Melting-out of Kentucky Bluegrass, a Low Sugar Disease

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ABSTRACT

Kentucky bluegrass succumbs to melting-out disease when sugar levels are low in turf tissue. The incidence of disease and the content of leaf sugar were negatively correlated to a highly significant degree in the cases of five varieties of bluegrass. Leaf sugars are lower and disease is more severe in turf mowed at 2.5 cm than when mowed at 5.0 cm. Shading reduces the sugar content in the host, and increases disease. An enrichment of host with sprays of glucose initially reduces disease severity but eventually increases disease by encouraging saprophytic growth of the pathogen in sod. Disease increased when host sugar levels were decreased in four ways: cutting height, shade, sugar supplement, and comparison of varieties. Thus, melting-out is a low sugar disease. Sugars may confer resistance to the host by being synthesized into fungitoxic phenols during invasion by the fungus or by inhibiting the synthesis of macerating enzymes by the pathogen. Phytopathology 60:1276-1278.

Additional key words: Helminthosporium vagans.

Horsfall & Dimond (4) observed that certain plant-pathogenic fungi preferentially attack host tissue low in sugar, and have referred to these as low-sugar diseases. *Helminthosporium vagans* Drechs. attacks Kentucky bluegrass turf (*Poa pratensis* L.) when the grass is growing succulently. Succulent growth in bluegrass turf, stimulated by close mowing and high N fertilization, encourages disease (1, 7). Low light intensity, which causes etiolated growth of the host, increases susceptibility to disease (6). Apparently, melting-out is a low-sugar disease.

To test this hypothesis, the relationship between melting-out disease and sugar content of leaves of Kentucky bluegrass was explored.

MATERIALS AND METHODS.—Melting-out disease was examined under conditions in which leaf sugar was varied four ways: (i) by variety; (ii) by cutting height; (iii) by shade; and (iv) by foliar sprays of glucose. The severity of disease in naturally infected turf browned from melting-out was estimated visually by using the Horsfall-Barratt (H-B) grades (3). Grades were then converted to per cent area of diseased turf.

Turf established for 3 or more years and growing in Cheshire sandy loam was fertilized in spring and fall with 10:6:4 fertilizer (50% insoluble N) at a rate of 4.9 kg/100 m^2 (10 lb./1,000 ft^2). Cutting heights were maintained at 2.5 and 5 cm (1 and 2 inches). Varieties of bluegrass examined were: Merion, Newport, Windsor, Park, and common Kentucky.

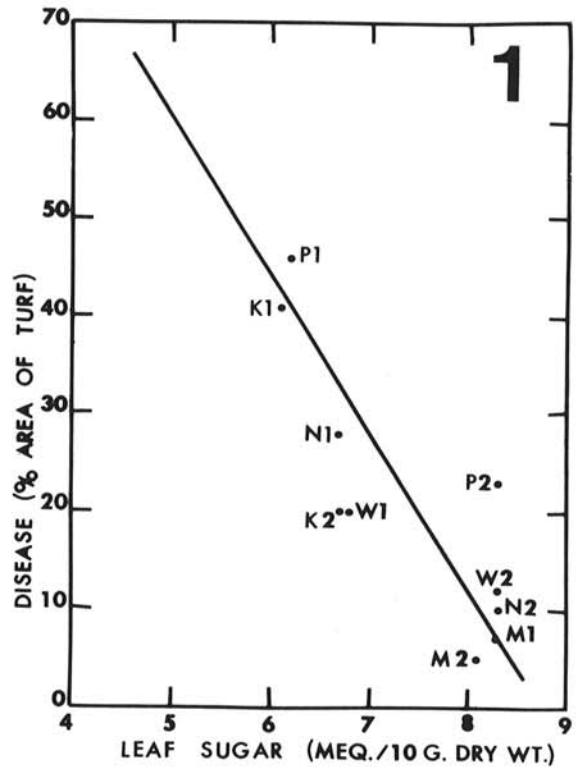
Four types of cloth that permitted 70, 50, 30, and 10% of sunlight to pass through were employed to give 30, 50, 70, and 90% shade, respectively. These cloths were mounted to frames (3 × 7 ft) which held the cloths 30 cm above turf in the field. Three frames of each type of shade cloth were employed. The shade treatments were initiated 1 May on turf of common Kentucky bluegrass maintained at 5 cm. After 4 weeks of shade, disease was estimated. Leaves developing under 5 days of shade were harvested for sugar analysis.

Leaves of Kentucky bluegrass turf maintained at a cutting height of 5 cm were sprayed with an aqueous solution of glucose. The solution was applied at a rate of 40.8 liters of spray/100 m^2 (10 gal/1,000 ft^2) of the turf with a proportional sprayer operating under a water pressure of 3.5 kg/cm^2 (50 psi). Glucose at rates of 91.6 and 305.2 g/100m^2 (3 and 10 oz/1,000 ft^2) was applied weekly for 4 weeks, starting in the last week of April. The treatments were repeated 3 times. Disease levels were evaluated each week for 2 months, starting when glucose was initially applied. H-B grades were plotted against time to obtain the increase in disease per day.

Reducing sugar of grass leaves was measured by the dinitro salicylic acid method of Miller (8). Samples of grass in the field were clipped, placed in plastic bags, and immersed in ice water for transportation to the laboratory. The samples were frozen and analyzed the next day. Samples of leaves (100 mg fresh wt) immersed in 6 ml water were boiled for 15 min to extract the reducing sugars. Three ml of the sugar extract were mixed with reagents. The mixture was placed in boiling water for 5 min to develop color, then cooled in ice water. The mixture was diluted 1:5 and analyzed photometrically at 590 mμ. The analysis is based on reduction of dinitrosalicylate to aminonitrosalicylate. Leaf material did not interfere with this reaction. The measure of optical density was converted to meq reducing sugar per g fresh wt of leaf by means of a standard curve of the reagents with glucose. The data were then expressed as meq per g dry wt of leaf.

RESULTS.—Natural infection by *H. vagans* developed in plots of bluegrass varieties in mid-May. Samples for sugar analysis were harvested at the time of disease estimation from plots maintained at 2.5 and 5.0 cm heights. The amount of diseased turf varied with variety of grass, and was greater in closely clipped turf. Sugar content of leaves varied with variety of grass and was lower in short turf. The correlation coefficient between sugar content of leaves and amount of disease was minus 0.96 (Fig. 1). Disease increases with the reduction in sugar content of leaves. Close mowing tends to lower the content of leaf sugar and increases disease.

The effects of shade on melting-out disease and sugar content of the host were studied independently. Starting on 1 May, turf was shaded continuously with



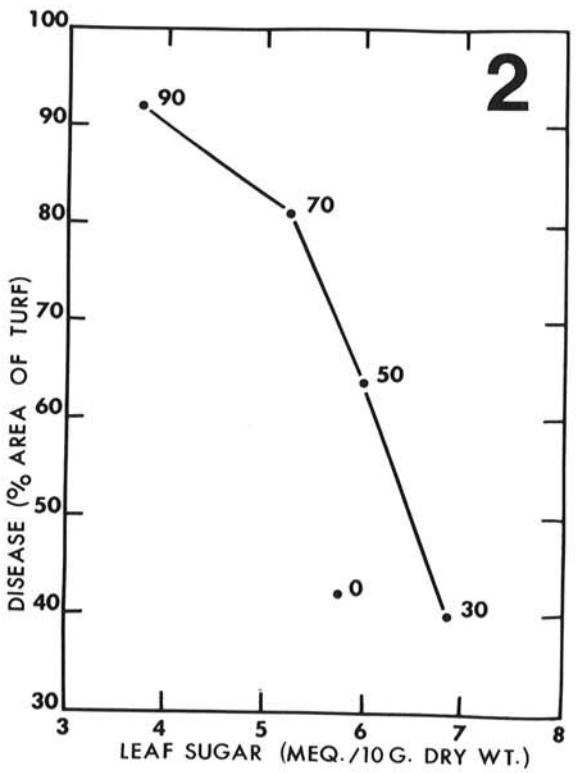


TABLE 1. Effect of glucose on the development of melting-out disease of Kentucky bluegrass turf

Glucose spray[a] (g/40.8 liters/100 m²) (oz/10 gal/1,000 ft²)	Development of disease[b] H-B units/day		
	2nd week	5th week	7th week
0	0.20	0.03	0.03
91.6 (3)	0.17	0.07	0.03
305.2 (10)	0.13	0.06	0.04

[a] Sprays of glucose applied at 0, 1, 2, 3 weeks.
[b] Horsfall-Barratt ratings recorded weekly were plotted against time to obtain rates of increase in disease per day.

cloth that blotted out 30, 50, 70, and 90% of sunlight. After 4 weeks of treatments, the area of turf brown from disease was estimated. Reducing sugars were analyzed in leaves taken from turf after 5 days of treatment. As intensity of shading increased, disease increased and leaf sugar decreased (Fig. 2).

The application of glucose to leaves of Kentucky bluegrass may affect disease two ways. The rate of disease development was initially reduced, then increased, and eventually was equal to that of the unsprayed turf (Table 1). The reduction in disease is proportional to the log-concentration of glucose in the spray. However, 5 weeks after sugar supplements were applied, fungal mycelium was growing on turf receiving sprays of glucose. Apparently the latent increase in disease is caused by the saprophytic build-up of inoculum of *H. vagans* on sugar in the sod. Presumably 3 weeks after the treatments were stopped, the pathogen had exhausted the external supply of glucose. At this time the rate of development in the sugar treated and in the check plots was approximately the same.

DISCUSSION.—When the sugar content of bluegrass is decreased, the turf succumbs to melting-out disease. Varieties of bluegrass susceptible to melting-out contain less sugar than varieties that resist the disease in the field. Closely clipped grass contains less sugar in leaves and succumbs more severely to disease than grass of a higher cut. On mowing grass closely, a large portion of leaf tissue in which sugars are synthesized is removed. Because the requirements for sugars are not affected appreciably, close mowing causes a net reduction in sugar content in the remaining leaves. The amount of disease induced by shade is proportional to the lowering of sugar content of the host. Shade reduces the intensity of sunlight received by the turf and reduces photosynthesis from which sugars are produced.

Initially, supplements of sugar reduced severity of melting-out disease, but later increased disease. The pathogen, *H. vagans,* may grow on sugar outside of the

Fig. 1-2. 1) Effect of leaf sugar on melting-out disease as influenced by variety and cutting height of turf. Variety of bluegrass: P, Parks; K, Kentucky; N, Newport; W, Windsor; and M, Merion. Cutting heights: 1 and 2 inches, respectively. The curve is the regression line of leaf sugar on disease ($b = -160^{**}$). 2) Effect of leaf sugar on melting-out disease as influenced by shade. The numbers designating position of data are per cent shade received by common Kentucky bluegrass.

host. Turf that can resist the pathogen may succumb to disease in the presence of a large amount of inoculum. A sugar supplement can confer resistance to disease, but for use in control of melting-out disease, the sugar must not induce the pathogen to grow saprophytically.

Undoubtedly, treatments to lower sugar content, especially a short cut and shade, directly affect host metabolites in addition to sugar. Moreover, a curtailment of the sugar supply will surely alter the composition and contents of compounds whose syntheses require sugar as a source of carbon. Changes in host metabolites can affect disease. Large amounts of glutamine exudated to the leaf surface may be required for *Helminthosporium sorokinianum* to infect bluegrass (2), but this is a highly specific case. Most facultative pathogens can grow on a wide variety of nitrogenous and carbon sources. An alteration of available food is not likely to play a major role in disease.

The disease resistance conveyed to Kentucky bluegrass by a high content of reducing sugar in the grass may arise from one of two means. A high sugar content may be required to inhibit the synthesis of macerating enzymes by the pathogen or to synthesize fungitoxic compounds by the host. Patil & Dimond (10) have shown that synthesis of polygalacturonase by *Fusarium oxysporum* f. sp. *lycopersici* and symptoms of tomato wilt are repressed in the presence of glucose. *Helminthosporium vagans* produces little pectinase activity in growth medium containing glucose, although the fungus is capable of producing considerable pectinase activity in growth media lacking simple sugars (S. Patil, *unpublished data*). The fungus, which penetrates leaves of bluegrass by way of the middle lamella between epidermal cells (9), may dissolve pectic substances between walls of adjacent cells enzymically as it enters the host. Conceivably, bluegrass tissue high in sugar content may repress production of pectic enzymes by *H. vagans* so that the pathogen cannot penetrate through the epidermal layer.

Many higher plants produce large amounts of phenolic compounds in response to an invading pathogen or wound (5). Some of the phenols are fungitoxic. Sugar may be required by the host in the synthesis of the phenols. Conceivably, in melting-out, pathogenesis is retarded by the production of fungitoxic compounds in bluegrass tissue high in sugar content on invasion by *H. vagans*. However, grass of low sugar content may fail to synthesize an effective concentration of toxicants when invaded by the pathogen and pathogenesis proceeds unhampered.

Melting-out is a low sugar disease, conforming to the hypothesis of Horsfall & Dimond (4). The amount of disease developing from natural inoculum of *H. vagans* increases with reduction in sugar content of bluegrass when that sugar content is varied by any of the four methods. *Helminthosporium vagans* is the third species of the *Helminthosporium* genus reported to be a low sugar pathogen. *Helminthosporium sativum* and *H. victoriae,* causing leaf spot of wheat and blight of oats, respectively, appear to attack plants of low sugar content (4).

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