Development of Common Blight and Accumulation of Fluoride in Red Kidney Bean Plants Exposed Continuously or Intermittently to Hydrogen Fluoride

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ABSTRACT

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Four-week-old bean plants (cultivar California Light Red Kidney) were spray inoculated with rifampin-resistant *Xanthomonas campestris* pv. *phaseoli* to establish a leaf-surface population on one leaf and a lesion on another leaf of each plant. In one experiment, plants were exposed to 0 or 1 μg F m^{-3} (as hydrogen fluoride) continuously or 3 or 5 μg F m^{-3} intermittently for 15 days. In the other experiment, plants were exposed continuously to 0 or 1 μg F m^{-3} for 15 days or 3 μg F m^{-3} for 5 days or 5 μg F m^{-3} for 3 days after inoculation. Fluoride treatments in both experiments resulted in a total pollutant dose of 15 μg F m^{-3} days. Diameters of lesions were measured and leaves were sampled periodically to determine fluoride accumulation. Intermittent exposure treatments had no effect on final lesion growth. However, lesion size and expansion increased linearly with increasing fluoride in foliage. Intermittent fluoride exposure had no effect on growth of epiphytic populations of the bacterium. The development of lesions and leaf-surface populations of the pathogen exposed continuously were not affected by the exposure regime or the concentration of fluoride in air or foliage.

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

Plants are rarely, if ever, exposed continuously to hydrogen fluoride (HF) in the field. The majority of exposures are intermittent, with the length, frequency of exposure, and concentration controlled by environmental conditions and proximity to a source. However, because of difficulties in monitoring HF and the lack of short-term air-monitoring data from the field, long-term, low-level exposures often have been used to simulate the occurrence of fluoride near a source. Many previous experiments have used continuous exposures to assess effects of pollutants on the development of disease (2–7). Inhibition of the development of common blight by continuous exposure to HF has been reported (5,7).

The interaction between the pathogen and its host may be affected not only by concentration of HF and the duration of exposure, but also by the frequency and sequence of exposures. The amount of fluoride absorbed by plants exposed to equal doses of HF may differ depending on whether the pollutant is supplied continuously or intermittently (8). Differences in the accumulation of fluoride, in turn, might affect populations of foliar pathogens both on the surface of the leaf and within the leaf. Alterations in physiological processes in response to accumulated fluoride may lead to reduced growth of the pathogen and ultimately inhibit development of the disease. Laurence and Reynolds (5) observed slower growth of populations of *Xanthomonas campestris* pv. *phaseoli* on the leaf surface of plants exposed continuously to HF before inoculation with the pathogen when compared with controls, suggesting that the effect was due to accumulation of fluoride in leaf tissue or fluoride-induced changes in the physiology of the host. On the other hand, no measurable differences in lesion size or latent period were found in response to preinoculation exposure with HF. Exposure to HF at increasing concentrations up to 3 μg F m^{-3} for 5 days immediately after inoculation resulted in smaller lesions initially and longer latent periods but had no effect on the final lesion size. Apparently, once the pathogen became established, development was no longer affected by the presence of HF in the air or fluoride in the leaves. Thus, a difference in the accumulation of fluoride among plants receiving the same total dose of pollutant, but exposed either continuously or intermittently to HF, may result in differences in initial growth of the pathogen and subsequent development of the disease (lesions) within foliar tissue.

Two experiments were conducted under controlled environmental conditions to determine the effects of continuous or intermittent exposure to HF on epiphytic growth of *X. c. phaseoli* on the leaf surface, as well as growth within the leaf during the pathogenic phase. The first experiment (designated "intermittent") was designed to evaluate the response of the pathogen to intermittent exposure to low concentrations of HF. Equal total pollutant doses were supplied, but with different exposure frequency, over a period of 15 days. The second experiment (designated "continuous") was designed to test whether continuous exposures that provide the same concentrations of HF and total pollutant dose as intermittent exposures affect pathogen and disease development.

MATERIALS AND METHODS

Plant culture and inoculation. Seeds of *Phaseolus vulgaris* L. 'California Light Red Kidney' were sown in 10-cm pots in a pasteurized potting mix of peat, sandy loam, and sand (1:1:1, v/v). After emergence, seedlings were thinned to one plant per pot. Plants were grown in a greenhouse supplied with filtered air and maintained at 25/20 C (day/night) with a 16-hr photoperiod provided by multivapor high-intensity discharge lamps. Fertilizer (20:20:20 NPK, diluted 1:16) was supplied in solution daily to maintain adequate plant nutrition. A rifampin-resistant strain of *X. c. phaseoli* was selected from yeast extract-calcium carbonate agar amended with 50 ppm rifampin (13). Four weeks after sowing, plants were inoculated with suspensions of the bacterium in sterile water containing between 10^9 and 10^{10} colony-forming units ml^{-1} and 0.025% Tween 80 (by volume). A leaf-surface population was established on the first trifoliolate leaf of each plant using low-pressure spray, and a lesion was initiated on a second trifoliolate leaflet by leaf infiltration using a high-pressure spray. Plants then were transferred to controlled-environment chambers maintained at 25/20 C (day/night) and 60/70% relative humidity (day/night) with a 14-hr photoperiod and light intensity (in the photosynthetically active range) of 600–700/0 μmol s^{-1} m^{-2} (day/night). Air supplied to the greenhouse and growth chambers was passed through both activated charcoal and Purafil II (Purafil, Inc., Atlanta, GA) to remove ambient pollutants.

Pollutant generation and air sampling. Concentrations of HF gas were generated by volatilizing aqueous solutions of HF in a heated air stream (11). The pollutant then was introduced into the filtered air supplied to each chamber. Air within each chamber was sampled continuously for 24-hr periods by drawing known volumes of the chamber atmosphere through NaOH-impregnated filter paper disks (11). Air flow through each of two sampling devices per chamber was measured at the beginning and end of each 24-hr sampling period with a Gilmont Calibrated and Correlated Flowmeter (No. F-1360, Gilmont Instruments, Inc., Great Neck, NY). Filters were removed, the fluoride was eluted in total ionic strength adjustment buffer (TISAB II, Orion Research, Inc., Cambridge, MA), and the concentration of fluoride was determined with a fluoride ion-specific electrode (Model 94-09, Orion Research, Inc.) and a Fisher Accumet Selective Ion Analyzer (Model 750, Fisher Scientific Co., Pittsburgh, PA).

Intermittent exposures. Plants in the chambers were exposed to 1 μg F m^{-3} continuously, or 3 or 5 μg F m^{-3} intermittently for 15 days, or were not exposed to the pollutant after inoculation with the pathogen. In intermittent exposures, the total dose of pollutant in each case was equal to the dose resulting from continuous exposure to 1 μg F m^{-3}. The concentration of HF and sequence of exposures for each of the four treatments are shown in Table 1.

Continuous exposures. Plants were exposed to 1 μg F m^{-3} in filtered air continuously for 15 consecutive days, or 3 μg F

TABLE 1. Intermittent hydrogen fluoride (HF) exposure regimes[a]

Concentration of HF in air (μg F m^{-3})	Days after inoculation															Pollutant dose (μg F m^{-3} days)
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
0	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	0
1	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	15
3			X			X			X			X			X	15
5					X					X					X	15

[a]Days on which exposures occurred are indicated with an "X."

TABLE 2. Continuous hydrogen fluoride (HF) exposure regimes[a]

Concentration of HF in air (μg F m^{-3})	Days after inoculation															Pollutant dose (μg F m^{-3} days)
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
0	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	0
1	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	15
3	X	X	X	X	X											15
5	X	X	X													15

[a]Days on which exposures occurred are indicated with an "X."

m^{-3} for 5 consecutive days, or 5 μg F m^{-3} for 3 consecutive days, or were not exposed to the pollutant immediately after inoculation with the pathogen. The exposures were conducted such that the total dose of pollutant in each case was equal to the dose resulting from continuous exposure to 1 μg F m^{-3}. The concentration of HF and the sequence of exposures for each of the four treatments are shown in Table 2.

In each experiment, over the entire 15-day exposure period, all three HF exposure treatments resulted in the same total dose of 15 μg F m^{-3} days. On days when plants were not exposed to HF, they were placed in a growth chamber supplied only with filtered air.

Measurement of lesions and epiphytic populations. Diameters of lesions were measured when they first became visible and again at the end of the experiment (15 days after inoculation). Lesion expansion was defined as the difference between the initial and final lesion diameter. The rate of lesion expansion was calculated by dividing lesion expansion by the number of days between the first appearance of the lesion and the last day of the experiment.

In each experiment, four first trifoliolate leaves per treatment were collected at intervals after inoculation for estimation of leaf surface populations of the pathogen. Leaves were collected 0, 5, 10, and 15 days after inoculation during intermittent exposures, and 0, 3, 5, and 15 days after inoculation during continuous exposures. The areas of the leaflets were measured using a LI-COR portable area meter (model LI-3000, LI-COR, Inc., Lincoln, NE), and each leaf was washed for 1 hr in 100 ml of sterile deionized water containing 0.025% Tween 80, by volume. The washings were serially diluted, plated on rifampin agar medium (13), and incubated at 27 C for 3 days. Colonies were counted to obtain estimates of leaf-surface populations of the bacterium.

Accumulation of fluoride in leaf tissue. After the final lesion size was recorded, four samples (of four second trifoliolate leaves each) were harvested, dried at 70 C for at least 24 hr, and analyzed for fluoride. Concentrations of fluoride in plant tissue were determined using the semiautomated method (1). Additional uninoculated plants were included in the third replication to provide information about how fluoride is accumulated over time. The first and second trifoliolate leaves from two plants per treatment were collected at 5-day intervals during the experiment, dried, and analyzed for fluoride.

In all three replications of the second experiment (continuous exposures), leaves from exposed but uninoculated plants were sampled to provide information about accumulation of fluoride in each of the different continuous exposure regimes. The first and second trifoliolate leaves from these plants were collected and dried, and the fluoride contents were determined as described above.

Experimental design and statistical analysis. Each of the experiments consisted of four treatments: a control (no HF) and three HF exposure regimes, each resulting in a total pollutant dose of 15 μg F m^{-3} days. In each experiment, the treatments were replicated three times (in time), and treatments were randomly reassigned to the four growth chambers for each replication. Within each replication were 16 plants per treatment with lesions and four plants per treatment per sampling day with epiphytic populations.

Data from the intermittent and continuous exposure experiments were analyzed separately. Analyses of variance were performed on the data for final lesion size, lesion expansion, and the rate of lesion expansion using replication and treatment as main factors. Regression analysis was used to evaluate development of the disease in response to either the concentration of HF in chamber air or the foliar concentration of fluoride. Each of the three lesion variables was regressed on the atmospheric concentration of HF during exposures. Each lesion measurement then was regressed on the concentration of fluoride in foliage at the end of the experiment. Only data from the first two replications of the intermittent experiment were used in this regression analysis because leaf samples for fluoride analysis were collected differently in the third replication.

The rate of growth of the pathogen on the leaf surface was determined for each combination of treatment and replication. Logarithms (base 10) of bacterial populations per unit area of leaf surface were regressed on time after inoculation. A common intercept (population size on day 0) and four separate slopes (population growth rates for each treatment) were estimated for each replication. Appropriateness of a common intercept for all treatments within a replication was based on the assumption that inoculations with the pathogen were uniform and therefore initial population densities were similar within each replication. Analyses of variance then were performed on the estimated growth rates using replication and treatment as main factors. Because a linear regression model did not provide an adequate description of growth of leaf-surface populations exposed continuously to HF,

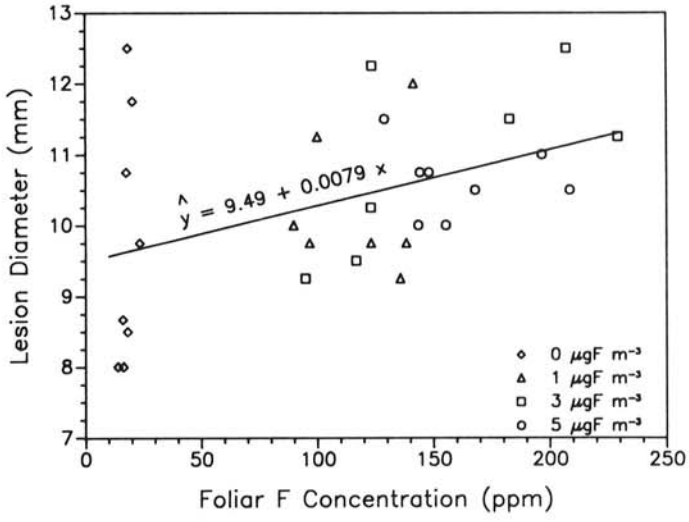

Fig. 1. Regression of diameter of common blight lesions on foliar fluoride content of leaves of red kidney bean plants exposed intermittently to hydrogen fluoride for 15 days.

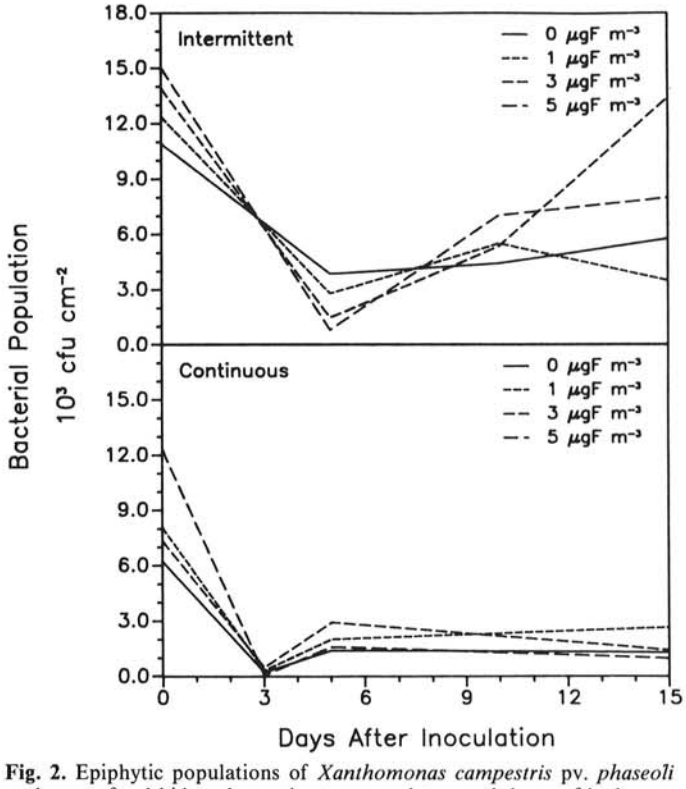

Fig. 2. Epiphytic populations of *Xanthomonas campestris* pv. *phaseoli* on leaves of red kidney bean plants exposed to equal doses of hydrogen fluoride supplied intermittently for 15 days (top), or continuously for 3, 5, or 15 days (bottom).

analyses of variance were performed on the population means on each sampling day, using replication and treatment as main factors.

Linear regression of concentration of fluoride in foliage on time was used to determine the rate of accumulation in each combination of treatment and replication for both intermittent and continuous exposures. The regression model included a common intercept (fluoride content on day 0) and separate slopes (fluoride accumulation rates) for each treatment and was used to fit data from each replication separately. For intermittent exposures, the rate of accumulation was calculated over the entire 15-day period, but for continuous exposure, the rates were calculated over the time interval that plants were exposed to the pollutant, that is, 3, 5, or 15 days. An analysis of variance was performed on the rate of accumulation to determine the effect of concentration of HF in chamber air on the rate of fluoride acumulation in foliage in each experiment. Analyses of variance also were performed on the concentration of fluoride in leaves at the end of each 15-day experiment to detect significant differences between HF exposure treatments (controls were not included in these analyses).

Two models were employed to describe and compare the accumulation of fluoride in leaves of plants exposed intermittently or continuously to HF over a 15-day period. The following linear regression of foliar fluoride content on pollutant dose was used to model the accumulation of fluoride during each experiment:

$$Y = \beta_0 + \beta_1 CT + \epsilon \qquad (1)$$

in which Y is the concentration of fluoride in leaf tissue (ppm), C is the mean concentration of HF in the chamber air (μg F m^{-3}), T is the length of the exposure period in days, ϵ is the error term, and β_0 and β_1 are parameters.

McCune and Hitchcock (12) suggested that fluoride concentrations within leaf tissue decrease in an exponential fashion when exposure to the pollutant is discontinued. They proposed a modified version of the linear dose model that incorporates this exponential decay function. The linearized form of this model,

$$\ln(Y) = \beta_0 + \beta_1 \ln(C) + \beta_2 \ln(T) + \beta_3 D + \epsilon \qquad (2)$$

in which Y, C, and T are the same as in the previous model, and D is the total number of days that the plants were not exposed to the pollutant before sampling, includes separate coefficients for concentration and duration terms (β_1 and β_2) as well as an additional term ($\beta_3 D$) to account for the loss or dilution of fluoride during periods when the plants were not exposed to the pollutant.

RESULTS

Lesion development. Intermittent HF exposure treatments, consisting of a control (no HF) and three HF exposure regimes resulting in equivalent doses, had no effect on final lesion size, lesion expansion, or the rate of lesion expansion. When these lesion variables were regressed on foliar fluoride content, there were significant linear increases in the size (Fig. 1) and expansion of lesions with increasing foliar fluoride content. Although statistically significant, the regressions accounted for only 16–18% of the total variability in the data.

Continuous HF exposure treatments, consisting of a control (no HF) and three HF exposure regimes resulting in equal doses, again had no effect on expansion or the final size of lesions. There was no effect of the concentration of HF in chamber air on the size or expansion of lesions, regardless of the exposure regime.

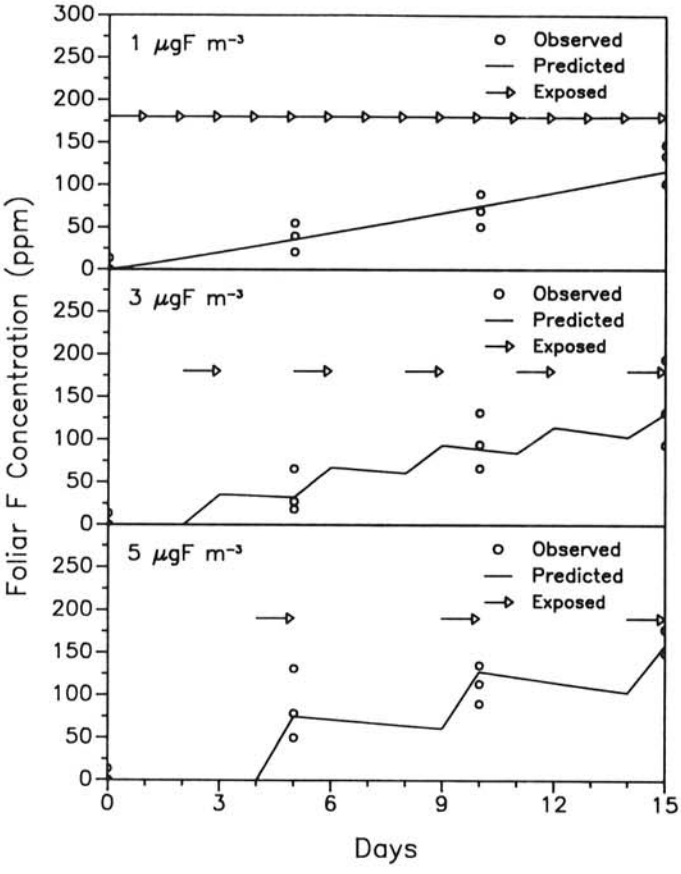

Fig. 3. Observed and predicted values for the concentration of fluoride in leaves of red kidney bean plants exposed intermittently to hydrogen fluoride at 1 μg F m^{-3}, 3 μg F m^{-3}, or 5 μg F m^{-3}. Accumulation of fluoride in leaves was modeled using equation 2.

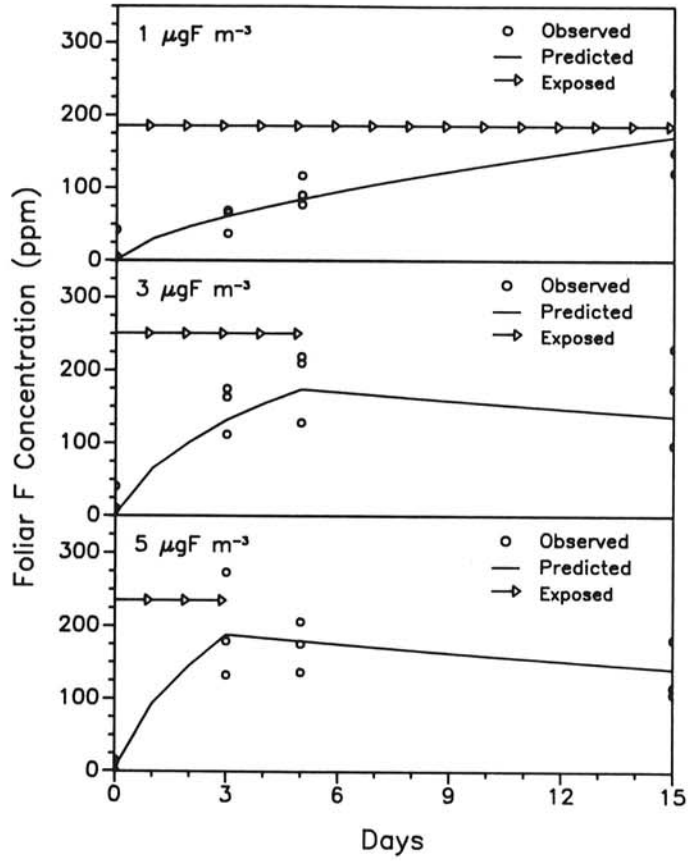

Fig. 4. Observed and predicted values for the concentration of fluoride in leaves of red kidney bean plants exposed continuously to hydrogen fluoride at 1 μg F m^{-3}, 3 μg F m^{-3}, or 5 μg F m^{-3}. Accumulation of fluoride in leaves was modeled using equation 2.

Epiphytic populations. Growth of populations of the pathogen on leaf surfaces was not altered directly by exposure to HF in the air or indirectly by the concentration of fluoride in the leaves, regardless of whether the exposures were continuous or intermittent (Fig. 2). There was no significant effect of concentration of HF in air on the rate of growth of leaf surface populations of the pathogen when exposed intermittently to HF. However, the variability among replications may have masked effects of concentration of HF in air on growth of epiphytic populations. The relationship between the rate of growth of leaf surface populations of the pathogen and the concentration of HF in chamber air may have been more obvious if the experiment had been repeated several more times or if variability had been reduced.

Accumulation of fluoride in leaf tissue. The concentration of fluoride within leaves before exposure to the pollutant ranged from 1.5 to 24.8 ppm fluoride with one particularly high sample containing 41.8 ppm fluoride. This background level of fluoride remained relatively constant in plants that were not exposed to fluoride over the 15-day period. Accumulation of fluoride in leaves subjected to intermittent exposures is shown in Figure 3. Uptake of fluoride in plants exposed to 5 μg F m^{-3} was greater and more rapid than that at the other concentrations during the first exposure but then declined to a rate of accumulation over the last 10 days similar to that in plants exposed to the two lower concentrations. This rapid initial uptake of HF during the first exposure followed by a decrease in the rate of fluoride accumulation during subsequent exposures has been noted in previous experiments (5). Due to the amount of HF taken up by leaves during the first exposure to 5 μg F m^{-3}, the fluoride content was higher after 15 days than in those plants exposed to lower concentrations, despite the similarity in accumulation rates among the three treatments after 5 days.

Accumulation of fluoride in leaves of plants exposed continuously to HF is shown in Figure 4. The amount of HF taken up by leaves during the exposures was significantly affected by the concentration of HF in air. The rate of uptake increased linearly with the concentration of HF (Fig. 5). However, at the end of the 15-day experiment, there were no significant differences among fluoride concentrations in leaves exposed to the three exposure regimes. Unfortunately, because of the way treatments were designed, the concentrations of HF in air and the durations of exposure were completely confounded. Although it was not possible to discern statistically whether the significant effect on the rate of accumulation of fluoride in leaves was in response to the concentration of HF in air or the duration of exposure, it is reasonable to assume that the observed increase in the rate of accumulation of fluoride in leaves was due to an increase in the concentration of HF in air and not a decrease in the duration of exposure.

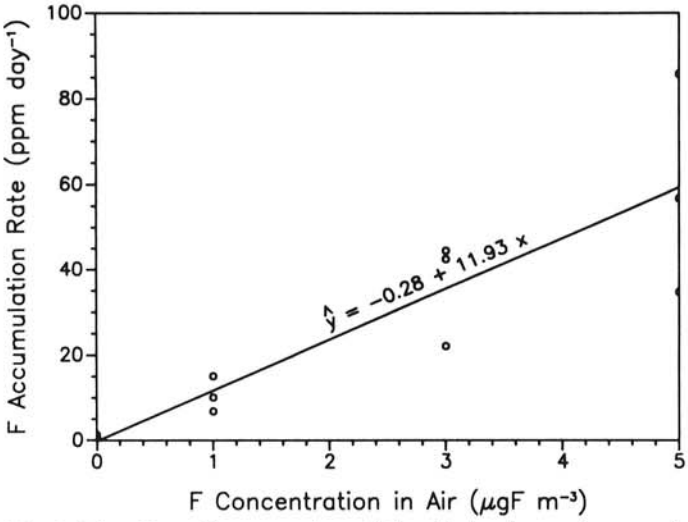

Fig. 5. The effect of concentration of fluoride in air on the rate of accumulation of fluoride in leaves of red kidney bean plants during continuous exposure to hydrogen fluoride.

The linear dose model (equation 1) was used to describe the accumulation of fluoride in leaves of red kidney bean plants exposed either intermittently or continuously to HF. The regressions were highly significant for both intermittent and continuous experiments. However, only 58 and 44% of the total variability in the observations was explained by dose alone. Application of the modified model (equation 2) to the observed data improved the overall fit, accounting for 72 and 65% of the total variability in observed concentrations of fluoride in foliage exposed either intermittently or continuously to HF, respectively. The observed and predicted values for each intermittent exposure treatment are shown in Figure 3, and those for each continuous exposure treatment are shown in Figure 4. The modified model (equation 2) is undefined at an exposure duration of 0 days or at an exposure concentration of 0 μg F m^{-3}. Therefore, data from control plants were not included in fitting either equation 1 or 2 so that the models would be comparable.

DISCUSSION

Failure of epiphytic populations of the pathogen to respond to intermittent exposures to low concentrations of atmospheric HF is consistent with results of previous experiments in which inoculated plants were exposed intermittently to HF over a 4-day period (5). However, in that study, continuous exposure to 3 μg F m^{-3} for 5 days after inoculation with the pathogen resulted in significantly smaller lesion size and leaf-surface populations when compared with the controls (5). The response to continuous exposure suggests the possibility of recovery during the periods between intermittent exposures. Results of the present study suggest that recovery during intervals between intermittent exposures probably is not responsible for the apparent insensitivity of epiphytic populations of the pathogen to intermittent HF exposure because continuous exposure also had no effect on growth of the pathogen on the leaf surface.

When inoculated plants were exposed to HF intermittently, neither the exposure regime nor the concentration of HF in air had any effect on common blight development in terms of size or expansion of lesions. However, disease development did increase with fluoride accumulation in the leaf. This result suggests that effects on disease development are due primarily to fluoride-induced changes in the host plant and emphasizes the importance of fluoride uptake by plants in determining the indirect effects of the pollutant on disease development. Thus, it may not be possible to predict indirect effects of atmospheric HF pollution based on the concentration, frequency, or total dose of pollutant unless the relationships among these exposure characteristics and accumulation of fluoride by the plant can be accurately quantified.

The disparity between continuous and intermittent exposures resulting in comparable HF doses with respect to fluoride accumulation by plants is illustrated by these as well as earlier results. In the present study, exposure to intermittent high levels of HF resulted in greater accumulation of fluoride in the foliage than continuous low-level exposure, even though plants received the same total dose of HF over the 15-day exposure period. MacLean et al (10) observed a similar phenomenon in forage consisting of a mixed planting of timothy and red clover exposed to HF. Accumulation of fluoride in forage after 14 days of continuous exposure to 1.6 μg F m^{-3} was significantly less than in plants given the same pollutant dose using intermittent 48-hr exposures to 3.4 μg F m^{-3} over the same 14-day period. The converse, however, also has been observed. MacLean and Schneider (9) found that, when comparable doses were achieved using the same concentration of HF (either 2.3 or 5.0 μg F m^{-3}) in continuous exposures for 10 days and in intermittent exposures over a period of 18 to 22 days, accumulation of fluoride in forage was greater in plants exposed continuously. These observations suggest that the frequency or sequence of exposure may be as important as concentration and duration in determining the effects

of HF.

Results obtained from efforts to model the accumulation of fluoride under a variety of exposure regimes could be useful in evaluating the potential for damage to crops in the vicinity of a source. Most effects of atmospheric HF on plant productivity are a consequence of the accumulation and concentration of fluoride in plant tissues. The use of air-monitoring data to assess the potential impact of HF pollution is contingent upon knowing the relationship between accumulation of fluoride by the plant and the various characteristics of exposure, including composition and concentration of the pollutant, as well as the duration and sequence of exposures. Because continuous, constant concentration exposures to HF seldom occur in the field, pollutant dose may not provide an accurate prediction of fluoride uptake by the plant. Peak concentrations and the duration of a pollutant episode should perhaps be considered separately rather than collapsed into a single dose term.

There are many other factors in addition to concentration of pollutant and duration of exposure that may influence the accumulation of fluoride in plants exposed to atmospheric HF under field conditions (8). The most important of these include the chemical and physical form of the pollutant or pollutants, plant characteristics such as stage of development during exposure, and environmental factors, particularly rainfall. Prediction of fluoride accumulation based on models that consider only concentration and duration of exposure was not accurate even under controlled conditions and is likely to be completely useless under highly variable conditions as occur in field situations. However, inclusion of other variables known to influence the uptake of HF may improve the model sufficiently to enable more accurate prediction of fluoride accumulation by plants and ultimately the effects on plant productivity in a wide variety of agricultural situations.

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