Although this simplifies considerable complexity it does allow us to evaluate whether traits that are believed to be important as adaptations to natural stress conditions may also confer some degree of resistance to anthropogenic stresses, and conversely whether traits associated with disturbed or competitive environments may be disadvantageous in polluted environments.

This section first discusses several features of plant life histories that are relevant to the potential for evolutionary responses to air pollution stress. Where appropriate, traits that enable populations to avoid pollution stress are contrasted with those that enhance exposure, thereby intensifying selective pressures for the evolution of resistance within populations. The section ends with a discussion of plant responses to air pollution in the context of Grime's theory of adaptive strategies. The response of trees to pollution stress is selected for special consideration since certain features of the population biology of trees pose unique problems for the assessment of evolutionary change.

Seed Banks

Seed banks are a feature of many plants, particularly those that occupy environments which are frequently disturbed or exhibit a high degree of environmental uncertainty. Seeds of most species experience transient dormancy, however of primary concern here are those that are capable of developing a persistent seed bank, which by definition is one in which seeds remain viable for a period of a least 1 year in the soil. From an evolutionary perspective seed banks can be considered as sources of migration from the past. Templeton and Levin (1979) have modelled the effect of a seed bank in an annual population on selection for a single locus trait with two alleles. In their model, mating is assumed to be random in a population of infinite size, with overdominant (heterozygote advantage) fitness and a selection coefficient of 0.01 (weak selection). The main conclusions of Templeton and Levin's study are (1) seed banks decrease the rate of approach to equilibrium allele frequencies and hence the rate of evolutionary change. The degree to which the rate of allele frequency change is reduced increases with the average number of generations that germinating seeds spend in the seed pool. (2) Differences among species in seed viabilities, germination probabilities and total seed production result in different rates of evolutionary change. From their work it is evident that an increase in the mean number of generations in the seed bank from 1 to 2 years reduces the rate of evolution by half. Since the presence of a seed bank does not influence equilibrium allele frequencies, similar conclusions can be drawn for the case of directional selection, a situation more likely to occur with air pollution resistance (see below). In this case selection should lead to fixation of the selected allele, with rates to fixation decreasing with increasing time in the seed bank. One caveat worth considering is that resistance to air pollutants is more often a quantitative trait (Roose et al. 1982) than one controlled by a single major gene (although see Engle and Gabelman (1966) for a possible example of single gene control). With quantitive inheritance of resistance, rates of evolution are expected to be generally slower with persistent seed pools enhancing this effect.

With this basic model in mind other factors relevant to rates of evolution of resistance to air pollutants can be superimposed, such as whether or not the population is declining in number versus growing. The effective memory of the seed pool increases in populations that are in decline. Alternatively, the effect of a growing population is to reduce the memory of the seed pool, as seeds from past generations are swamped by the numbers of seeds produced by the larger population of recent years. Plant populations may experience an initial decline with the onset of pollution stress as susceptible individuals are eliminated. In this initial phase the contributions of the prepollution population to the seed bank will retard the evolution of a resistant population. However, if a second phase of rapid growth and expansion of the population occurs, particularly as more sensitive competitors are eliminated, the seed pool may no longer retard the rate of evolution of resistance to the same extent.

It is also possible that different contributions to the seed pool by plants of different size could have a similar effect. It is well established that the correlation between plant size and reproductive output is in the order of 0.90 for annuals (Heywood 1986). If individuals more resistant to pollution have significantly greater growth and subsequent seed production than sensitive individuals, then the tendency for seed pools to delay the evolution of resistance would be ameliorated. This would be the case if the reported differences in foliar injury and yield reductions of tolerant and sensitive genotypes under short-term pollutant exposures (reviewed in Roose et al. 1982) are translated into significant size differences over greater lengths of time. The significance of seed banks to rates of evolutionary response is likely to be less in perennial plants, particularly those of considerable longevity (e.g., forest trees). Dormancy periods tend to be relatively short in many tree species and in some tropical species may be absent altogether. However, perenniality itself may retard evolutionary change in much the same way as seed banks, since both create opportunities for matings between generations.

Phenotypic Plasticity

One of the primary distinctions between most animals and plants is the role that apical meristems play in permitting continual embryological development of the plant (White 1984). The plastic responses of the growth and morphology of plants to external environmental conditions is derived from this meristem potential. There is considerable evidence indicating the importance of plasticity in plant responses to air pollution. Pollution stress may elicit both qualitative and quantitative changes in the allocation of rsources to plant tissues and organs (reviewed by Lechowicz 1987). Both SO_2 and O_3 tend to suppress root growth more than shoot growth, thereby reducing rootshoot ratios. Allocation of resources to leaves tends to increase under SO_2 pollution whereas that to stems decreases. Reproductive investment is often reduced under pollution stress leading to a variety of responses (see below). Many other examples of plastic responses are reported throughout the air pollution literature, some of which are clearly under genetic control.

It has been suggested that phenotypic plasticity, developmental homeostasis, and genetic polymorphism represent alternative strategies for dealing with environmental unpredictability and heterogeneous environments (Jain 1979). Polymorphism has been thought to be adaptive where heterogeneity is manifested strongly in spatial aspects of the environment (e.g., mosaic soil conditions; see Snaydon and Davies 1972). Environmental heterogeneity is relatively predictable in this case, permitting adaptive gene frequency changes to occur over time. Plasticity and homeostasis, on the other hand, have been thought of as adaptive responses in environments characterized by unpredictable short-term changes that are manifested within the life cycle of individuals and which require immediate responses. Although it is often suggested that an inverse relationship between plasticity and heterozygosity may exist, a recent review of phenotypic plasticity in plants casts doubt upon this dichotomy (Schlichting 1986). Some studies have found an inverse relationship between heterozygosity and phenotypic plasticity, others have failed to corroborate this relationship. As Schlichting (1986) points out there is no apparent reason why the presence of genetic variability in a population should oppose the evolution of appropriate plastic responses unless there is an extra cost to an organism that is both plastic and heterozygous. As yet there is little empirical evidence for such a cost.

Plastic responses may be adaptive in the short term, but it is questionable whether plasticity alone could sustain growth and reproduction in the long term in populations subjected to chronic pollution stress. Plasticity could be a costly stress response involving increased expenditure to compensate for the decreased efficiency of photosynthesis through foliar injury. If pollution stress is persistent and severe enough, the plant may overextend its ability to maintain itself, resulting in greater susceptibility to pest and disease pressures (Lechowicz 1987). Furthermore, although plasticity can be selected, there are developmental and architectural constraints on the evolution of plasticity (Watson and Casper 1984). Therefore, although a high level of plasticity may help compensate for short-term pollution stress, it is unlikely to provide a mechanism of resistance in evolutionary terms. Populations with a high degree of phenotypic plasticity may, however, be just as likely to exhibit genetic variation for resistance as those that are less plastic.

Life Forms

The classification of Raunkiaer (1934) provides us with a means of evaluating life forms according to their potential susceptibilities to abiotic stresses (Hutchinson and Harwell 1985). With respect to air pollution, we can rank plants based on the degree to which phenology, architecture, and growth involve protection of sensitive meristems from gaseous air pollutants. Life forms whose perennating buds or shoot apices are borne well above ground (phanerophytes) are afforded little protection from exposure to air pollution. In addition, these life forms are often canopy species which form the first zone of pollutant capture (a characteristic exemplified by trees). The chamaephytes and hemicryptophytes are more capable of avoiding pollution stress since their sensitive meristems are borne either close to, or at ground level, and their above ground shoots senesce at the onset of unfavorable growing seasons. Perennials whose meristems are either protected underground as rhizomes, bulbs, or tubers (geophytes) or underwater (helophytes and hydrophytes) are probably best able to avoid direct meristem damage from atmospheric pollutants, however, the dissolution of air-borne pollutants into aquatic environments may constitute another form of pollutant stress.

The importance of meristem position is clearer if we consider the interaction of phenology with seasonal cycles of pollution. In parts of Europe pollution episodes can be most intense during the winter months (e.g., SO_2; see Venne et al. 1989). Life forms with subterranean organs will therefore avoid pollution stress. Dormancy of above ground buds and the absence of a canopy in deciduous trees may also restrict injury to growing plant organs. In parts of North America, some air pollution is greatest during the summer months (e.g., ozone pollution, acid advection fog). Where this occurs it can coincide with periods of peak plant growth. Therophytes (annuals) that complete their entire life cycle in a short time span may be under intense selection pressures from pollution stress at each developmental stage of the life cycle, from germination to reproduction. It is also worth considering the possibility that tropical plants, which often lack a dormant growth phase, may suffer from continual exposure to atmospheric pollutants, and that this susceptibility is compounded by the predominance of the phanerophyte life form in the tropics (Raunkiaer 1934).

It follows from the discussion above that clonal populations may be more resistant to air pollution stress than populations which rely exclusively on seed reproduction. The lower risk of mortality to vegetative offspring, whose growth can be sustained under adverse conditions by mobilization of resources from older ramets, may enable clonal growth and regeneration under conditions too stressful for establishment from seed. The storage reserves available to seeds at germination are usually much less than those available to their vegetative counterparts and, furthermore, the more rapid growth rates of seedlings may result in greater rates of pollutant absorption. However, if pollution levels vary greatly within plant communities, according to their structural complexity, it is possible that larger vegetative offspring (e.g., suckers and offshoots in trees) represent more extensive targets. In this situation seedlings may be afforded more protection by surrounding plants because of their small size. The relationship between plant size and susceptibility to pollution stress is probably not a straightforward association, but may depend more on the spatial and temporal location of growing points in relation to the timing and nature of pollution episodes.

Plant Strategies and Pollution Stress

Earlier in this section Grime's (1979) concept of adaptive plant strategies was introduced as a possible framework for predicting the stress responses of different plants to air pollution. This approach may be of value since the potential for evolution of pollution resistance is unlikely to be independent of other selective forces in the environment. The most important issue is whether life forms already adapted to surviving in stressful habitats will be "preadapted" to tolerating air pollution stress. This could be achieved through stress avoidance by means of slow growth rates, infrequent flowering, and the capacity to shutdown photosynthesis under adverse conditions. Other traits characteristic of stress-tolerators include perenniality, longevity of leaves and roots, and the ability to sequester resources. These may also provide plants with the resilience to endure periods of stress. However, several traits (e.g., longevity, infrequent reproduction) that may enhance resistance also delay rates of evolutionary change in populations and hence stress tolerators as a group may be slow in responding through genetic changes at the population level to air pollution stress.

Competitors are adapted to habitats with high productivity. Accordingly, they exhibit high rates of growth and resource acquisition, traits that enable them to compete effectively in dense stands of vegetation. As vegetation closes during the growing season they respond to increasing competition by rapid and continual readjustment of their absorptive surfaces. This involves large changes in reinvestment of their captured resources. Whether this kind of growth response can be maintained under chronic pollution stress is questionable. It seems likely that resource reserves would be rapidly exhausted, thus decreasing competitive ability and possibly increasing susceptibility to pest or pathogen attack. Since flowering is usually delayed until after periods of maximum growth, it may be suppressed if resource acquisition is compromised. This factor, and restricted opportunities for seedling establishment in productive habitats, both suggest that rates of evolutionary change in populations of competitors may be slow. Under stressed conditions allocation of resources to storage organs and to vegetative expansion may be at the expense of sexual reproduction.

The response of ruderals to stress conditions is markedly different from that of either competitors or stress tolerators. Many ruderals are annuals, and one adaptation to disturbed habitats is rapid completion of the life cycle. Onset of flowering occurs early in the growing season and there is usually a large investment of resources into seed production. Response to unfavorable conditions often includes the capacity to sustain limited seed production even under conditions of severe stress. It is here then that one might expect to find fertile ground for evolutionary change. The annual production of great quantities of seed in combination with good opportunities for seedling establishment are likely to provide the raw material on which natural selection can act. If the growing season coincides with periods of high pollution stress, this will serve to intensify selection pressures. It is also worth considering that the open structure of disturbed habitats may render ruderal species particularly vulnerable to exposure to air pollution. Furthrmore, if large areas of woody vegetation are destroyed through chronic air pollution and are replaced by open environments largely devoid of plant cover, it is likely to be ruderal species that rapidly colonize these new environments because of their high dispersability and rapid reproductive rates.

Colonizing populations may initially suffer considerable pollution induced mortality, however, the high selection pressures that this involves are likely to lead to the rapid evolution of resistant populations, particularly in species with short generation times. Although levels of genetic variation in many annual colonizers of disturbed environments are low in comparison with longer-lived plants (reviewed in Barrett and Shore 1989), sufficient genetic variation is likely to occur to enable evolutionary response to air pollutants, particularly where tolerance is polygenically controlled (see below). Two of the case studies on the evolution of populations resistant to air pollution involve ruderal species (*Geranium carolinianum*, see Taylor and Murdy 1975; *Lepidium virginicum*, see Murdy 1979).

Trees and Pollution Stress

Much attention has been focused on the impact of pollution stress on trees because they are highly visible components of ecosystems and because of their economic importance. Among the great diversity of tree life histories can be found species that fall within two of Grime's three primary strategies (see Fig. 18d in Grime 1979). Therefore, rather than discuss attributes of woody plants in the context of his theoretical framework, it is simpler to consider what particular genetic and ecological features of tree populations are likely to influence evolutionary responses to air pollutants.

Trees as a group contain higher levels of genetic variability than most other plant groups (Hamrick 1979; Ledig 1986). Therefore, it seems unlikely that the evolution of pollution resistance will be constrained by lack of appropriate genetic variation. Furthermore, unlike some annuals and short-lived perennials population sizes in many tree species, particularly the dominant species of temperate forests, can be very large, thus reducing effects of genetic drift. Large effective population sizes provide greater opportunities for selection response. Thus, it seems unlikely that stochastic forces will have the same influence in trees as in many annual colonizing species; unless population sizes are notably small or reduced dramatically by pollution-induced mortality. While the genetic potential for evolutionary response is likely to be present in populations of many tree species, the time scale and the dynamics of change are likely to be much slower than in herbaceous plants. Since trees are largely outcrossing the persistence of resistant genotypes, particularly when rare, may be short-lived since recombination will reshuffle tolerance genes into different genetic backgrounds during each mating cycle. The tempo of evolutionary change will be retarded if gene complexes conferring resistance need to be assembled from different individuals in the population and then refined and improved by selection. This constraint will be less severe, however, where many individuals contain resistance genes and levels of selective mortality are high.

Another potential constraint on evolutionary response to air pollution concerns the nature of recruitment patterns and the degree of spatial and temporal environmental variation in many forest environments. "Seedling banks" are a common feature of late successional tree species with growth rates of seedlings often extremely slow and mortality levels high. If conditions determining seedling mortality are different or more varied than those determining adult survival, then seedling populations will not necessarily be "preadapted" to the selection pressures they encounter as adults. For example, if pollution stress is only one of many factors contributing to seedling mortality, then surviving seedlings will not necessarily be the most resistant to pollution. Likewise, seedlings carrying resistance genes may succumb to other stresses. These factors will retard the evolution of resistance since the fate of resistance genes in the population will depend on the many factors influencing seedling survival.

The major factor influencing the nature of evolutionary change to air pollutants in tree populations is their great longevity. Since generation times of many tree species involve decades or centuries rather than years, it seems likely that most of the genetic effects that are likely to occur within populations will result from viability selection against sensitive genotypes. This process gives rise to populations composed of adults, that through possession of superior genotypes, are able to tolerate pollution stress. Although recombination and repeated mating cycles can potentially lead to improved resistance this is likely to occur over very long time scales in most tree species. Because of this our ability to measure evolutionary changes, resulting in increased adaptedness to pollution stress within tree populations, will be severely limited in comparison with most herbaceous plant populations. This difficulty may account for the recent interest in attempts to reveal fertility selection and gametophytic fitness differences in forest trees in response to pollution stress (Cox 1989; Venne et al. 1989).

Reproductive Systems

Sexual reproduction in flowering plants consists of flowering, pollination, fertilization, seed maturation, and dispersal. These events occur consecutively and the imposition of stress during any or can result in loss of reproductive potential with consequences for plant fitness. Most studies on the effects of air pollutants on plants have investigated vegetative parts with a particular emphasis on the relationships between foliar injury, photosynthetic rates, growth, and productivity (reviewed in Heath 1980). Far less work has been conducted on the influence of pollution stress on sexual reproductive processes despite the importance of this stage in the life cycle in relation to genetic transmission and population genetic structure. One of the problems in studying the effects of pollution on sexual reproduction, in contrast to vegetative growth, is that the events involved are complex, sensitive processes that often occur over limited time periods. As a consequence it can be extremely difficult to pinpoint which stages in the reproductive cycle are responsible for losses in reproductive potential, particularly where experiments are conducted under field conditions.

As discussed earlier a common reproductive response to stress factors is a curtailment of resources allocated to reproduction. This plastic response is usually manifested by either the inhibition of flowering or the abortion of buds, flowers, fruits, or seeds. The particular response will depend on the timing, duration, and intensity of stress (Lloyd 1980; Stephenson 1981). Where air pollution levels increase gradually, causing inhibitory effects on growth, the most likely effect on reproduction will be a reduction in levels of flowering, particulary in perennial plants (Bonte 1982; Ernst et al. 1985;

Lechowicz 1987; Taylor and Bell 1988). Although there are many reports of the stimulation of flowering by stress conditions, this often presages early mortality as a consequence of the cost of reproduction in individuals already severely limited by lack of resources. Where pollution is episodic in nature and populations become exposed to toxic levels during reproductive activity, responses are likely to be considerably more complex. They may involve effects on gametogenesis causing sterility of pollen and ovules as well as impaired reproductive function causing reductions in pollen viability, pollen germination, fruit and seed set (Houston and Dochinger 1977; Murdy 1979; DuBay and Murdy 1983; Cox 1988a,b). While there are a growing number of reports of the detrimental effects of air pollution on plant reproduction under field conditions, in many cases it is unclear which stage(s) in the reproductive process are responsible for observed reductions in fertility.

Pollination Systems

The transfer of compatible pollen between conspecific individuals is a critical stage in the mating cycle of outcrossing plants. The specific pollen vector(s) involved (e.g., wind, animals, water) depends on the pollination system of the species (reviewed in Faegri and van der Pijl 1971). In plants pollinated by animals (e.g., bees, flies, butterflies, birds, bats), there is the possibility that in heavily polluted areas the acute toxicity of pollutants will have a direct effect on the composition of the pollinator fauna. This could result in a reduced frequency of visits or loss of particular pollinator species. Such an effect seems unlikely for low-level regional air pollution but could be significant in highly polluted urban and industrial areas or at sites in close proximity to point sources. Tropical forests would be especially sensitive to such effects since most tree species are animal pollinated and outcrossing. Air contaminants are unlikely to directly influence pollen transport in wind-pollinated species (Smith 1981), but have been reported to be noxious to some pollinating insects causing reductions in their number (Bonte 1982). Reduced frequencies of pollinator visits may not necessarily lower fruit and seed set, since in many zoophilous plants reproductive output is apparently not pollen limited (Willson and Burley 1983). However, qualitative and quantitative changes in the pollinator fauna may have more subtle influences on mating through alterations in foraging behavior and its influence on male fertility and patterns of gene flow. Such effects may not be detectable by monitoring patterns of seed production in populations and may require the use of genetic markers (Brown et al. 1985).

Flower orientation may also be of significance in affording protection against certain types of air pollutants, particularly those that dissolve in rain. Tubular or bowl-shaped flowers that are held in an upright position may fill with rain, whereas those that are pendulous are not effected in this way. Related species of *Primula* with contrasting flower orientations possess pollen that respond differently to immersion in water (Eisikowitch and Woodell 1974). This suggests that chemical adaptations preventing germination may occur in species with exposed flowers. Other features of floral biology such as the phenology of flowering, the timing of stigma receptivity, and anther dehiscence, and the shape, structure, and orientation of reproductive parts are likely to influence the degree of sensitivity to air pollutants displayed by individual species (Cox 1984). In addition, the physical location of species in a community (e.g., understory, overstory) and hence the degree of protection they obtain from neighboring species may be important in modifying the impact of air pollutants on reproductive processes. Field studies that take into account the ecological heterogeneity of natural communities are needed to assess the variations in reproductive responses that are likely under natural conditions.

Pollen-Pistil Interactions

Following the deposition of pollen on stigmas of conspecific plants, a series of complex interactions occur between the male gametophyte and tissues of the maternal plant. The interactions are susceptible to a variety of stress factors, and it is known that pollen germination and pollen tube growth are among the more sensitive indicators of atmospheric pollution (Feder 1968; Stanley and Linskens 1974; Cox 1987). Wolters and Marten (1987) have reviewed in detail the effects of air pollutants on pollen biology and document numerous studies involving different pollutants (e.g., SO_2, O_3, CO_2, NO_2) and plant taxa. From these studies it is clear that pollen viability, pollen germination, and pollen tube growth can all be negatively affected by air pollutants both in vitro and in vivo. Although observed patterns vary with environmental conditions, species, and pollutant, in vivo pollen germination and pollen tube growth is usually more tolerant to air pollutants than under in vitro conditions. This difference is thought to reflect the extra buffering capacity of the stigma surface and the protection given by maternal tissue once pollen tubes have entered the style.

The extent to which pollen-pistil interactions are influenced by air pollutants in particular species is likely to depend on specific features of their compatibility systems, the size, structure, and cytochemistry of stigmatic surfaces, and the time over which pollen-pistil interactions normally take place. In species in which individual flowers last for several weeks, stigmatic receptivity and pollen tube growth are often of extended duration. By contrast, in flowers in which the anthesis periods last for less than a day, pollen germination, pollen tube growth, and fertilization often occur within hours of pollen deposition on the stigma. Primack (1985) has reviewed the literature on floral longevity and provides ecological and evolutionary explanations for the patterns observed. From the perspective of air pollution it would seem likely that the longer pollen remains on the stigma before germination and penetration of stigmatic tissues, the more vulnerable it may be to the effects of chemical modifications of the stigmatic secretion by air pollutants. Species with stigmas of large surface area (e.g., many wind-pollinated plants and also *Oenothera parviflora*; see Cox 1984) may be more susceptible to pollution-induced changes in pollen-stigma interactions than taxa with small stigmas. Comparative studies of the effects of air pollutants on the pollination process in closely related species with contrasting floral traits would be required to evaluate these ideas.

A particularly provocative suggestion concerning pollen-pistil function is the possibility that pollution-induced stresses may alter microgametophytic selection leading to fitness effects in the sporophyte generation (Mulcahy 1979; Searcy and Mulcahy 1985; Wolters and Martens 1987). Searcy and Mulcahy (1985) found that there was parallel expression of metal tolerance in pollen and sporophytes of two *Silene* spp. and in *Mimulus guttatus*. Pollen from metal-tolerant plants was able to germinate and grow in vitro at concentrations of metals which markedly inhibited the pollen from nontolerant individuals. They suggested that if tolerance is due to genes expressed in both the diploid sporophyte and the haploid microgametophyte, it could result in the rapid development of populations resistant to heavy metals. A different evolutionary scenario was envisioned by Cox (1984), who suggested that microgametophytic selection favoring pollen tolerant to the low pH conditions associated with acid precipitation might be selectively advantagous if sporophytes producing the pollen were locally adapted to acidic soils, but disadvantageous where they occur on calcareous soils.

Evolutionary changes through selection at the gametophytic stage of the life cycle of plant populations require a number of conditions to be met. First, pollen loads on stigmas following pollination need to be sufficiently high to enable microgametophyte competition to occur. The most intense competition will occur where ovule number per flower is low and pollen loads on stigmas are high. This condition occurs in many angiosperms but probably rules out the conifers since the number of pollen grains per ovule is normally less than five in most groups examined (Venne et al. 1989). Second, pollen genes must be expressed both post-meiotically and in the sporophyte generation resulting in a positive correlation between gametophytic and sporophytic vigor. There is some evidence for this overlap in gene expression (Tanksley et al. 1981; Willing and Mascarenhas 1984). Finally, for evolutionary changes to occur, heritable differences in pollen performance must occur *within* natural populations. Although many studies have demonstrated selective fertilization on the basis of pollen genotype (reviewed in Marshall and Ellstrand 1986; Snow 1986), few have examined the heritability of pollen performance. In one of the only studies of plants obtained from within a natural population, Snow and Mazer (1988) found no evidence in the wild radish (*Raphanus raphanistrum*) of heritable variation in pollen competitive ability. If a gene which greatly accelerated pollen tube growth arose in a population it would rapidly spread to fixation (Haldane 1932). However, genetic variation for pollen tube growth rate could exist within natural populations if there was a negative genetic correlation between the performance of the pollen and that of the zygotes which it produced. Thus, variation could be maintained if selection occurred in opposite directions in the haploid and diploid phases of the life cycle. Unfortunately, it is not yet clear whether evolutionarily significant amounts of gametophytic selection occur within natural populations, because as yet there is little evidence for heritable variation in pollen competitive abilities. Although it seems reasonable to assume that pollen competition occurs frequently in nature, it is not yet known whether this has led to fixation of genes that influence the fitness of sporophytes (Charlesworth et al. 1987).

Mating Systems and Gene Flow

Mating patterns in plant populations are largely governed by their breeding systems and the nature and magnitude of gene flow within and between populations. This stage in the reproductive cycle is of major importance in determining the genetic structure of populations and their potential for evolutionary change (Richards 1986). Roose et al. (1982) have suggested that outbreeding species are more likely than inreeding species to evolve resistance to air pollution stress. This suggestion follows from the observation that populations of outbreeders usually contain greater stores of genetic variation than inbreeders (Stebbins 1957; Jain 1976). Thus, it would be anticipated that greater opportunities for selection responses occur in outcrossing populations if they contain greater genetic variability for air pollution resistance. Although this suggestion seems reasonable on the basis of experience with the evolution of heavy metal tolerance, there are a number of reasons why we should not discount the possibility that inbreeding species may also rapidly evolve resistance to air pollutants where appropriate environmental conditions occur.

A larger proportion of the total genetic variation in inbreeding species is distributed among populations rather than within populations as is found in outbreeding species (Loveless and Hamrick 1984). However, the total amounts of genetic variation in species with contrasting levels of inbreeding versus outbreeding tend to be broadly similar. Furthermore, models of quantitative genetic variation, under different systems of mating, indicate that even in species which practice considerable inbreeding, high rates of mutation at loci controlling polygenic traits generate considerable amounts of quantitative genetic variation (Lande 1977). This variation is sufficient to allow rapid adaptive responses and considerable phenotypic divergence (Lande 1980).

Since air pollution effects are often regional in nature, with diffuse boundaries, large numbers of populations of a given species are likely to be exposed to altered selection pressures. This form of mass selection in inbreeding species may result in a greater likelihood that some populations will contain the necessary variation in comparison with cases of localized pollution stress. Where heavy metal contaminated soils or emissions from a single point source occur, relatively small numbers of individuals are usually exposed and, in these circumstances, absence of genetic variation within populations may frequently limit evolutionary responses. Under these circumstances the population genetic structure associated with outbreeding species would be more likely to maintain the necessary variation for selective responses. However, with regional air pollution selection responses may be extensive rather than localized, providing opportunities for interdemic selection in inbreeding species.

Another difference between the evolution of resistance to air pollution in comparison with heavy metals concerns the selective regime and type of population processes that are likely to occur. Because of the localized nature of mine wastes, a major force retarding the evolution of heavy metal resistance is the extent of gene flow from neighboring environments containing nonresistant plants. Indeed, it has been argued that resistant populations of some grass species have evolved reproductive isolating mechanisms in the form of altered flowering times and self-fertilization. These are believed to have developed in response to the disruptive effect of gene exchange and its effect in breaking down adaptive gene combinations responsible for resistance (Antonovics 1968). In the case of regional air pollutants, however, gene flow from sensitive or "unselected" populations is unlikely to be of major importance unless pollution sources are highly localized. In this case, wind-pollinated populations, particularly of tree species, are most likely to exchange genes over long distances and selfing populations are the least likely to do so. Since gene flow in flowering plants, irrespective of breeding system, is most often rather localized (Levin and Kerster 1974) it seems unlikely that genes for resistance to air pollution will be prevented from spreading within local populations owing to high levels of gene exchange with populations from unpolluted areas.

One of the likely population-level responses to severe air pollution stress is an alteration in the size and density of plant populations. Concomitant with increasing levels of stress is the selective mortality of individuals and species. Some cases of forest decline in Europe, for example, have resulted in dramatic reductions in population numbers of many forest tree species, resulting in open stands composed of small numbers of individuals. Similar patterns are evident in various parts of North America where high levels of pollution-induced mortality have resulted in sparsely vegetated areas containing few surviving plants. In these situations, resistant genotypes of short-lived species may soon multiply and population sizes have the potential of returning to their original levels, or even increasing in size, if few species can tolerate the polluted environment (e.g., *Deschampsia cespitosa* at Sudbury, Ontario; Cox and Hutchinson 1981). However, in longer-lived plants this process is likely to be considerably slower, giving rise to small populations composed of scattered individuals. In addition to increasing opportunities for genetic drift, this type of population structure could have potentially important consequences for the mating systems of survivors.

In small populations, particularly those at low density, levels of inbreeding may increase through self-fertilization or because of matings between related individuals. Where inbreeding occurs in normally outcrossing taxa, this usually leads to reduced fitness of progeny (Charlesworth and Charlesworth 1987). Under stress conditions, differences between offspring that result from outcrossing and selfing are usually magnified. In polluted environments, fitness decline may occur when survivors have descended from a small number of resistant individuals. In species incapable of self-fertilization, owing to self-incompatibility or dioecism, low density can potentially result in significant reductions in the reproductive output of individuals. These conditions may favor the evolution of self-fertilization, particularly in herbaceous groups (Baker 1955; Jain 1976; Lloyd 1979). This adaptive shift in mating system seems unlikely for most long-lived organisms, such as trees, because of their high genetic loads and the complex environments which they occupy (Ledig 1986). However in short-lived species, selfing may develop as a consequence of low density conditions in polluted environments and act secondarily as a mechanism for maintaining gene complexes that confer resistance to pollution stress.

Population Genetic Considerations

The evolution of resistance to air pollutants in plants is influenced by features of the life history and reproductive biology of individual species. This is because the ecological and demographic characteristics of populations have an important influence on population genetic structure, and, in addition, mating systems regulate patterns of genetic transmission and levels of recombination. Whether resistance develops in plant populations ultimately depends, however, on the presence of heritable variation for the ability to grow and reproduce in environments affected by air pollution. Genetic studies of the inheritance of resistance to air pollution are rudimentary but what little data is available indicate that resistance usually behaves as a quantitative trait governed by many genes with additive effects (Taylor 1978; Roose et al. 1982). If this turns out to be the case in most plants, then the evolution of resistance reduces to the problem of directional selction on a quantitative trait and the major empirical issues concern whether or not experiments can be devised to measure selection intensities and selection responses accurately.

Selection Responses

Selection pressures on sensitive genotypes will vary with the kinds and amount of atmospheric pollution that occur. In cases of severe pollution, for example, at sites in close proximity to smelters, power stations, and refineries, high levels of mortality would result in extremely high selection intensities. These conditions are similar to those involving heavy metal contaminated mine waste, and, in common with these situations, we may anticipate rapid selection responses if appropriate genetic variation is present within populations (Roose et al. 1982). A more complex situation prevails, however, where low levels of air pollution occur on a regional level or where pollution is episodic in nature. Unfortunately, we know relatively little about the effects on fitness of these types of pollution stress, but unless reductions in growth or fecundity are large, selection responses are likely to be considerably slower, particularly in long-lived, outbreeding plants with seed banks. At present the most convincing evidence for the evolution of resistance to air pollution involves short-lived herbaceous species where populations have been exposed to relatively high levels of pollution stress. In the case of the evolution of SO_2 resistance in the annual *Geranium carolinianum*, this has apparently occurred in approximately 30 generations (Taylor and Murdy 1975; Taylor 1978).

The amount and rate of response to directional selection on a quantitative trait is affected by the number and average effect of genes controlling the trait (additive effects) and the occurrence of dominance, epistasis, and pleiotropy (nonadditive effects). Other factors that limit or constrain the response to selection include (1) negative genetic correlations among fitness components, (2) the occurrence of favorable alleles at certain loci in gametic disequilibrium with alleles at other loci with negative effects on fitness, (3) additive genetic variance exhausted by selection, and (4) finite population size effects or inbreeding leading to a loss of heritable variation. Although it is often difficult to isolate which of these factors are important, phenotypic responses following relaxation of selection can help distinguish between constraints that result from exhaustion of variability versus negative genetic correlations among fitness components. No phenotypic change would occur if quantitative genetic variation is limiting response, whereas a change would be expected if negative genetic correlations among fitness components are involved (Falconer 1981; Hedrick 1985).

These considerations may be important for the evolution of air pollution resistance for several reasons. First, mechanisms of resistance may involve traits that are of adaptive significance in unpolluted environments (e.g., growth rates, patterns of stomatal opening and closure, and cuticle thickness), and, as a result, we may expect fitness constraints to be associated with the evolution of resistance (Roose et al. 1982). Fitness costs could be manifested by negative genetic correlations between traits conferring greater resistance and features of plant growth and reproduction that contribute to fitness. Similarly, where plant populations are exposed to several atmospheric pollutants simultaneously, the resistance mechanisms required to effectively combat each one may involve different sets of genes. In some cases the genes may be negatively correlated with one another, so that the selection response would be constrained.

Unfortunately, little is known about the genetic architecture of resistance to air pollutants or whether cotolerances, of the type reported for heavy metals (e.g., Cox and Hutchinson 1979), are likely to occur for different air pollutants. (Preliminary evidence does suggest that cotolerances to air pollutants can evolve; see Taylor and Bell 1988). In addition, where genetic studies have been conducted they have usually involved plants of similar age and developmental status screened for a single pollutant under a small number of doses. This approach is chosen to maximize the differences in response among genotypes. Under field conditions, however, selection pressures are a good deal more complex because of heterogeneous age structures, selection intensities that vary in space and time, and exposure to several pollutants at one time. This complexity makes it extremely difficult to predict with any certainty the types of selection responses that are likely to occur, particularly for regional air pollution involving an array of low-level pollutants.

Allelic Variation and Pollution Stress

Abundant evidence exists for the occurrence of genetic variation in resistance to different air pollutants at the species, cultivar, population, and genotype level (reviewed in Roose et al. 1982; Karnosky et al. 1989). Far less is known, however, about how pollution stress influences patterns of genetic variation in natural populations and whether selective mortality causes significant reductions in the kinds and amounts of genetic variation. It seems reasonable to assume that in environments that are exposed to severe pollution, and in which mortality levels are very high, loss of genetic diversity will occur. This will be particularly likely for alleles that occur at low frequency. However, to what extent this loss of allelic diversity may handicap the evolutionary potential of resistant populations is by no means clear. Moreover, how low-level pollution on a regional scale may influence population genetic structure, through effects on viability and fertility, is complicated because of the diverse historical, ecological, and genetic factors that also regulate patterns of genetic diversity.

Recently a number of workers have investigated the relationships between variation in resistance to air pollution stress and patterns of genetic diversity by the use of isozyme techniques (Mejnartowicz 1983;

Scholz and Bergmann 1984; Bergmann and Scholz 1985; Müller-Starck 1985; Geburek et al. 1987). In these studies two basic approaches have been employed using European tree species (Scots pine, Norway spruce, and European beech). In the first (e.g., Scholz and Bergmann 1984), plants originating from different provenances and maternal families were subjected to controlled fumigations (SO_2), their responses were monitored, and individuals were genotyped at several isozyme loci. The second approach (e.g., Müller-Starck 1985) involved comparisons under field conditions of the patterns of isozyme variability in trees which displayed contrasting symptoms to air pollution damage (i.e., no apparent damage or irreversible injury). In both types of study, significant differences were detected between the two groupings in the number and frequency of alleles at polymorphic loci as well as in levels of heterozygosity (see below). In most comparisons, trees in the tolerant group exhibited higher levels of genetic diversity than the sensitive group. These findings led several investigators to suggest that variation at some isozyme loci—e.g., ACP, Mejnartowicz (1983); G6PDH, Bergmann and Scholz (1985); and GDH and AAT, Geburek et al. (1987)—may be of adaptive significance in contributing towards resistance to pollution-induced stresses (and see Bergman and Scholz, 1989) and that heterozygote superiority (overdominance) may account for the apparently higher levels of heterozygosity in resistant genotypes (Geburek et al. 1987; Karnosky et al. 1989).

Although isozymes provide the most convenient means of assaying the amounts of genetic variation within plant populations, they are of less value in determining the nature of selection pressures operating on the variation. Despite two decades of detailed experimental investigations of enzyme polymorphisms, there is still relatively little evidence that most allelic variation at isozyme loci is maintained by selection in natural populations (Lewontin 1974, 1985). Many studies have attempted to establish a causal link between a particular electromorph and a selective factor, but in the vast majority of cases it has not been possible to distinguish whether observed fitness differences are the result of the alleles being maintained or the genetic background of the individuals carrying them. Because of these difficulties, as well as a large body of theoretical work, most workers in evolutionary genetics have abandoned attempts to establish measurable fitness differences that accompany electrophoretic differences. Instead, it is usually assumed that most variation is either selectively neutral or under such weak selection that it is impractical to measure. As a final note of caution, it is important to recognize that although studies may elucidate the functional significance of a particular enzyme to pollution or other forms of stress, this does not necessarily indicate that fitness differences between allelic variants of the enzyme occur.

Several of the studies in which different classes of allelic variants at isozyme loci were thought to be causally related to pollution stress-involved samples of trees obtained from different populations. This type of sampling scheme confounds many sources of genetic variation making it difficult to ascribe the observed differences to a single factor, e.g., pollution stress. Samples of resistant and susceptible genotypes of Norway spruce (Bergmann and Scholz 1985) differed principally in the presence of a higher number of low frequency alleles in the resistant plants. In this case it seems more likely that these alleles simply mark unique genetic backgrounds that possess other characteristics that confer tolerance. Even where more controlled sampling has been undertaken within a local area, e.g., a five-hectare plot of *Fagus sylvatica* (Müller-Starck 1985), the observed differences in allelic diversity and heterozygosity may result from sampling effects. Sample sizes were quite small ($n = 44$ trees per tolerant and sensitive group), and no particular attempt appears to have been made to control for age, size, or the presence of population substructure. Other studies of *Fagus sylvatica* have demonstrated spatial and temporal genetic differentiation at isozyme loci on a local scale indicating considerable differentiation among subpopulations (Gregorius et al. 1986). The sampling problems associated with these pollution studies confound many factors that are likely to influence patterns of genetic variation in forest trees. As a result it may be premature to conclude that there is any biological significance to the observed differences in allelic variation at isozyme loci that have been reported between resistant and sensitive trees.

At present there is too little empirical data to assess the genetic consequences of various types of pollution-induced stresses. In theory, strong directional selection or the occurrence of an adaptive bottleneck associated with the evolution of resistance may lead to an erosion or rapid loss of genetic variation within populations. However, the critical issues depend on the number of individuals carrying resistance genes, the strength of selection against susceptible individuals, and the population recovery rate following the initial decline. Clearly, in cases where only a few variants capable of tolerating anthropogenic stresses are responsible for founding new resistant populations, bottlenecks in allelic variation may be anticipated (e.g., *Deschampsia cespitosa* at Sudbury; see Bush and Barrett 1989). This may occur in heavy-metal tolerant populations that invade mine wastes because of the low frequency of resistant individuals (e.g., Gartside and McNeilly 1974; Bradshaw 1984) that usually occur in unpolluted areas (although see Verkleij et al. (1989) for an example where no differences in genetic diversity were detected between populations on polluted mine sites and nonpolluted areas). However, this process seems less likely to prevail where low-level pollution effects are manifested over large areas and mortality levels are not sufficient to eliminate large numbers of individuals within local populations. Loss of some allelic variation is an inevitable consequence of pollution-induced stress. Whether such losses are biologically relevant to the evolutionary potential of natural populations is another matter.

Heterozygosity and Pollution Stress

The relationship between fitness and heterozygosity has been a major theme in evolutionary genetics since Lerner (1954) argued that heterozygosity gave organisms greater developmental and genetic homeostasis. These features are believed to enhance the ability to maintain high fitness over a broad range of environmental stresses. Although some evidence exists for a relationship between heterozygosity and fitness traits in some animal groups (e.g., Zourous et al. 1980; Leary et al. 1983), studies on plants have provided a more complex picture. For example, in forest trees heterozygosity and fitness components show a variety of relationships depending on the scales of measurement, genetic background, environment, and age (Mitton et al. 1981; Strauss 1987). Where positive relationships have been detected these have usually been in older forest stands and may reflect the greater homeostasis of heterozygotes, in the face of year to year variation, and/or the accentuation of differences by competition between heterozygotes and homozygotes as the canopy closes (Ledig et al. 1983).

The observation that heterozygosity at isozyme loci is higher in resistant individuals than sensitive ones in field trials where tree populations were exposed to multiple stress factors (several pollutants and natural stress conditions) has been interpreted as evidence in support of the classical hypothesis of heterozygote superiority (Karnosky et al. 1989). In addition, it has been claimed that high heterozygosity results in a "high adaptive potential for populations exposed to varying stress factors" (Geburek et al. 1987). There are several reasons why these conclusions may not be fully justified. Mitton and Pierce (1980) and Chakraborty (1981) have shown that heterozygosity measured at a small number of loci is a poor indicator of total genomic heterozygosity for an individual. Relatively small sample sizes for individuals and isozyme loci were employed for comparing heterozygosity in resistant and nonresistant trees (Mejnartowicz 1983; Müller-Starck 1985; Geburek 1987), and problems concerned with the confounding of genetic, environmental, and developmental variables already eluded to above are also evident in these analyses. However, even if concerns over sampling error are cast aside, their are still problems concerning the interpretation of the causes of the apparent heterozygote superiority that was observed. Many years of detailed biometrical studies of plants (e.g., see Jinks 1983) indicate that true overdominance either does not exist or is very rare. In addition, studies of isozyme variation in forest trees have usually failed to detect any heterosis associated with single isozyme loci (Ledig et al. 1983). Because of these results it seems more reasonable to interpret the apparent heterozygote superiority found in some studies as the result of inbreeding depression in homozygotes as a result of coancestry of descent (Ledig et al. 1983). Despite their outcrossed mating systems, populations of many tree species practice some inbreeding as a result of limited pollen and seed dispersal. With this mating pattern and high genetic loads, offspring carrying varying numbers of deleterious genes are produced at every generation giving rise to wide fitness variation (Ledig 1986). It seems more likely that it is this source of variation that accounts for the association between fitness components and heterozygosity rather than overdominance at isozyme loci or groups of linked genes that they mark.

Conclusions

In this chapter an attemp has been made to evaluate some of the features of plant life histories and reproductive systems that may facilitate or constrain the evolution of resistance to air pollutants in natural populations. Much of the discussion has been speculative since there is still insufficient empirical data to allow a comparative survey of a wide variety of plant species. Although there is a growing literature documenting effects of various atmospheric pollutants on plants, most studies have measured a small number of response variables under controlled conditions in plants of economic importance. This work is of importance in determining which aspects of plant growth and reproduction are sensitive to pollution stress, however, it is of less value in assessing how pollution in natural communities affects the life time fitness of genotypes within populations. Yet, a complete demonstration of natural selection will require this information in combination with studies of the heritability of traits conferring resistance (Endler 1986).

The best evidence for the evolution of resistance to air pollutants involves comparative data obtained from populations exposed to different levels of pollution (Taylor and Murdy 1975; Bell and Mudd 1976; Murdy 1979; Horsman et al. 1979; Ayazloo and Bell 1981; Ernst et al. 1985; Taylor and Bell 1988). In these studies genotypes from populations growing in highly polluted areas were found to be more tolerant to controlled exposures than genotypes obtained from unpolluted sites. In all cases the species involved in these studies are relatively short-lived herbs. The vast majority of examples of natural selection in wild populations of plants also involve herbaceous plants (see Table 5 in Endler 1986). This pattern highlights the difficulties that are associated with providing convincing experimental evidence of the direct effects of natural selection in long-lived organisms. Karnosky et al. (1989) recently stated that "air pollution has served and continues to serve as a strong natural selection factor in the evolution of forest ecosystems (Scholz 1981; Sinclair 1969)." A major future challenge will be to devise experiments to confirm this assertion. Although population biologists have recently devised techniques for the measurement of the direction and intensity of natural selection on phenotypic traits in natural populations (Arnold and Wade 1984a,b; Lande and Arnold 1983; Endler 1986), it remains to be seen whether these approaches can be usefully employed to study evolutionary processes in forest trees.

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