Influence of Low Plant and Soil Water Potentials on Diseases Caused by Soilborne Fungi

R. James Cook

Research Plant Pathologist, Regional Cereal Disease Research Laboratory, Agricultural Research Service, U.S. Department of Agriculture, Pullman, Washington 99163.

Scientific Paper No. 3915, College of Agriculture Research Center, Washington State University.

Introduction.—Plant water stress implies a strain or tension on the plant, or a weakening of it, because of an inadequate water supply. Soil moisture stress is sometimes used in soils literature to denote the combined effects of soil moisture tension and the osmotic pressure of the soil solution (34). In both cases the intent is to convey a concept of water deficit, using a qualitative expression without units of measure. No matter how descriptive and useful the term "stress" may be, it cannot thus far be defined and hence has limited use.

Water, like heat, is best described in terms of its potential energy (water potential), defined (39) as the capacity to do work relative to the work capacity of pure, free water. The units commonly used are joules/kg (free energy units) or bars (1 bar = 100 joules/kg) (5, 16, 45). Recall that water with a work capacity greater than that of pure, free water (e.g., water under pressure or elevated above the reference point) is at a positive water potential (+ bars). When the same principles of physics are applied, water with work capacity less than pure, free water (e.g., with salts or sugars dissolved, or adsorbed to a matrix such as soil) is at a negative water potential (−bars). The lower the water potential the greater the water stress, as we would usually use the term. In short, water potential provides a quantitative description of the status and dynamics of water in soils, plants, or microorganisms.

Water flows from areas of high to low potential energy in accordance with the second law of thermodynamics until the two areas are in equilibrium (at the same potential), unless prevented from doing so by a water impermeable barrier. The

flow of water from soil to roots (unsaturated conductivity), its flow into roots (absorption), its movement up the xylem, and its passage into the atmosphere (transpiration) are examples of water movement from higher to lower water potential.

Soil water potential is the sum of the matric potential (negative) and the osmotic potential (negative), but it includes certain other negligible components (39). The water potential of a plant cell is the sum of the osmotic potential (negative) and the pressure potential (positive or negative) (38). Water potential of the fungus likewise is the sum of the osmotic potential and turgor. A low soil water potential occurs with a low matric water potential (low soil water content), a low osmotic water potential (high salts), or both. A low plant (or fungus) water potential occurs with low osmotic water potential (high solutes), low turgor, or both. A low plant water potential will tend to lower the soil water potential, and vice versa, as the two approach equilibrium. The fungus water potential will be determined by its surrounding environment, plant or soil; but because of its small mass, it will have negligible effect on the water potential of its environment.

The effects of low water potentials on soilborne fungal diseases of plants can be classified as: (i) restrictive effects on pathogen growth; (ii) effects on pathogen-antagonist interactions; and (iii) effects on the host-pathogen interaction. This paper will give only a brief discussion of these three categories. Other recent reviews (16, 17, 28) may be consulted for more detailed and expanded discussions of the influence of water potential on soilborne diseases of plants.

Restrictive effects on pathogen growth.—A close correlation exists between the influence of water potential on disease and its influence on growth of the causal fungus (17). For example, *Phytophthora cinnamomi* Rands (1, 40), *Ophiobolus graminis* Sacc. (18, 36), *Rhizoctonia solani* Kühn (20, 36), and *Thielaviopsis basicola* (Berk. & Br.) Ferr. (36) all grow optimally at −5 bars or wetter, are reduced in growth rate by half or more at −20 to −25 bars, and grow slowly or not at all at water potentials below −50 to −60 bars; and all cause greatest disease in wetter soils (17). In contrast, the root, foot, stem, and seedling diseases caused by *Fusarium solani* (Mart.) Appel & Wr. emend. Snyd. & Hans. and *F. roseum* Lk. emend. Snyd. & Hans. are among the few associated with dry soils (18); all studies thus far show that growth by strains of these two species is optimal at −10 to −30 bars water potential, is reduced by half at −40 to −60 bars, and is prevented only if the water potential approaches or drops below −100 bars (15, 18, 36). Another example is Penicillium seed decay of wheat in dry seedbeds; capability of the pathogen for growth (26, 29) and occurrence of disease (27) are both associated with very low water potentials. The vascular parasites *F. oxysporum* Schlecht. emend. Snyd. & Hans., *Verticillium albo-atrum* Reinke & Berthe., and possibly *Cephalosporium gramineum* Nisikado & Ikata are apparently exceptional fungi, capable of growth at low water potentials (down to −100 bars and lower) (7, 31), but causing serious disease only in wetter soils (17). A reason for this exception has been proposed (17) and will be discussed further in this paper under host-pathogen interaction.

This close relationship between ability (or inability) to grow and to cause disease at low water potentials suggests that, in general, organisms incapable of growth below −40 to −60 bars water potential will be restricted primarily to areas (or years) of high rainfall or to irrigated crops, and that only those capable of growth well below −60 bars will be troublesome in the drier situations. Although this pattern seems to hold, such requirements within the pathogen seem extraordinary, since the crop plants themselves commonly wilt at −10 to −20 bars. Why must a pathogen be capable of growth so far below the wilting range of its host to cause disease? At least part of the answer probably relates to the fact that most soilborne pathogens exist and infect plant parts largely in the tillage layer where drying occurs first and most intensively. Except where rains or irrigations are frequent or shading is heavy, the pathogen is generally exposed to water potentials considerably below those of its host with roots deep in moister horizons of the soil profile.

Good evidence for disease restriction in dry soil through a water potential restriction of the pathogen itself is provided by our studies in the Pacific Northwest, USA, on take-all of wheat caused by *Ophiobolus graminis*. This disease is associated throughout the world almost exclusively with wet soils (22, 24). In our region, it occurs in wheat under irrigation or in the high-rainfall areas west of the Cascades (14). Losses are most serious when the pathogen infects early, or when it can spread relatively unchecked throughout a season. In our dryland winter wheat fields, water potentials in the tillage layer at fall seeding time are commonly below −100 bars. The seed is planted and germinates in the moist soil 15-20 cm deep, beneath a dust mulch. Subsequent fall rains moisten the tillage layer, but usually by then winter temperatures are low and limit the fungus. A period of favorable water potential and temperature occurs briefly in spring, but shortly thereafter (generally in May) the surface 10-15 cm is again too dry for *O. graminis*, and by June the surface 30 cm is again near −100 bars. Clearly, such low water potentials would limit the growth of *O. graminis*, whose growth essentially ceases at −40 to −50 bars (18) (Fig. 1).

Water potentials restrictively low to plant pathogens undoubtedly also develop in furrow-irrigated fields where the host is grown on elevated beds (17). A dry soil layer generally persists at the surface of these beds, because capillary flow upward from the irrigation furrow does not keep pace with evaporative loss from the bed surface. In addition, salts accumulate in zones near the surface which lower the total soil water potential still more (43, 44), and undoubtedly helps to determine the kinds of pathogens which become active. Probably

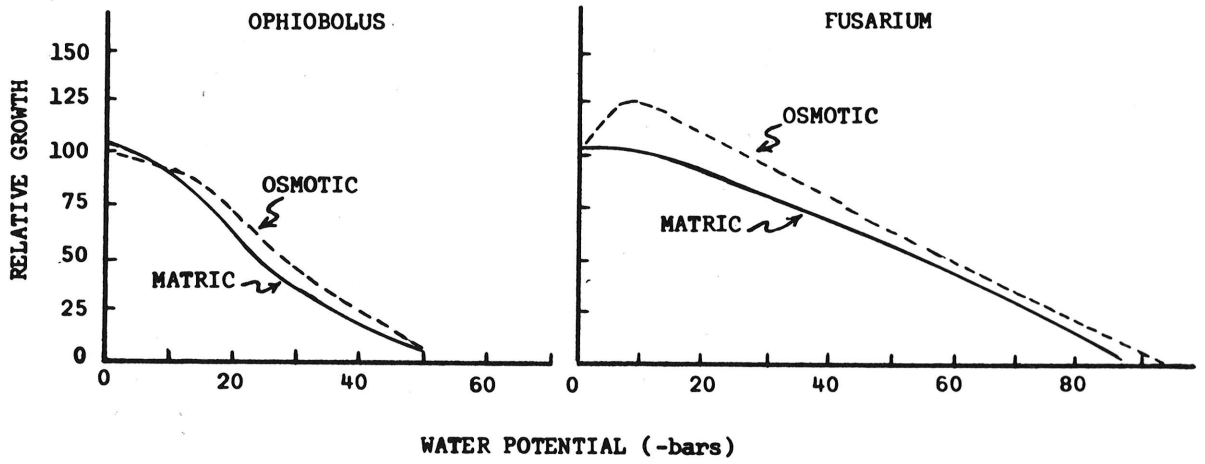

Fig. 1. Relative mycelial growth of *Ophiobolus graminis* and *Fusarium roseum* f. sp. *cerealis* 'Culmorum' in sterile soil at different matric vs. osmotic water potentials. Redrawn from Cook et al. (18).

those pathogens capable of growth at very low water potentials; i.e., *Fusarium* species and *Verticillium albo-atrum*, will be restricted the least by the high salts.

Many soilborne plant pathogens are actually stimulated in growth, not depressed, by a slightly lowered osmotic water potential. The phenomenon was apparently discovered first in certain food spoilage bacteria (37). Halophilic bacteria have a high internal salt content and their enzyme systems are salt tolerant, sometimes even salt dependent (11). Even with nonhalophiles, however, some salts in the external medium are generally favorable, possibly because they help the organism maintain a proper internal osmotic potential (2, 10, 37). No growth stimulation has thus far been noted where the water potential control is accomplished strictly through matric means. As pointed out (2), with reduced matric water potential the organism may be limited by its inability to assimilate adequate salts readily from the medium to lower its internal water potential, yet this it must do to maintain turgor as the soil dries. Without an external source of salts, the organism must rely on uptake of nutrients and intracellular synthesis of the solutes needed to satisfy the internal water-potential requirements, which is probably a less efficient mechanism than having salts readily available.

The following are examples of the stimulatory effects of osmotic water potentials on soilborne plant pathogens. *Fusarium roseum* f. sp. *cerealis* (Cke.) Snyd. & Hans. 'Culmorum' grows about half again as fast at −8 bars as at −1.2 bars osmotic water potential (18) (−1 to −1.5 bars is the water potential of ordinary nutrient agar media). In contrast, it grows about the same or slower at −8 vs. − 1.2 bars matric water potential. *Sclerotinia borealis* Bubak & Vleugel grows 5 times as fast at −20 to −30 bars as at −1.2 bars osmotic water potential (6). *Phytophthora cinnamomi* (1) and *Rhizoctonia solani* (20) are also stimulated to greater growth rates when the osmotic water potential is dropped to about −5 to −10 bars. *Verticillium albo-atrum* apparently does not grow at temperatures above 32 C on standard agar media but grows at 35 C if the osmotic water potential of the media is lowered to about −25 bars (31). The optimum temperature for growth of *Cercosporella herpotrichoides* Fron. is progressively higher as the osmotic potential is lowered from near zero to about −20 bars (8). *Ophiobolus graminis* is thus far unique among the fungi studied in that it responded about the same to osmotic and matric water potential and showed no clear optimum with reduced osmotic water potential (18). There is no evidence that the salts as such are toxic to fungi as they are to higher plants, nor do they act as nutrients. Instead, the effect can be interpreted in terms of the osmotic water potential created.

The differential response of pathogens to matric vs. osmotic water potentials is important. Pathogens are exposed mainly to matric water potentials in soil, but as they enter the host, osmotic control may dominate. This emphasizes the importance of using both an osmotic and a matric system in conducting water-relations studies.

Effects on antagonist-pathogen interaction.—A low water potential may free a soilborne pathogen from the usual dampening influence of the background soil microbiota, by limiting growth of other organisms more than that of the pathogen itself. Conversely, it may accentuate antagonism by limiting growth of the pathogen more than that of the associated microbiota. The former probably applies mainly to those pathogens able to grow at the lower water potentials, and the latter to those with a relatively high water-potential requirement. These contrasting effects undoubtedly reinforce the pattern noted above; namely, that pathogens capable of growth in dry soil are troublesome primarily under these conditions and those less able to grow in dry soils are more troublesome in wet soils.

The luxuriant growth of *Fusarium roseum* f. sp.

cerealis 'Culmorum' in dry soil is an excellent example of escape of antagonism by virtue of ability to grow at very low water potentials (15). In soil wetter than −8 to −10 bars, chlamydospores germinate, but fungus growth ceases shortly thereafter, new chlamydospores form, and germ tubes lyse; at lower water potentials the fungus grows in soil for a week and longer after chlamydospore germination (Fig. 2). Growth of the pathogen itself is prevented only at water potentials below −85 to −90 bars. The lack of continued growth at the higher water potentials is apparently due to effects of soil bacteria that are relatively inactive below −8 to −10 bars (16). In an investigation of its ability as a saprophyte, the fungus colonized a progressively higher percentage of wheat straw in soil as the soil water potential was lowered to about −90 bars (13); even though based on pure culture performance, activity should have been progressively less, not more, down to −90 bars. Apparently, reduced antagonism of this pathogen in the drier soil more than offset its handicap of reduced growth rate.

One probable cause of hyphal lysis in soil is starvation due to rapid depletion of the nutrients by soil bacteria. Support for this explanation is evident by the very remarkable cessation of lysis of germlings with slight drying of the soil. Most actinomycetes and fungi are active below −8 to −10 bars (26) and consequently must not play a very significant role in the lysis of this fungus. Obviously the water potential of the soil must be taken into account when the importance of lysis in the life cycle of a soilborne pathogen is assessed.

The suggestion that low water potentials accentuate antagonism of pathogens with high water-potential requirements is speculative at this point, but it seems probable. For example, a pathogen such as *Ophiobolus graminis* that grows progressively more slowly as the water potential drops below zero will certainly lose competitive advantage, particularly among saprophytic fungi and perhaps actinomycetes. Any change in the physical environment detrimental to growth of the pathogen, unless it confers proportional constraints on competitors, will exert an impact on the pathogen disproportionately large for the magnitude of change. In essence, the slightly unfavorable environment provides partial control, and soil organisms less affected by the condition add to or reinforce the control.

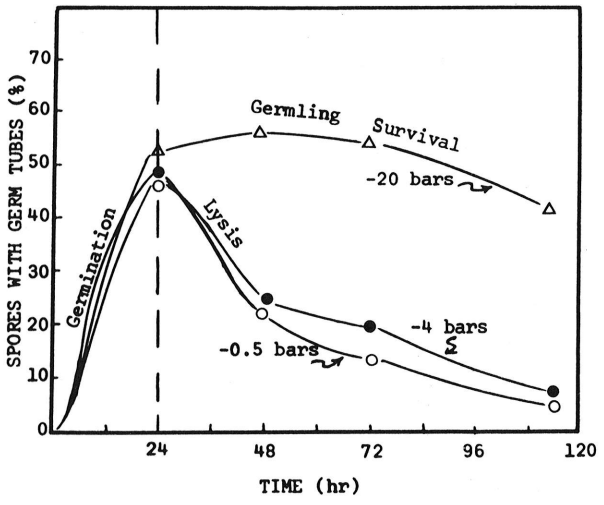

Fig. 2. Preserving influence of a low soil water potential (−20 bars in this case, but the same results were obtained over the range −10 to −75 bars) on germlings of *Fusarium roseum* f. sp. *cerealis* 'Culmorum' in nonsterile Ritzville silt loam amended with glucose (2,500 ppm C) and ammonium sulfate (250 ppm N). Redrawn from Cook & Papendick (15).

A low water potential may also prevent growth of would-be antagonists of resting propagules. This is the case with *Cephalosporium gramineum* (cause of Cephalosporium stripe disease of wheat), which survives as mycelium in wheat straw. At 15 C the fungus survives well in buried straw at soil relative humidities (RH) below 82% (about −270 bars) because would-be antagonists are inactive. However, between 90 and 86% RH (−150 to −210 bars), *Penicillium* species overrun it. Above 96% RH (about −75 bars), *C. gramineum* produces an antibiotic that protects against colonization by soil fungi (7). A similar phenomenon of good survival at 33 and 100% RH but poor survival at 75-85% RH has been reported for *Gibberella zeae* (Schw.) Petch in straw buried in nonsterile soil (9).

Influence on the host-pathogen interaction.—Very little experimental information exists on the influence of plant water potentials and, more particularly, low plant water potentials on the host-pathogen interaction. However, when what we know of the water-potential concept is applied to the limited existing data and to past observations, some very interesting and probably significant effects become evident.

Knowing that water flow occurs in response to water-potential gradients, and that flow will occur until equilibration is complete; i.e., no difference in potential exists, we can assume that:

(i) A fungus situated in host parenchyma tissue will be in equilibrium with the water potential of that tissue; i.e.:

$$\text{osmotic}_{\text{fungus}} + \text{turgor}_{\text{fungus}} = \text{osmotic}_{\text{host}} + \text{turgor}_{\text{host}}.$$

As the tissue breaks down, the turgor component of host cells will disappear and the total water potential of the fungus will then approach the osmotic water potential of the necrotic host tissues.

(ii) A fungus cell situated in the vascular elements will be in equilibrium with the water potential of the vascular fluid (primarily negative pressure; i.e., fluid under tension, but some osmotic).

Thus, inside the host a cortical-decay organism is exposed primarily to osmotic, and a vascular parasite primarily to matric (negative), water potential, unless it spreads laterally into stem or root parenchyma

tissues. Performance of a pathogen in pure culture in response to low osmotic vs. matric water potentials should indicate how it might respond to comparable water potentials if inside a host. Any deviation from normal pure-culture response will probably depend on the host itself; i.e., the extent to which host resistance factors are affected.

It seems probable that disease will be least severe at water potentials more favorable to metabolic activity of the host than to that of the pathogen, and most severe when the converse is true. Water deficits in particular are known to limit many of the normal and necessary physiological processes within higher plants (19, 23). As with antagonist-pathogen interactions, if the particular low water potential is adverse to the host but does not impose comparable constraints on the pathogen, disease should be favored, but if it restricts pathogen physiology relatively more than that of the host, disease should be absent or mild.

The occurrence of severe Fusarium crown and foot rot of wheat in the Pacific Northwest is probably the result of plant water potentials that are harmfully low to host physiology but nearly ideal for the pathogen, *F. roseum* f. sp. *cerealis* 'Culmorum'. Past field observations had established that the disease occurs
area (20-35 cm annual precipitation) of eastern Washington and northeastern Oregon (12). Growers refer to it as *dryland foot rot*. The near absence of lysis of pathogen germlings in dry soil (15); i.e., the ability of the pathogen to escape antagonism when water potentials are low, may account in part for the general confinement of disease to dry areas, but does not fully explain the distribution. Low plant water potentials are equally important, perhaps even more so than simply lack of antagonism. Before the magnitude and epidemiological significance of low host water potentials are discussed further, however, it will be necessary to describe the crop-soil-water system itself.

Dryland wheat is produced in the Northwest on summer-fallowed soil; i.e., with the precipitation received during the crop year plus that stored in the profile from the previous year. The soil ranges from silt loam to fine sandy loam and is loess deposited generally 180 cm or more deep. This profile is normally at field capacity (40-45 cm of water in 180 cm of soil) by late March or early April of the crop year. This must subsequently provide most of the water for the crop, since rainfall occurs largely in the cooler months (October - April). As the roots penetrate into the profile, they consume the finite quantity of water to progressively greater depths. A normal crop yielding 40-50 bu/acre removes all available water (20-25 cm; R. I. Papendick, *unpublished*) to at least 180 cm before ripening (32).

Culmorum exists almost entirely in the surface 10 cm of soil, which is the tillage layer. The pathogen establishes itself in the roots and crown in the fall (12), but disease remains mild, often inconspicuous, until water stress begins to develop. Water stress alone is well known to hasten maturity of wheat, but when combined with the rapid and total crown and basal stem decay that characteristically follows, plant death is ruinously premature.

We are attempting to relate plant water potentials to disease development by monitoring both leaf water potentials [by thermocouple psychrometry for the osmotic component and pressure bomb for total water potential (5, 45)] and soil water extraction (by neutron probe to 210-cm depth and by thermocouple psychrometry) for different wheats subjected to different fertility rates, seeding dates, and row spacings under field conditions (33) (R. I. Papendick & R. J. Cook, *unpublished data*). Through May, all plants regardless of treatment have leaf osmotic water potentials of −20 to −25 bars (−15 to −20 bars total; −4 to −5 bars turgor during the day). A late-sown plant with little nitrogen (N) and exposed to little competition will rarely drop below about −30 bars osmotic water potential by the late dough stage. Fusarium crown and foot rot remains mild or nonexistent in these plants, in spite of infection. Plants in dense plantings and with high N, on the other hand, because they grow more luxuriantly, consume the limited water more rapidly and develop leaf osmotic water potentials down to −40 bars or slightly lower by late June (dough stage) (Fig. 3). Severe disease develops in these plants. Moreover, these low water potentials relate to the N treatment and are not induced by the disease itself, since comparable values can be measured in noninfected plants treated with high N in dense stands. The stimulatory effects of high N on stress, and on disease, can be offset almost completely by spreading the rows farther apart, from 30 or 40 cm to 80 or 90 cm. This indicates that N soil; is not responsible for increased disease, but rather water stress caused by plant response to high N. Early seeding, because it also promotes greater plant growth, also enhances stress—and disease. Similarly with varietal types, those that grow luxuriantly during the season generally show greatest stress and disease, and those that grow more conservatively show little or no disease unless fertilized heavily.

The development of severe disease in plants at −30 to −40 bars osmotic water potential cannot be explained entirely in terms of the good growth made by the pathogen at this osmotic water potential. If it were this simple, disease should be more severe in April and May when osmotic water potentials are the most ideal for Culmorum, about −20 bars. Instead, water potentials in the very dry range are undoubtedly harmful to the wheat plant and this must tip the balance between host and pathogen, making disease development possible.

In contrast to that of Culmorum, growth of *Ophiobolus graminis* is greatly subdued at −20 to −30 bars and essentially prevented at −40 bars. This pathogen must surely be restricted in growth within host by such low osmotic water potentials of the tissues. This would further explain the restriction of take-all to the wetter areas.

A similar rapid disease progression after a period of water stress has been described for charcoal rot of

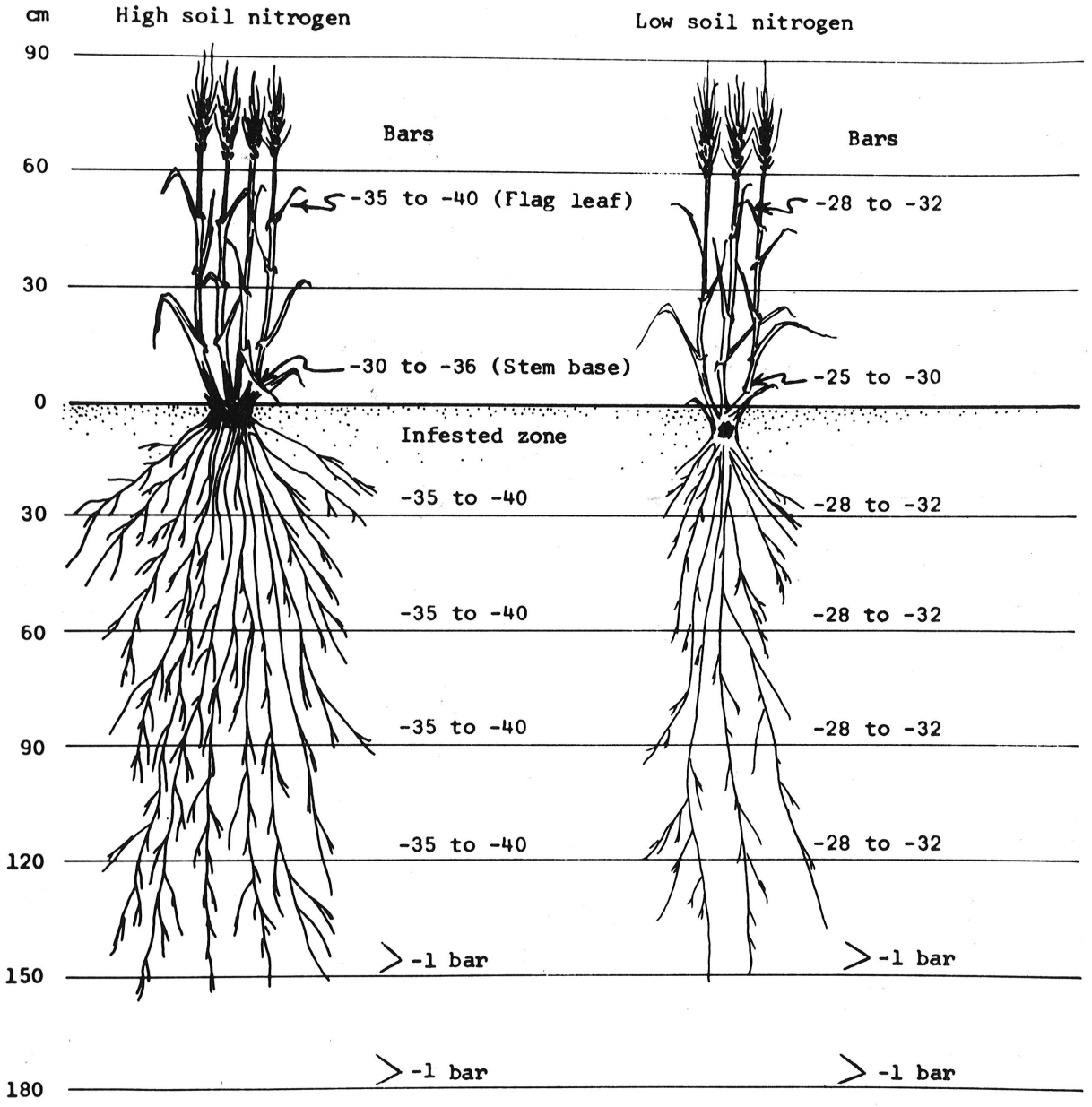

Fig. 3. Typical soil and plant water potentials recorded in the field for *Triticum aestivum* 'Nugaines' (winter wheat) at middough stage, grown under Northwest USA dryland conditions with high (60-120 lb N/acre) vs. low (20-40 lb N/acre as residual in the profile, none applied) nitrogen fertility, in the presence of a soil infestation of *Fusarium roseum* f. sp. *cerealis* 'Culmorum'.

sorghum (21) and cotton (25) caused by *Macrophomina phaseoli* (Maubl.) Ashby. With sorghum, the stress apparently permits established but previously latent or subdued infections to proliferate suddenly, resulting in a quick onset of disease.. Water stress is most conducive to the disease if it occurs between bloom and maturity (21). Possibly, like Culmorum, *M. phaseoli* may be capable of growth at fairly low water potentials. Also, as suggested for Culmorum, the progress of *M. phaseoli* within the host may go relatively unnoticed until host barriers are lowered by water potentials adversely low to the plant, at which time the pathogen spreads relatively unchecked.

Bruehl & Manandhar (8) point out that the shift in optimal growth in *Cercosporella herpotrichoides* from low temperatures at high water potentials to progressively higher temperatures as the osmotic potential drops is probably in line with the kind of temperature and osmotic-potential shift that occurs

in the wheat plant as the season progresses. Prolonged cool, moist weather is required for dissemination and infection, but once in the plant, this pathogen comes under the influence of the osmotic water potential of the stem. A common observation is that established infections continue to grow in spite of the onset of a warm, dry season. As the season progresses, the temperature rises within the stem, and the osmotic water potential drops. This keeps conditions somewhat near optimal for growth of the pathogen.

Vascular parasites depend on the transpirational stream within their host to carry them upward as microconidia. As the soil water potential drops, that of the plant also drops, and at some critical point stomata close and transpiration largely ceases. When this happens, presumably the upward spread of the pathogen will then cease as well (17), or be slowed enough to permit the host occlusion reaction (3, 4) to seal it off. This may explain the long-established fact that vascular diseases are characteristically more severe in wet than in dry soil (11, 30, 35, 41, 42). Possibly the dry soil, by cutting off transpiration, prevents internal spread of the pathogen. This is also consistent with the field observations that wilt develops most rapidly during hot, dry weather; this kind of environment will accelerate transpiration—and internal spread of the pathogen. Finally, this could explain why vascular parasitic fungi are exceptional in being capable of growth at water potentials down to −90 to −110 bars and lower, but cause the most severe disease in wetter soils; infection may actually occur in drier soils, but internal spread and hence wilt symptoms require wet soil. It would appear that in the case of wilts, low water potentials make the plant more resistant rather than more susceptible to disease.

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Pathogen-Induced Changes in Host Water Relations

J. M. Duniway

Department of Plant Pathology, University of California, Davis 95616.

The author thanks J. E. DeVay and J. M. Gardner for their collaboration in the experiments on cotton and tobacco, respectively.

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