Pathogen-Induced Changes in Host Water Relations

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A portion of the literature in plant pathology is devoted to the water relations aspects of disease physiology (11, 12, 14, 16, 38). However, our present knowledge of pathogen-induced changes in host water relations is limited in terms of the number of host-pathogen combinations and water relations parameters which have been examined. In this presentation, that portion of the literature which describes the physical aspects of water relations in diseased plants will be reviewed, and mechanisms by which various pathogens alter host cell turgor and water status will be discussed. As in previous and more inclusive reviews on the topic (11, 12, 38, 43), there will be some emphasis on vascular wilt diseases.

Pathogen-induced changes in any one or combination of several parameters will affect the water status and turgor of the host. The water status of plant tissues can be described in terms of potential energy by the equation:

$$\Psi = \Psi_s + \Psi_p + \Psi_m$$

where Ψ is total water potential and Ψ_s, Ψ_p, and Ψ_m are solute, pressure, and matric potentials, respectively (35). Matric potentials in leaves are usually considered to be negligible (1, 35). In cells, pressure potential is identified with turgor pressure, and solute potential depends on the concentration of osmotically active solute retained within membranes. The water potential of various plant parts is a function of resistance to water movement and rates of water movement in the plant-soil continuum and of the availability of water in the soil (7, 35). New data on Verticillium wilt of cotton and the hypersensitive reaction of tobacco to incompatible plant-pathogenic bacteria, as well as published data, will be used to illustrate the role of several water relations parameters in pathogen-induced changes in host cell turgor and water potential.

MATERIALS AND METHODS.—*Biological materials.*—Cotton plants (*Gossypium hirsutum* L. 'Acala SJ-1') were grown in a greenhouse at 24 ± 3 C and 50 ± 10% relative humidity. Stems were inoculated with nondefoliating strain SS4 of *Verticillium dahliae* Kleb. when the first lobed leaves were almost fully expanded. Approximately 0.05 ml of inoculum containing 10^6 conidia/ml were introduced into a stem wound made with a dissecting

needle just below the cotyledons. Stems of control plants were treated with sterile water. Tobacco plants (*Nicotiana tabacum* L. 'Havana') were grown in the same greenhouse and used for experimentation when they had five to six fully expanded leaves. Interveinal portions of young leaves were injected with *Pseudomonas syringae* van Hall, *P. fluorescens* Migula, or sterile water. Bacterial cells were washed by centrifugation, and water suspensions of 10^9 cells/ml were injected with a syringe (21). All three treatments were applied to different interveinal portions of the same leaf, and at least five tobacco plants were used in each experiment.

Observations and measurements were made under greenhouse conditions, unless stated otherwise. Portions of leaves on inoculated cotton plants wilted 8 to 16 days after inoculation, and wilting commonly preceded chlorosis. Variation in turgor and leaf water content was due to disease development in inoculated plants and due to withholding water from the soil of healthy plants. Water injected into tobacco leaves disappeared within 1 hr, and neither the water nor the *P. fluorescens* treatment had a visible effect on turgor. In contrast, portions of leaves injected with *P. syringae* were collapsed 5 to 6 hr after treatment and turned necrotic within 24 hr. Variation in the water content of tobacco leaves which had previously been injected with water was obtained by allowing excised leaves to transpire to a predetermined weight.

Relative water content.—Disks 1.4 cm in diam were cut from cotton leaves at various times after inoculation and from injected portions of tobacco leaves 8 hr after treatment. The disks were immediately weighed, and floated on water at 25 C in a saturated atmosphere and 100 ft-c of light. Disks were periodically blotted dry, weighed, and refloated. I judged turgidity by pressing opposite disk margins between two fingers. When leaf disks regained turgidity their relative water content (θ) was calculated by the equation:

$$\theta = [(w_f - w_d) / (w_t - w_d)] \times 100$$

where w_f is the fresh weight before floating, w_t is the turgid weight after 12 hr of floating, and w_d is the oven dry weight (1). Error due to edge injection in cotton was minimized by floating leaf disks adaxial surface down.

Leaf solute potential.—Disks from healthy and Verticillium-infected cotton leaves with similar water contents were floated for 12 hr, washed, blotted dry, and frozen in small plastic bags. Disks were cut from portions of tobacco leaves that had been injected with water or bacteria 8 hr previously or from frozen tobacco leaves and were floated and subsequently frozen by the methods described for cotton. Sap was expressed from thawed leaf disks and placed in a vapor pressure osmometer to determine solute potential (14).

Diffusive resistance.—A porometer (14) was used to measure the diffusive resistance of leaves to water vapor loss in a controlled-environment chamber (80 w m^{-2} of light in the 300 to 700 nm waveband at 24 ± 1 C and 68 ± 2% relative humidity). The diffusive resistance of tobacco leaves was measured as a function of time after treatment, and the diffusive resistance of cotton leaves was measured as a function of the relative water content of the leaf area exposed to the porometer cup.

RESULTS AND DISCUSSION.—*Components of total water potential and cell turgor.*—It is necessary to distinguish experimentally the relative roles of water and solute loss from within cells and the resulting changes in water, solute, and pressure potentials in pathogen-induced turgor losses. The importance of this distinction is increased by the frequent hypothesis that pathogen-synthesized toxins markedly affect host membrane permeability and therefore the retention of solute within cells (44). A loss of cell solute can, of course, result in the loss of some water, but water losses from cells and resulting low water potentials usually occur with little or no change in solute potential. This second type of water loss is reversible, as long as the loss is not too extreme, and the wilting induced by several vascular pathogens (14, 25, 40), a virus (20), rust (16), and root rot (9, 31) has been demonstrated to be reversible. Data in Fig. 1 show that wilting induced by *V. dahliae* in cotton is also reversible. Disks from healthy and diseased cotton leaves which were wilted regained turgor and had equivalent fresh weights per unit area when floated on water. (Final disk area and therefore fresh weight depended on the degree of leaf wilting at which disks were cut). In contrast, Fig. 1 shows that turgor loss induced in tobacco by *P. syringae* is irreversible and disks of this treatment increased only slightly in fresh weight when floated on water. Control disks at the same initial fresh weight regained turgor. Whereas the hypersensitive reaction of tobacco to *P. syringae* is generally thought to be an irreversible process (21), the irreversible nature of the turgor loss has not been demonstrated previously.

Where water loss and wilting are completely reversible, as demonstrated for cotton (Fig. 1), the most useful measure of tissue water content is relative to the water content observed at full turgor (1). In a given type of leaf undergoing dehydration, relative water content has a characteristic relationship to leaf water potential (1, 35). The data in Fig. 2 demonstrate that diseased cotton leaves first wilted at slightly lower relative water contents than did healthy leaves. Observations similar to those presented here for cotton showed that the relationship between leaf wilting and water potential is nearly identical for healthy and Fusarium-infected tomato plants (14). Harrison (25) worked with potato plants infected with *V. albo-atrum* and found the relative water contents associated with leaf wilting to be similar for healthy and infected plants. These correlations between initial wilting during dehydration and some measure of leaf water status indicate that infection did not change the solute and pressure components of leaf water potential significantly.

Aside from the vascular wilts, there are few published measurements of water status in diseased

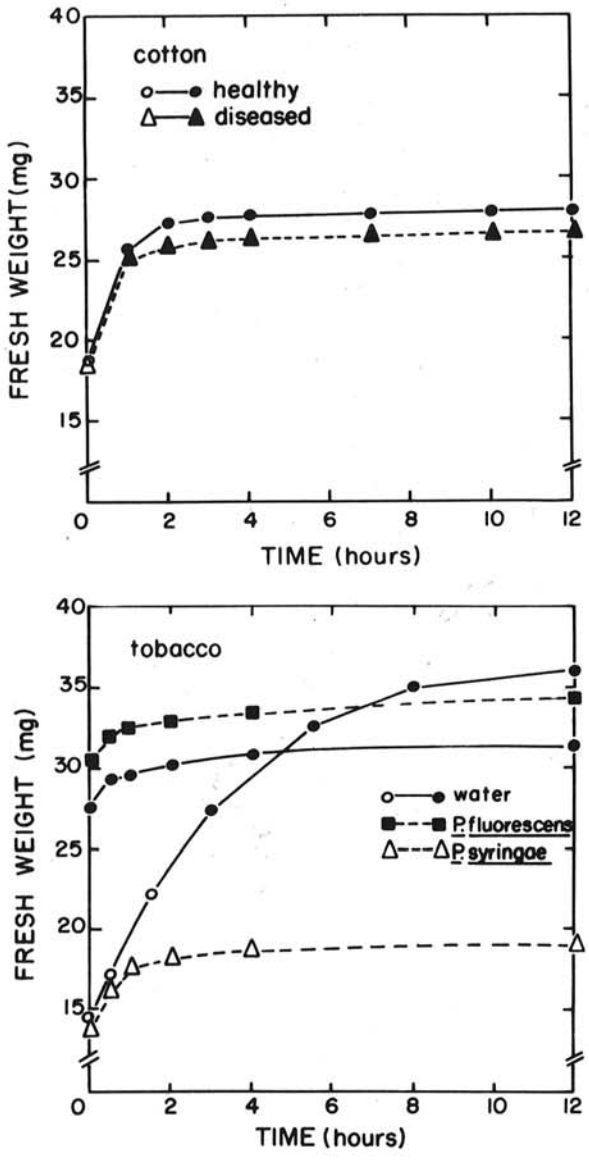


Fig. 1. Fresh weights of leaf disks measured as functions of time after floating on water. Treatments are indicated in the figure, and open and closed symbols represent wilted and turgid disks, respectively. Each point is the mean of five or more disks and SE $\leq$ 1.2 mg for all points.

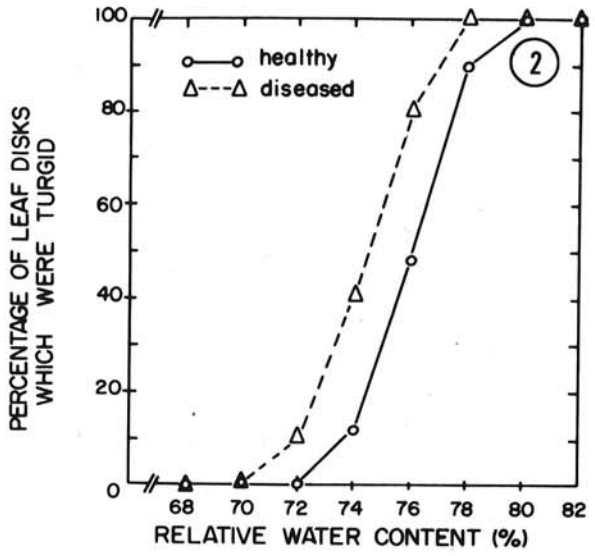
plants. Plant water potentials have been shown to decrease with time after root infection by *Verticicladiella wagenerii* in pine (27). An unidentified root pathogen suspected to be *Phytophthora cinnamomi* caused wilting and abnormally low water potentials in rhododendron (9). Large numbers of sporulating rust infections have been demonstrated to cause abnormally low water potentials and wilting to occur in bean plants (16).

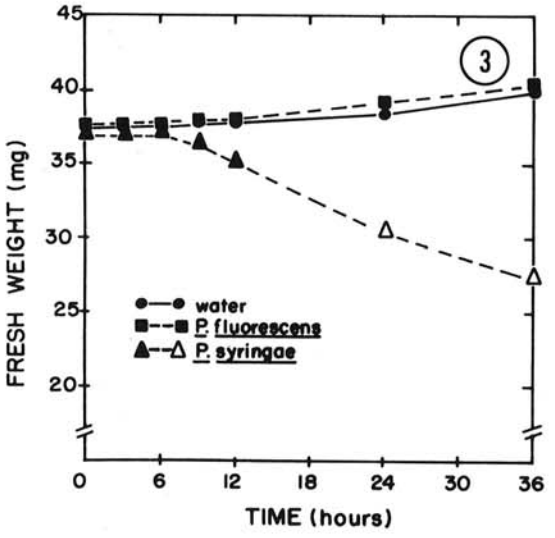
The solute potential of leaf cells can be estimated directly. Since solute potential varies with cell volume (1, 35), estimates of solute potential in healthy and diseased tissues should be measured at or corrected to the same cell volume. Lack of data on cell volume complicates interpretation of existing plasmolytic estimates of cell solute potential in diseased plants (39). Changes in the amount of solute retained within cells will, of course, affect pressure potential; i.e., the relationship between water and solute potential. Thermocouple psychrometry has been used to show that rust infection has no effect on the relationship between water and solute potential in bean leaves (16), and the pressure chamber technique is potentially useful for examining this relationship in diseased leaves (42).

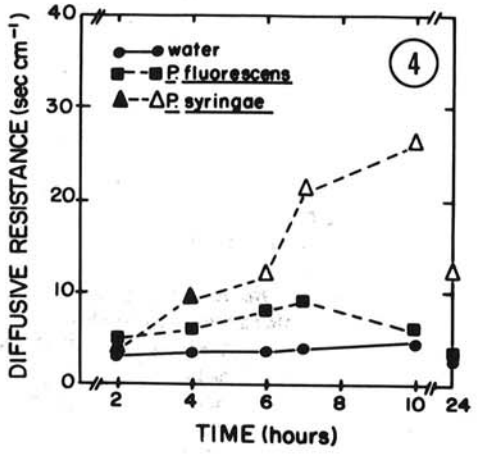
Solute potentials in healthy and diseased tissue can be meaningfully compared at full turgor where cell volume is maximal and water potential is nearly zero (35). Table 1 shows that healthy and Verticillium-infected cotton leaves had nearly the same solute potential at full turgor, and similar results have been reported for Fusarium wilt in tomato (14) and bean rust (16). The solute potential of tobacco leaves undergoing a hypersensitive reaction to *P. syringae* was investigated by the same methods. Data in Table 2 demonstrate that injection with *P. fluorescens* did not affect solute potential significantly, whereas injection with *P. syringae* or freezing caused marked and similar increases in the solute potential of floated leaf disks. Evidently the irreversible turgor loss caused by *P. syringae* results from a marked increase in the permeability of cell membranes to solute and, therefore, contrasts with the other examples of turgor losses in infected plants which are discussed here.

The permeability of host cell membranes to solute has frequently been examined by measurement of electrolyte loss with a conductivity meter. For example, tobacco leaves undergoing a hypersensitive reaction to bacteria were found to have a greater electrolyte loss than control leaves (21), as were leaves affected by Fusarium wilt in tomato (6) and Verticillium wilt in chrysanthemum (24). Whereas high rates of electrolyte loss from diseased tissues may reflect an increase in membrane permeability, the exact role of the observed electrolyte losses in cell water relations is not clear. For one thing, osmotically induced water deficits and subsequent recovery increase electrolyte loss from plant cells (10), and it is necessary in some instances, such as Fusarium wilt in tomato (6, 14), to judge whether abnormal electrolyte loss from diseased leaves results from or contributes to the observed turgor loss.

Wilting due primarily to solute loss from within cell membranes, unlike wilting due only to water loss, is expected to occur even when water potential of the surrounding medium and transpiration are zero. The hypersensitive reaction of tobacco to *P. syringae* demonstrates this point. Disks were cut from leaves immediately following injection and were floated on water. Figure 3 shows that *P. syringae* caused significant decreases in the fresh weight and turgor of floating disks in a saturated atmosphere. The wilting induced by *V. dahliae* in cotton does not occur when transpiration is zero. Infected cotton plants did not







wilt when kept in a mist chamber between 8 and 18 days after inoculation, but did wilt without recovery when removed from the mist chamber or when maintained without mist for 16 days after inoculation. Elimination of transpiration by the use of mist also prevents wilt symptoms of the black shank disease in tobacco (31).

Transpiration rate.—The transpiration rate of leaves depends on the vapor concentration gradient and resistances to water vapor diffusion between interior leaf surfaces and the ambient air (7, 35). Resistances to gas diffusion between leaf cells and the bulk air also govern the rate of CO_2 uptake, and recent studies on the photosynthesis of diseased plants have analyzed these resistances (17, 23). The equation for transpiration can be written in the following form:

$$E = (c_w - c_a) / (r_a + r_l)$$

where E (g cm^{-2} sec^{-1}) is the transpiration rate per unit leaf area, c_w and c_a (g cm^{-3}) are, respectively, the water vapor concentrations at the surfaces of the mesophyll cell walls and in the bulk air, r_a (sec cm^{-1}) is the diffusive resistance to water vapor across the boundary layer which sheathes the leaf, and r_l is the diffusive resistance in the leaf. The diffusive resistance in the leaf includes stomatal and cuticular resistances, which are parallel to each other and are the leaf parameters in transpiration most likely to be affected by disease. Resistance to water vapor diffusion within mesophyll air spaces is relatively small, and the assumption that the concentration of water vapor at evaporating surfaces within leaves is the saturation vapor concentration at leaf temperature is valid under any but the most extreme conditions (28).

The above analysis indicates that changes in cell permeability are not likely to increase transpiration by affecting the availability of water to evaporating surfaces in leaves, as has been hypothesized to occur in diseased plants (e.g. 26, 39, 45). Permeability changes are likely to affect the accumulation of solute by guard cells and could conceivably cause stomata to open or close abnormally. The nature of

Fig. 2-4. 2) Percentages of leaf disks judged to be turgid plotted as functions of increasing relative water content. Each point represents at least 16 determinations on healthy and diseased cotton leaves. Relative water contents were rounded off to the nearest even percentage. **3)** Fresh weights of floating tobacco disks at various times after injection with water, *Pseudomonas fluorescens*, and *P. syringae*. Intercellular spaces remained filled with water or bacterial suspensions throughout the experiment. Open and closed symbols represent wilted and turgid disks, respectively. Each point is the mean of five disks and SE $\leq$ 1.6 mg for all points. **4)** Diffusive resistances of the abaxial surface of tobacco leaves at various times after injection with water, *P. fluorescens*, and *P. syringae*. Open and closed symbols represent wilted and turgid conditions, respectively. Each point is the mean of five measurements.

TABLE 1. Relative water contents and leaf solute potentials in healthy and diseased cotton plants

Plant	Visible condition of leaves	Relative water content (%)	Leaf solute potential[a] (bars)
Healthy	Turgid	82	−10.2[b]
	Wilted	61	−11.6
Diseased	Turgid	82	−11.4
	Wilted	65	−11.8

[a] Determined at 100% relative water content.
[b] Significantly different at $P = .05$.

TABLE 2. Leaf solute potentials in tobacco leaves treated by injection with water, *Pseudomonas fluorescens* or *P. syringae* in water suspension, or by freezing

Treatment	Visible condition of tissue	Leaf solute potential[a] (bars)
Water	Turgid	−7.8
P. fluorescens	Turgid	−8.4
P. syringae	Wilted	−0.8[b]
Frozen	Wilted	−0.6[b]

[a] Determined after disks were floated for 12 hr.
[b] Significantly different at $P = .05$.

the effect would depend on the relative turgor changes of guard cells and surrounding epidermal cells. Data in Fig. 4 indicate that stomatal closure results from the permeability increase (Table 2) (21) which is a part of the hypersensitive reaction of tobacco to *P. syringae*. Portions of leaves injected with *P. syringae* had significantly greater diffusive resistances to water vapor loss than did *P. fluorescens*- and water-injected portions of the same leaves 4-24 hr after treatment, and the increase in resistance of the *P. syringae* treatment preceded turgor loss. Clearly, transpiration is not a primary cause of the turgor loss, but comparison of Fig. 1 and 3 shows that transpiration can hasten turgor losses which are primarily due to solute loss.

The transpiration rates of plants infected by vascular and root pathogens have commonly been measured as functions of time after infection. Generally, the transpiration rates of plants with vascular disease are similar to the rates of healthy plants before symptoms are visible, but decrease markedly as wilt symptoms develop (2, 11, 14, 17, 26, 38, 40). Two root diseases, namely root stain in pine incited by *V. wagenerii* (27) and black shank in tobacco (34), are characterized by water stress or wilting and by decreasing transpiration rates during disease development.

Pathogen-induced changes in leaf water potential will affect transpiration rate because low water potentials in leaves cause stomatal closure (14, 16, 35) and have an aftereffect in inhibiting stomatal opening (19). The importance of water potential in determination of the transpiration rate of plants with vascular disease is illustrated by the work of Beckman et al. (2). They found that the transpirational behavior of banana plants infected with *P. solanacearum* could be reproduced in healthy plants by withholding water. Normally, stomatal closure in response to low water potentials provides a feedback mechanism by which plants tend to maintain some balance between the rates of water uptake and loss. Therefore, an analysis of transpiration relative to leaf water status not only will help to explain variation in transpiration rate but also will show those changes in transpirational behavior which are likely to affect plant water status.

The diffusive resistances of healthy and Verticillium-infected cotton leaves are plotted as functions of decreasing relative water content in Fig. 5. The diffusive resistance of diseased cotton leaves was invariably as high as or higher than the diffusive resistance of healthy leaves at the same relative water content. Similar results but based on leaf water potential were obtained for Fusarium wilt of tomato (14). Harrison (26) demonstrated that transpiration rates of healthy and V. albo-atrum-infected potato leaves are similar when measured as functions of decreasing relative water content. The above data indicate there is no change in the transpirational behavior of leaves affected by vascular pathogens which can contribute to wilting.

Transpirational behavior affects plant water status not only directly but also indirectly by affecting the rate at which soil water is consumed. Perhaps because of this central importance and the ease with which transpiration can be measured, transpiration is the water relations parameter which has been most commonly measured in studies on foliar disease. Rust infections have generally been found to increase transpirational water loss (15). More detailed experiments have demonstrated that prior to sporulation rust infections actually reduced transpiration, and that only as sporulation occurs does transpiration increase (15, 45). Rust infections in bean in all stages between flecking and sporulation inhibit stomatal opening in the light, and the increase in transpiration with sporulation is thought to result from damage to the cuticle (15). In contrast to healthy leaves and leaves affected by vascular pathogens, the diffusive resistance of bean leaves with sporulating uredia remained almost unchanged as relative water content was decreased from 95 to 50% (16). Evidently, stomatal closure does not limit transpiration in rust-infected leaves after sporulation (15, 16) and the rust-infected plant suffers from a continuous water loss. This water loss is of serious consequence any time evaporative demand exceeds the capacity of the plant-soil system to supply water at small water potential gradients (16).

Powdery mildew effects on transpiration rate are similar to those described for rust in that infections in the early stages of development reduce transpiration, whereas in the late stages of development infections increase transpiration, and the increase is particularly

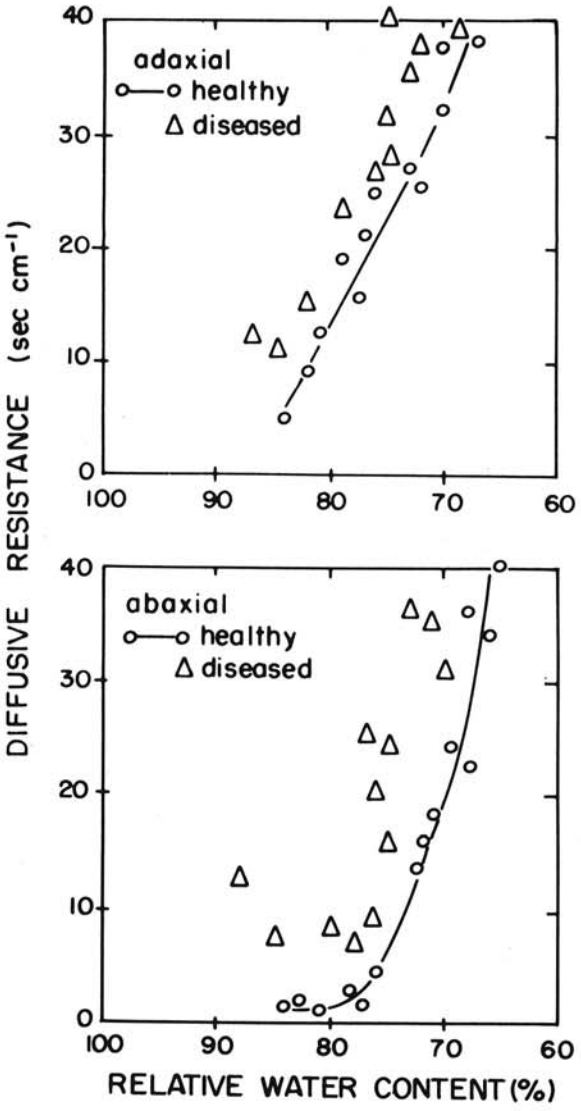


Fig. 5. Diffusive resistances of the adaxial and abaxial leaf surfaces of healthy and diseased cotton plants plotted as functions of decreasing relative water content. Each point represents one measurement of both parameters.

evident at night (32, 45). A recent study (30) suggests that powdery mildew infections partially inhibit opening and closing movements of stomata in barley. However, the relative importance of stomatal, cuticular, and perhaps mycelial water loss from powdery-mildewed leaves is not presently clear.

Infections of leaves by other pathogens have been shown to alter transpiration rate. For example, tobacco leaves infected with *Peronospora tabacina* have lower transpiration rates than noninfected leaves in the light but have higher rates in the dark (8). There are several reports that infection of leaves by viruses reduces transpiration (23). Infection of sugar beet by beet yellows virus has been clearly demonstrated by Hall (23) to induce partial stomatal closure. *Phytophthora infestans* causes abnormal stomatal opening in invaded regions of potato leaves (18). Perhaps this opening contributes to the necrosis which finally characterizes late blight lesions.

Water movement in the liquid phase.—An electrical analogue provides a useful description of water transport in the plant-soil system, and water in the liquid portion of the transpiration stream moves through a series of resistances and along gradients of decreasing water potential (7, 35). Resistance to water movement in soil increases markedly as soil dries, and growth in root length progressively determines the availability of water to plants as existing soil water is consumed (35). Rust and powdery mildew infections have been shown to reduce root growth (5, 16, 29). Other foliar pathogens as well as root pathogens may also be suspected of decreasing root distribution in soil.

Roots offer one of the major resistances to water movement within plants (3, 4, 7, 35, 37), and root resistance has been observed to vary with a number of factors including transpiration rate (7, 35, 37). Perhaps the most reliable estimate of resistance to water movement in infected roots was obtained with mycorrhizal soybean plants (33). The mycorrhizal relationship reduced root resistance in a low nutrient soil without increasing root growth. An addition of nutrients to the soil eliminated the difference between mycorrhizal and nonmycorrhizal plants. Infection of tomato roots by *Fusarium oxysporum* f. sp. *lycopersici* had little effect on resistance to water flow through root systems (14), but the black shank disease of tobacco markedly increased the apparent resistance of root systems (34). The observation that wilting in black shank is reversible only when water is supplied to the stem above stem lesions (31) suggests that xylem resistance is abnormally high. In contrast, wilting induced by tobacco etch virus in tabasco pepper is reversible when root tips are excised under water (20). Bushnell & Rowell (5) correlated premature desiccation of rust-infected wheat plants with a decline in root growth and CO_2 evolution. They concluded that premature desiccation resulted from a loss of root function. However, there is little doubt that cuticular transpiration was also a factor (16), and it should be noted that death of roots does not necessarily increase root resistance (37).

Estimates of resistance to water flow in the aerial portions of diseased plants have been made almost exclusively in plants with vascular wilt disease. Reduced dye or isotope movement in plants with vascular disease is frequently taken as evidence for a high resistance to water flow (11, 12, 38, 43), but solute distribution is not always indicative of the magnitude or location of resistances to water flow. Quantitative estimates of resistance have been obtained by forcing water through excised stem segments. By this technique the resistance of tomato stems with Verticillium wilt was found to be as much as 200 times the resistance of healthy stems (40) and the resistance of twigs with late stages of oak wilt was found to approach infinity (22). More commonly, vascular pathogens have been found to increase the

resistance of stem segments by 4 to 60 times the healthy value (11, 12, 38, 43). The resistance of stem segments from Fusarium-infected tomato plants decreased markedly with water flow, and accurate estimation of resistance in infected xylem was not possible by this technique (14). The possibility that similar errors occur in stem segments affected by other vascular pathogens has not been investigated.

Considerations of stomatal behavior and resistance to water movement in healthy and Fusarium-wilted tomato plants indicate that only a very large increase in xylem resistance can be expected to cause wilting. Stomatal closure in response to low water potentials (14) tends to prevent lasting effects of increased resistance on leaf water potential. The resistances of healthy stems and petioles were found to be very small when resistances were calculated from the dimensions of the xylem and Poiseuille's law (12, 13, 14) or from steady-state measurements of transpiration and leaf water potential (13). In the latter measurements, leaflets offered the chief resistance in the above-ground portions of healthy tomato plants. In a Fusarium-infected plant where the experimental leaf appeared turgid, the steady-state measurements showed that stem resistance was approximately 500 times the resistance of healthy stems. Petiole and leaflet resistances were normal, and the 500-fold increase in stem resistance only doubled the total resistance between ground level and the experimental leaf (13). When the experimental leaf of infected plants appeared wilted, stem resistance was high, but more importantly petiole resistance was commonly infinite (13). With the exception of Fusarium wilt in tomato and possibly oak wilt (22), the resistances to water flow which have been reported in plants with vascular wilt disease (11, 12, 38, 40, 43) do not appear to be large enough to account for the observed wilting. However, estimates of xylem resistance are sometimes higher than the estimates given here (3, 4), and there is abundant evidence that vascular pathogens do affect xylem function to some degree (11, 12, 38, 43). Furthermore, the data reviewed here indicate that parameters other than resistance to water movement do not contribute to wilting in vascular disease.

There is always the possibility that pathogens affect resistance to water movement in leaves, a resistance which is relatively large in healthy plants (3, 4, 13). Unusual resistance to water flow can be suspected to occur in leaves when pathogens cause only portions of leaf blades to wilt. This type of wilt symptom occurs in the stripe disease of wheat (36) and in Verticillium wilt of cotton. The difference in relative water content between various parts of the same cotton leaf is very much greater in Verticillium-infected leaves than in healthy leaves, as is shown in Fig. 6. These unusual differences in relative water content were not duplicated in healthy leaves when all but one major leaf vein were severed and are indicative of a very high resistance to water flow within infected leaf blades. Infected cotton leaves and Verticillium-infected chrysanthemum leaves (24) do not regain turgor when excised under

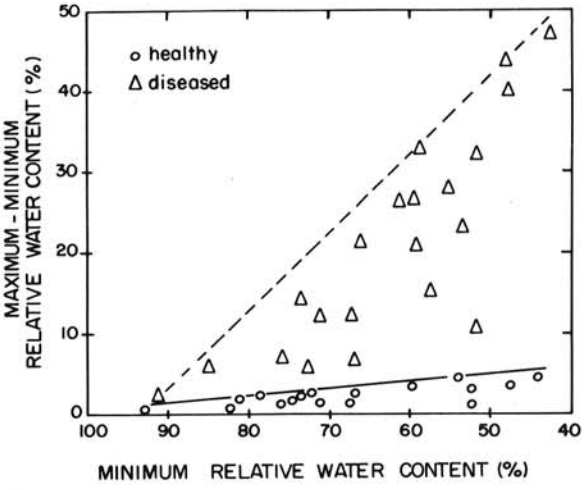


Fig. 6. Difference between the maximum and minimum relative water content observed within a healthy or diseased cotton leaf plotted as a function of the minimum relative water content for the same leaf. Each point represents one leaf, and the relative water contents of six disks from each leaf were determined individually. Lines are drawn through the extreme points for each treatment.

water at the base of the leaf blade. The lack of recovery in infected chrysanthemum was attributed to membrane damage (24), but no data were presented which preclude the occurrence of high resistance in infected leaf blades.

When disks were cut from wilted cotton leaves and disk margins were supplied with water, continuous measurements of water content with a beta gauge (1, 17) showed that the rate of water uptake at any given relative water content was the same in disks from healthy and Verticillium-infected leaves (*unpublished data*). Verticillium and Fusarium wilts in tomato have previously been shown not to affect rates of water uptake or loss by leaf disks (13, 40). Therefore, any unusual resistance to water movement in these leaves is probably associated with the xylem. Aspermy virus infection of tomato also interferes with water uptake in excised leaves, but apparently does not cause depression of leaf water potential in plants (41). Perhaps aspermy virus also increases liquid and vapor phase resistances to water loss from leaf cells to the air.

Concluding remarks.—The biochemical mechanisms by which pathogens may affect host water relations will not be reviewed here. Nevertheless, some comment on the common hypothesis that toxins mediate pathogen-induced changes in host water relations (12, 38, 43, 44) is in order. The toxin hypothesis is attractive in some of those cases where pathogens induce physiological changes in uninvaded portions of the host. Several wilt-inducing materials have been prepared from cultures of pathogens and in some cases from diseased plants, and the mode of action of these materials has been examined (44). Yet, no one has compared the

modes of action of a wilt-inducing toxin and the parasite on the physical aspects of host water relations, and from a water relations standpoint, the current literature on the role of wilt-inducing toxins in disease is incomplete.

In closing, it should be noted that any unusual level of water stress which results from infection will affect a number of physiological processes, including growth and yield. The work of Bushnell & Rowell (5) is one illustration of this point. They found a brief drought to be more damaging to the yield of rust-infected wheat plants than to that of healthy ones. The adverse effects of water stress on several physiological phenomena have been examined in a quantitative manner (e.g., the following paper). Only in the case of stomatal opening and photosynthesis in plants with vascular disease (14, 17, 26) has the possible role of pathogen-induced water stress in disease physiology been examined quantitatively. Hopefully, consideration of the physical aspects of plant water relations will become a more integral part of research on the physiology and yield of disease plants.

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