Effects of Hydrogen Fluoride on Growth, Common Blight Development, and the Accumulation of Fluoride in Field-Grown Red Kidney Beans

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ABSTRACT

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Field-grown California Light Red Kidney bean plants were spray-inoculated with a suspension of rifampin-resistant *Xanthomonas campestris* pv. *phaseoli* and exposed intermittently to hydrogen fluoride (HF) at 0, 2, or 4 μg F m^{-3} in open-top chambers during the summers of 1984 or 1985. Plants were exposed for 8 hr day^{-1}, 2 days each week for 9 wk in 1984 or for 8 hr day^{-1}, 4 days each week for 10 wk in 1985. Foliar accumulation of fluoride, disease severity, and epiphytic populations of the pathogen and other (unidentified) leaf surface microorganisms were determined weekly. The area under disease progress curve and final disease severity were not affected by exposure to HF, but the apparent infection rate increased with an increase in concentration of HF in 1985. There was no effect of exposure to HF on growth of epiphytic populations of the pathogen or on the populations of other epiphytic bacteria during either year. However, in both years the growth rate of fungal populations increased with an increase in concentration of HF. Yield was not affected by HF in 1984 but decreased with an increase in concentration of fluoride in foliar tissues in 1985.

Additional keywords: air pollution, epidemiology, pollutant-pathogen interactions.

Xanthomonas campestris pv. *phaseoli*, the bacterium responsible for common blight of beans (*Phaseolus vulgaris* L.), is an important pathogen of beans in almost all areas of the world where beans are grown. Like many other plant diseases, epidemics resulting in significant reductions in yield occur sporadically when environmental conditions are favorable for disease development. Air pollutants are components of the atmospheric environment and may also have an important impact on disease development.

Gaseous hydrogen fluoride (HF), the most phytotoxic of the major air pollutants, is emitted into the atmosphere as a by-product of many industrial processes. Fluoride (F) injury to vegetation generally results from uptake of HF through stomates and gradual accumulation of F in the foliar tissue over a period of time. The effects of atmospheric F on the development of disease in plants are not well understood at present. Exposure to F has been reported to cause changes in disease development, and several examples of F-induced modifications of disease have been summarized in a recent review (6).

Effects of F on development of diseases caused by bacterial pathogens have been reported by several investigators. Exposure to F had no effect on foliar symptoms of halo blight, caused by *Pseudomonas syringae* pv. *phaseolicola* (Burkh.) Dows., in snap beans subjected to the pollutant either before or after inoculation with the pathogen. However, a significant increase in the frequency of stem collapse occurred in plants exposed to the pollutant when compared with nonexposed controls (13). In contrast, low concentrations of HF (1 or 3 μg F m^{-3}) applied continuously for 5 days after inoculation of red kidney beans with *X. c. phaseoli* resulted in longer latent periods than those observed in inoculated plants not exposed to the pollutant. Decreases in both initial lesion size and growth of epiphytic populations of the pathogen were observed when compared with nonexposed controls (9). This evidence suggests that exposure to F may influence growth of bacterial pathogens and development of disease under controlled environmental conditions and continuous pollutant exposure regimes. However, there is no evidence that similar interactions occur in the field near sources of the pollutant. Moreover, it is difficult to generalize the responses of the plant-pathogen interaction with reference to atmospheric F primarily because so few examples have been documented.

An experiment was designed to assess the impact of intermittent exposures to low concentrations of HF on the development of common blight in red kidney beans under field conditions over an entire growing season. Specific objectives of the research were to determine the effects of HF exposure on: development of common blight epidemics; growth of epiphytic populations of the pathogen and of naturally occurring populations of leaf surface microorganisms that might influence pathogen populations; growth, partitioning of dry matter, and yield of red kidney beans; and the accumulation of F in plant tissue.

MATERIALS AND METHODS

Plant culture. Seeds of *Phaseolus vulgaris* 'California Light Red Kidney' were planted on 7 June 1984 and 6 June 1985 at the Boyce Thompson Institute field site located 1 km east of the Cornell University campus in Ithaca, NY. Seeds were planted with a commercial planter at a depth of 2.5–4.0 cm in 75-cm rows at an average density of 20 seeds per meter, according to recommendations for dry bean production in New York (14). Sixteen plots, each 3 m in diameter, were selected just before inoculation on the basis of uniformity of emergence and spacing of seedlings and apparent vigor of the plants. Due primarily to uneven pollutant distribution within chambers (see Results), plants located within 25 cm of the plenum were considered to be border plants and were not sampled during the experiment or included in yield measurements in either year.

Pathogen culture and inoculation. Four weeks after sowing and before the initiation of pollutant exposures, seedlings were inoculated with a strain of *X. c. phaseoli* resistant to 50 ppm of rifampin (17). On 2 July 1984, three plants in the center of each plot were spray-inoculated with a suspension of the bacterium in sterile water at a concentration of 5.4×10^8 colony-forming units (cfu) per milliliter. In 1985, all plants within each plot were sprayed to runoff with a suspension of the bacterium in sterile water (approximately 4.5×10^9 cfu per milliliter).

Pollutant exposures and air monitoring. Plants were exposed to HF in open-top chambers (3 m in diameter, 2.4 m high) constructed of a cylindrical aluminum frame covered with transparent plastic film panels (3). A blower attached at the base of each chamber provided a flow of ambient air of approximately

42.5 m^3 min^{-1}. Air distribution within the chamber was accomplished by means of a perforated plenum located at ground level around the inside perimeter of the chamber, with holes oriented vertically (11). Ambient plots (also 3 m in diameter but not enclosed by a chamber) were included as an additional control to estimate possible effects attributable specifically to the presence of the chamber itself.

Plants were exposed to HF at 0, 2, or 4 μg F m^{-3} for 8 hr during the day, twice each week for nine consecutive weeks in 1984. Each treatment was replicated four times in a randomized complete block design. The experiment was repeated in 1985 using the same atmospheric concentrations of HF, but the frequency of exposure was increased from 2 to 4 days each week for 10 consecutive weeks. The length of time between the end of one exposure period and the beginning of the next ranged from 14 to 134 hr in 1984 and 14 to 86 hr in 1985. The pollutant was generated by volatilizing aqueous solutions of hydrofluoric acid in a hot air stream, which was then delivered into the blower unit supplying air to each chamber (12). Because incoming air was not filtered, plants were exposed not only to HF but also to ambient oxidants. The average daily concentration of ozone was approximately 0.04 ppm (0900–1600 EST) during the experimental period each year.

Air within each chamber was sampled continuously during each 8-hr exposure period by drawing known volumes of chamber air through 47-mm-diameter NaOH-impregnated filter paper disks (Whatman No. 41), supported in Millipore Swinnex polypropylene holders (Cat. SX00 04700, Millipore Corp., Bedford, MA)(14). Volumetric flow of air through the filters was regulated by a limiting orifice and recorded both before and after sampling. At the end of each exposure, the filters were collected and eluted in TISAB (TISAB II, Orion Research, Inc., Cambridge, MA). The concentration of F was determined using an ion-specific electrode (Model 94-09, Orion Research, Inc.). Two sampling devices were mounted on a vertical steel rod located at the center of each chamber. The height of the devices was periodically adjusted over the course of the season so that air was sampled just above the level of the plant canopy. Air in ambient plots was not sampled.

The distribution of F in two of the chambers (one supplied with 2 μg F m^{-3} and the other with 4 μg F m^{-3}) was determined using static samplers several times during the course of the 1985 experiment (13). Pieces of NaOH-impregnated filter paper (3.75 × 4.50 cm) were placed at regular locations within the chamber just above canopy height (approximately 0.5 m above the soil surface). After 24 or 48 hr, the concentration of F was determined as before. These samplers provided an estimate of pollutant flux, or the total quantity of F collected on a surface in a given period of time, expressed in μg F per static sampler (16.88 cm^2) per 24- or 48-hr period.

Disease assessment. Disease progress was monitored weekly during the 1984 experiment by recording the total number of lesions present on foliage within each plot, excluding those on the three inoculated plants. Lesion area was not measured in 1984 because it was too small to measure accurately by visual inspection. In 1985, disease progress was monitored weekly by recording both incidence and severity of common blight within each plot. Disease incidence was determined by counting the number of individual plants with visible common blight lesions on the foliage and was expressed as the proportion of diseased plants in the total population of plants within each plot. An estimation of disease severity was based on a random sample of 13 trifoliolate bean leaves, not removed from the plants, within each plot each week. The percentage of necrotic leaf area due specifically to *X. c. phaseoli* (based on symptoms) was estimated visually for each leaflet of each randomly selected leaf using Key No. 3.3.1 (common bacterial blight of beans, leaf symptoms) in the Manual of Disease Assessment Keys (4).

Epiphytic populations. In 1984, two samples (two leaflets per sample) were collected weekly from the central row within 25 cm of the inoculated plants in each plot to determine leaf-surface populations of *X. c. phaseoli* as well as other naturally occurring fungal and bacterial epiphytes. Two additional samples were collected each week from the same row to determine the leaf-surface populations of naturally occurring fungi and bacteria only. In 1985, two randomly selected leaflets were removed from each of two rows within each plot at weekly intervals. Samples were taken from different plants each week and an effort was made to avoid very old, nearly senescent leaflets or very young, rapidly expanding ones.

After collection, areas of the leaflets were measured, and the leaflets were washed for 1 hr in 100 ml of sterile deionized water containing 0.025% (v/v) Tween 80. The washings were serially diluted, plated on rifampin agar medium (18) as well as on nutrient and potato-dextrose agar media, and then incubated at 27 C. After 3 days of incubation, the colonies of *X. c. phaseoli*, other bacteria, and fungi were counted to obtain estimates of leaf surface populations of microorganisms.

All rifampin-resistant bacterial strains recovered from washed leaflets were presumed to be *X. c. phaseoli* but were tested for pathogenicity on bean. Colonies were transferred to fresh plates of rifampin agar medium and incubated for 48 hr at 27 C. Leaves of greenhouse-grown California Light Red Kidney bean plants were injured with a sterile pincushion, and swabbed with the test strain using a cotton-tipped applicator. Symptoms of common blight were generally observed within 2–3 wk after inoculation, and served as a positive confirmation of the identity of the organism. Additional plants were wounded each week in the same manner and then swabbed with sterile water to serve as checks. Identification of epiphytic bacteria and fungi (other than *X. c. phaseoli*) was beyond the scope of the experiment and was not attempted.

Plant growth and yield measurements. In 1984, an additional sample of leaf tissue for F analysis was collected weekly from each plot (12 leaflets per plot) during the last 4 wk of exposures. Fluoride content was measured on a dry weight basis (μg F g^{-1}) by the semiautomated method (1,2).

In 1985, one row in each plot was designated for sampling to determine partitioning of dry matter by the plant over the course of the experiment. Beginning at one end of the row, the aboveground portions of two consecutive plants were removed each week for 10 wk. Each plant was separated into stems, leaves, and pods, placed in a forced-air drying oven (80 C) for 24–48 hr, and then weighed. Leaf tissue was analyzed for F content (1,2).

At the end of each season, beans were harvested from each plot. Mature pods were stripped from the stems and placed in a drying oven for 24 hr before seeds were removed and weighed.

Statistical analysis. Because of differences in the method of inoculation and pollutant exposure regime in 1984 and 1985, the data from each year were analyzed separately.

The mean percent disease severity for each plot and week was transformed using the logit transformation, and the regression of logit on time was calculated. The slope of this regression line, which is an estimate of the apparent infection rate (r value), was calculated for each plot (15). The area under disease progress curve (AUDPC) was calculated for each plot as an additional measurement of the severity of the epidemic (5,21). Disease progress in 1984 was determined by recording the number of lesions in each plot at weekly intervals after inoculation. Natural logarithms of lesion counts were regressed on time after inoculation for each plot. The resulting estimate of the slope for each regression indicates the rate of common blight development in each plot. The maximum intensity of common blight was expressed as the maximum number of lesions observed on foliage in each plot during the 9-wk exposure period. Analyses of variance were performed on the rate of progress (increase in lesion count over time) and the maximum intensity of common blight using block and treatment as main factors.

Effects of treatment on the development of common blight in 1985 were analyzed as follows. Analyses of variance were performed on the maximum disease severity (week 8), the apparent infection rate, and the AUDPC, using block and treatment as main factors. Single-degree-of-freedom contrasts were used to identify significant differences among the treatment means. When significant effects of exposure to HF were observed, an additional analysis was performed by regressing the measurement of disease

development on concentration of F in air (ambient plots not included).

Linear regressions of $\log_{10}$-transformed populations of epiphytic fungi on time were performed for data from each plot in 1984 and 1985. The growth rates of these populations were estimated by the slopes for each regression.

A linear regression model was used to describe the increase in concentration of F with time in leaves of plants exposed to 2 or 4 μg F m^{-3} in 1985. Linear regression of foliar F content on pollutant dose (the product of the mean concentration and the duration of exposure) was used to model the accumulation of F in leaf tissue after 9 or 10 wk of intermittent exposure to HF in 1984 or 1985. In each case, higher-order terms were added and statistically compared with the simple linear model. In addition, lack-of-fit tests were conducted and residual plots were examined to determine adequacy of the linear model.

Linear regression of dry mass of stems, leaves, and pods on time was used to model plant growth. The proportions (dry mass) of stems, leaves, and pods of the total aboveground mass of each plant were calculated. Linear regression was also used to model the change in the proportion of each component over time. In each case, higher-order terms were added as necessary and the resulting models were compared statistically with the simple linear model. Lack-of-fit tests were conducted and residual plots were examined to determine adequacy of the linear model. An analysis of variance was performed on the yield, in terms of dry mass of seeds per plant, using block and treatment as main factors. Single-degree-of-freedom contrasts among the treatment means were used to identify specific treatment effects. Regression analysis was also used to determine the nature of the relationship between atmospheric concentration of HF and yield and between foliar F concentration and yield.

RESULTS

Accumulation of F in leaf tissue. Although the concentration of HF within open-top chambers has been shown to be fairly uniform across the chamber (13), pollutant flux as measured by static samplers was not. In general, the distribution of F across the chamber was uneven in the region where plants were growing. Relative flux was lowest in the central portion of the chamber where the air sampling device was located and increased sharply near the wall of the chamber. The highest relative flux was measured just inside the plenum (approximately 33% higher than flux in center of chamber). The pattern of distribution of F within the chambers was similar, regardless of the atmospheric HF concentration.

In both years, F content of leaf tissue increased significantly with exposure to HF (Table 1). Linear regression of the concentration of F in foliage after 9 or 10 wk of intermittent exposure to HF on total pollutant dose provided a reasonable prediction of F accumulation (Fig. 1). The addition of higher-order terms did not improve the fit of the model and the lack-of-fit of the linear model was not significant for 1984 or 1985 data. Pollutant dose alone accounted for 93 and 85% of the variability observed in foliar F concentrations at the end of the 1984 and 1985 exposure periods, respectively. Plants exposed only 2 days each week accumulated nearly twice as much F per unit dose as plants exposed more frequently. Fluoride concentrations in both ambient and control plots remained relatively constant over the exposure period, but during both years, foliar concentrations of F were consistently higher in leaves from control chambers (no HF added) than those from ambient plots. This was most likely a result of greater air flow through the chamber than through ambient plots. Concentrations of F in the foliage of plants exposed twice each week to 2 or 4 μg F m^{-3} remained relatively constant over the last 4 wk of exposures, with the exception of a slight decrease during the seventh week. During the second field experiment when the frequency of exposure was increased from 2 to 4 days each week, the concentrations of F within HF-exposed leaves increased linearly (data not shown) over the 10-wk period and were considerably higher than those of the previous season (Table 1). Very high foliar concentrations of 844.3 ppm F and 529.8 ppm F were found in plants from one chamber receiving 4 μg F m^{-3}. These values were considerably higher (3 or 4 ×) than the other replications of this treatment and, therefore, were declared outliers and omitted from subsequent analyses. Fluoride-induced injury to foliage was not observed during either year despite the high concentrations present in some plants.

Disease development. Disease progress during each year is summarized in Table 2. Disease progress (the increase in number of

TABLE 1. Accumulation of fluoride in leaves of red kidney bean plants exposed intermittently to hydrogen fluoride (HF)

Atmospheric HF concentration (μg F m^{-3})	Foliar F concentration (ppmF)[a]	
	1984[b]	1985[c]
0 (ambient)	12.3 (2.2)	32.8 (7.1)
0	14.7 (1.2)	20.5 (4.0)
2	87.8 (14.6)	131.7 (7.4)
4	175.5 (8.9)	212.4 (35.5)[d]

[a] Values are means of four replications with standard errors in parentheses.
[b] Plants exposed for nine consecutive weeks.
[c] Plants exposed for ten consecutive weeks.
[d] Based on three replications.

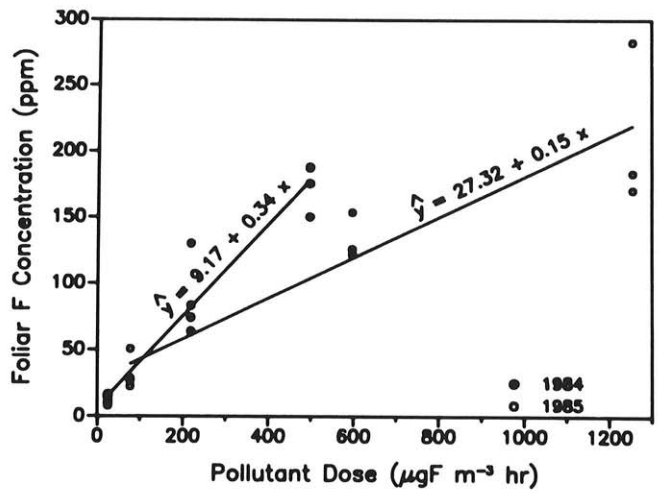

Fig. 1. Regression of concentration of fluoride (F) in leaves of field-grown red kidney bean plants on total pollutant dose (μg F m^{-3} hr). Plants were exposed intermittently to hydrogen fluoride for 9 wk in 1984 or for 10 wk in 1985.

TABLE 2. Treatment means and standard errors of parameter estimates of disease progress in field-grown red kidney bean plants exposed intermittently to hydrogen fluoride (HF) for 9 wk in 1984 or 10 wk in 1985

Atmospheric HF concentration (μg F m^{-3})	1984		1985		
	Rate of disease progress[a]	Maximum number of lesions	AUDPC[b]	Apparent infection rate (r)[c]	Maximum severity[d]
0 (ambient)	0.599	13.0	0.130	0.759	7.57
	(0.243)	(5.7)	(0.031)	(0.047)	(2.29)
0	0.969	7.8	0.151	0.392	6.38
	(0.292)	(2.5)	(0.065)	(0.125)	(3.26)
2	1.249	24.3	0.144	0.560	5.23
	(0.365)	(7.2)	(0.040)	(0.077)	(2.33)
4	1.257	8.0	0.144	0.725	5.62
	(0.550)	(3.0)	(0.019)	(0.088)	(1.24)

[a] Estimate of slope from regression of $\log_e$-transformed lesion counts on time after inoculation.
[b] Area under disease progress curve.
[c] Estimate of the slope from the regression of logit-transformed disease severity on time. Excluding ambient plots, the regression of r on HF concentration was significant at $P = 0.03$ (equation: $y = 0.392 + 0.083x$).
[d] Leaf area affected (%) 12 wk after planting and 8 wk after inoculation.

lesions over time) was not significantly affected by the atmospheric concentration of HF or by the concentration of F within foliar tissue when plants were exposed to HF 2 days each week.

In 1985, exposure to HF had no effect on the maximum disease severity or the AUDPC. However, the apparent infection rate increased linearly with increasing concentration of F in air. Although the appearance of diseased individuals was more rapid among plants in control chambers than in ambient plots during the first several weeks following inoculation, there was no effect of HF exposure on the incidence of disease during the remainder of the exposure period. Disease incidence increased with time, and although severity remained low, nearly all plants within each plot had a least one common blight lesion by the end of the season.

Epiphytic populations. Because of mortality in storage, only 50 out of 167 rifampin-resistant bacterial isolates recovered from field samples in 1984 were tested for pathogenicity on bean. All 50 isolates were found to be pathogenic. In 1985, a total of 281 isolates were recovered, and 272 of these were found to be pathogenic. The pathogenicity of the remaining nine isolates could not be ascertained because of leaf abscission before symptom development.

Populations of leaf surface microorganisms fluctuated considerably with time over the course of each experiment in response to a continuously changing environment. There was no difference in the size of pathogen populations among the four F treatments at any of the weekly sampling intervals. During the first several weeks of exposures in 1984, leaf-surface populations of the pathogen increased but then decreased during the remainder of the experiment (Fig. 2A). In 1985, epiphytic populations of the pathogen increased slightly during the first week after inoculation but then decreased during the remainder of the experiment (Figure 2B), despite the increase in both incidence and severity of the disease. Also in 1985, leaf-surface populations of the pathogen were considerably larger than the previous year, due to the fact that all plants within each plot were inoculated in 1985, as opposed to only three plants within each plot inoculated in 1984. In 1984, establishment of epiphytic populations depended on natural mechanisms (i.e., wind-driven rain) to move bacteria from inoculated to adjacent, uninoculated plants.

Epiphytic populations of other bacteria and fungi increased with time during both years. Some differences in populations of naturally occurring epiphytic bacteria were observed among treatments during the fourth and seventh weeks in 1984 and the fourth and fifth weeks in 1985. The increase in $\log_{10}$-transformed populations of fungal epiphytes was linear with time and estimates of the rates of increase in 1984 and 1985 are shown in Table 3. In both years, the rate of increase in fungal populations was greater in plants exposed to 2 or 4 μg F m^{-3} than in controls.

Plant growth and yield. The yield of beans (dry mass of seeds per plant) was not affected by exposure to atmospheric HF in 1984. Although there was no effect of HF-level in 1985, there was a significant linear relationship between yield (seed dry mass per plant) and the concentration of F in leaf tissue after 10 wk of intermittent exposures (Fig. 3). An increase in the foliar concentration of F was associated with a decrease in the yield of dry beans per plant.

Dry mass of stems and leaves increased rapidly during the first 3–4 wk of the exposure period (Fig. 4A and B). Flowering, which began to occur during the third week, was followed by a decrease in the rate of growth of stem tissue and, in many plots, a substantial decrease in the dry mass of leaves. Pod mass increased with time after flowering (Fig. 4C). Variability among treatments increased substantially after the fifth week of exposures, suggesting that exposure to F was beginning to have some impact on partitioning of dry matter by the plant.

Linear regression of plant dry mass on time was used to model growth of stems, pods, and the total aboveground portions of individual plants within each plot. Addition of a second-order term did not significantly improve the fit of model, and the lack-of-fit of the linear model was not significant in the case of mass of stems or pods. However, dry mass of leaves was not adequately described by a simple linear model. Analyses of variance were performed on the growth rates (slope estimates) of stems and pods using block and treatment as main factors. No significant differences were observed among growth rates due to exposure treatment.

Mean proportions of stem, leaf, and pod dry mass per plant for each treatment after 10 wk of intermittent exposure to HF are shown in Table 4. An analysis of variance was performed on the mean proportion of dry matter in stems, leaves, and pods for each plant at the end of the 10th week, using block and treatment as

TABLE 3. Apparent linear rates of increase in $\log_{10}$-transformed populations of epiphytic fungi on the leaf surface of red kidney bean plants exposed intermittently to hydrogen fluoride (HF)

Atmospheric HF concentration (μg F m^{-3})	Apparent rate of population increase ($\log_{10}$ cfu per week)[a]	
	1984[b]	1985[c]
0 (ambient)	0.175 (0.069)	0.141 (0.012)
0	0.152 (0.035)	0.119 (0.005)
2	0.251 (0.019)	0.155 (0.011)
4	0.287 (0.023)	0.154 (0.009)

[a] Values are means of four replications, with standard errors in parentheses.
[b] Plants exposed for nine consecutive weeks.
[c] Plants exposed for ten consecutive weeks.

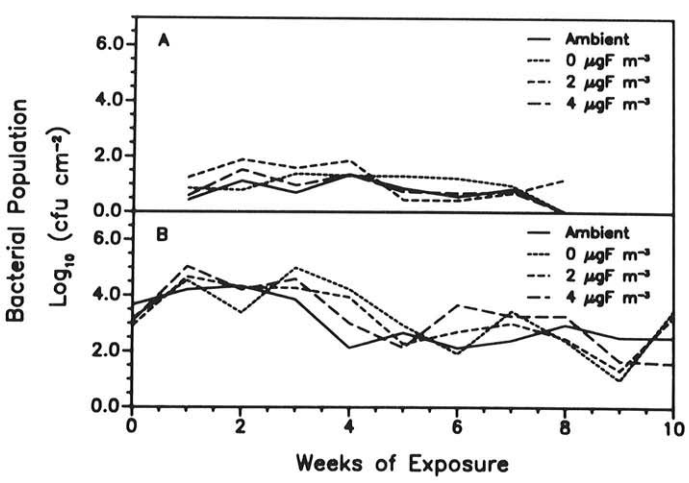

Fig. 2. Growth of epiphytic populations of *Xanthomonas campestris* pv. *phaseoli* on the leaf surface of field-grown red kidney bean plants exposed intermittently to hydrogen fluoride, **A,** for nine consecutive weeks in 1984, or **B,** for 10 consecutive weeks in 1985.

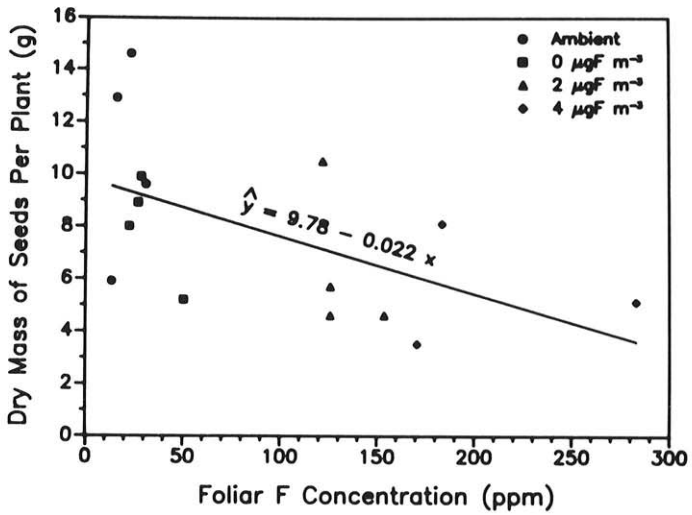

Fig. 3. Decrease in dry mass of seeds per plant with increasing concentration of fluoride (F) in foliar tissue after 10 wk of intermittent exposure to hydrogen fluoride.

main factors. There were no significant differences among treatments in terms of the final proportion of stem, leaf, or pod dry mass.

Partitioning of dry matter was determined by expressing the dry mass of stem, leaf, and pod components as a proportion of the total aboveground mass of each individual plant. In terms of proportion of aboveground mass, stem mass remained nearly constant over the 10-wk exposure period, while dry mass of leaves decreased with time, and dry mass of pods increased with time after flowering, as leaf mass decreased.

Linear regression was used to model the change in proportions of leaf and pod mass per plant with time after the initiation of F exposures. Analyses of variance were performed on the linear rates of change (slope estimates) using block and treatment as main factors. Although the overall treatment effect was not significant, single-degree-of-freedom contrasts among the treatment means indicated that there was no difference between the rate of decrease in proportion of leaf mass per plant in control chambers compared with ambient plots, but that the proportion of leaf mass per plant decreased significantly faster in plants from ambient plots and control chambers than in plants exposed to 2 or 4 μg F m^{-3}. Significant differences in the rate of increase in proportion of pod mass were observed among treatments. However, the observed differences were primarily due to a significant chamber effect. The rate of increase in pod mass proportion was significantly greater in plants from ambient plots than in those from the control chambers and was most likely in response to a higher air flow through the chamber relative to ambient plots.

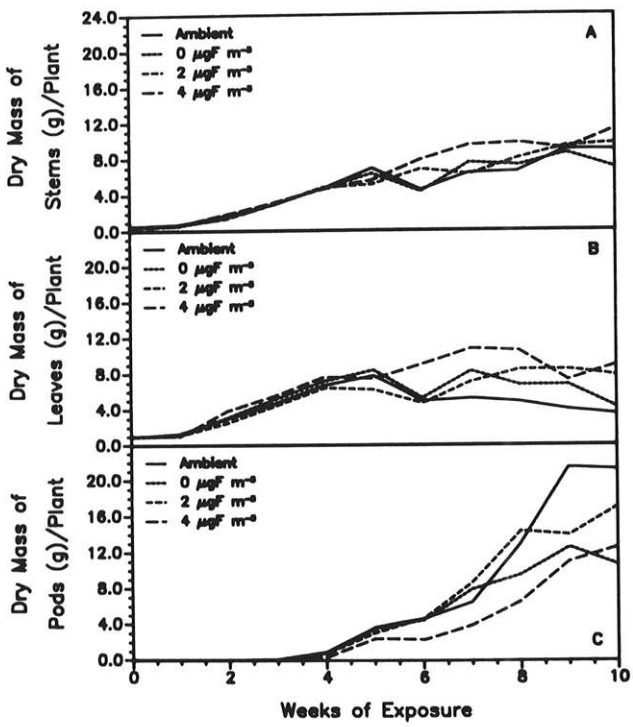

Fig. 4. Dry mass of **A,** stems, **B,** leaves, and **C,** pods of field-grown red kidney bean plants exposed intermittently to HF for 10 wk in 1985.

TABLE 4. Treatment means and standard errors (in parentheses) of aboveground proportion of stem, leaf, and pod mass per plant after 10 wk of intermittent exposure to hydrogen fluoride (HF)

Atmospheric HF concentration (μg F m^{-3})	Proportion of total aboveground mass		
	Stem mass	Leaf mass	Pod mass
0 (ambient)	0.267 (0.017)	0.100 (0.026)	0.632 (0.042)
0	0.328 (0.011)	0.213 (0.041)	0.458 (0.049)
2	0.275 (0.027)	0.207 (0.048)	0.517 (0.060)
4	0.346 (0.052)	0.269 (0.040)	0.386 (0.092)

DISCUSSION

Results of these experiments suggest that the primary effect of atmospheric HF on development of common blight is not a direct effect of gaseous HF on the pathogen but an indirect effect of accumulated F on the host. There is no evidence, by any measure of disease, that the atmospheric concentration of HF is associated with adverse effects on the pathogen. Indeed, the apparent infection rate increased with level of HF, while epiphytic populations of the pathogen remained unaffected by exposure to the pollutant. The plant host, however, was adversely affected by exposure to HF. Exposure to the pollutant resulted in significant reductions in the rate of decrease in proportion of leaf dry mass, suggesting a delay in maturation, due perhaps to a suppression of carbon transport from leaves to pods and/or roots. Although the effect on pod mass was not statistically significant and the change in dry mass of roots was not measured, this trend was reflected in the yield of beans at the end of the season (Fig. 4C). While not affected directly by F in air, significant reductions in yield were associated with increasing concentration of F in leaves. Reduced yield (seed mass) in 1985 and delayed development (proportion of biomass) suggested that physiological processes were indeed affected, but exposure to HF had very little impact on development of common blight. Consequently, the ability of the host to accumulate F determines the likelihood of an effect on disease progress.

The observed effect on yield was not closely associated with atmospheric concentration of HF but with ppm F in foliage. This suggests that the concentration of F in air, by itself, is not necessarily a good predictor of F-induced effects in general. The value of atmospheric concentration, compared with foliar concentration of F as a predictor of effects (i.e., reduced yield) was probably limited not only by inherent differences among individual plants with respect to the ability to accumulate F from air, but also by the observed variability in pollutant flux within the chamber. Even though the linear dose model provided a good prediction of F uptake, the variability in atmospheric concentrations and pollutant flux and lack of short-term air monitoring equipment make it difficult to predict the accumulation of F in plants in the field.

The lack of response of disease development to exposure to HF observed during 1984 and limited response in 1985 may have been due to slow development and low severity of disease or to less accumulated F (in 1984), or both. Whether disease development under more conducive conditions, resulting in more severe epidemics of common blight, would have been affected by the same exposure regimes remains unknown. If the apparent infection rate is increased by elevated levels of F, as suggested by the results of the 1985 experiment, then disease would progress more rapidly, perhaps resulting in greater reductions in yield and greater economic loss. However, most of the evidence from controlled environment studies suggests that development of individual common blight lesions is suppressed by exposure to atmospheric pollutants, including HF (7–10). If expansion of individual lesions is inhibited by exposure to HF, then the rate of epidemic development would likewise be reduced in the presence of the pollutant.

On the other hand, disease development may have been unaffected by exposure to F because the doses were too low to induce a response. Beans are relatively tolerant to F in terms of foliar symptoms (16). Although the mechanism of tolerance is not known, presumably the capacity of the plant to withstand some level of F without any apparent effect indicates that normal physiological processes are not appreciably disrupted. It is likely that indirect effects of atmospheric F on development of the disease are a consequence of modification of host physiology in response to accumulated F in plant tissue. Hence, effects on disease development in response to a pollutant burden would not be expected to occur unless physiological processes have been affected.

It is apparent from these experiments that indirect effects of F exposure on bean production through modification of common

blight development are not likely to be important. At low levels of common blight severity, there was little effect of F on development of the disease, although there was some evidence that the rate of infection may be greater (relative to controls) when exposed intermittently to 2 or 4 μg F m^{-3} over a 10-wk period. Significant reductions in yield that were measured were attributed to F accumulated in leaves, but this effect was found only in 1985 when the mean concentration of F in foliar tissue reached a maximum of approximately 212 ppm. No effects on yield were observed in 1984 when the mean concentration of F in foliar tissue at the end of the season was approximately 176 ppm. Atmospheric F might be expected to have a greater effect on disease development in plants more sensitive to F or in F-tolerant species growing in areas where atmospheric concentrations are unusually high.

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