Epidemiology and Control of Bean White Mold

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ABSTRACT

Epidemics of white mold occurred 8 to 14 days after full bloom in snapbean plantings irrespective of planting dates and environmental conditions during the blossom period. First infections usually occurred in the axils of lower branches at the site of lodgment of cast bean blossoms, and were caused by mycelium that emerged from blossoms in which the causal fungus, *Sclerotinia sclerotiorum,* was established. Subsequent spread occurred by in situ contact of healthy with infected tissues, and by distribution of infected plant parts by various agents. Direct invasion of healthy growing bean tissues by the primary infectious agent was not observed. Senescent and dead blossoms invaded by the fungus thus are essential intermediaries in disease development. Foliage sprays with benomyl applied a few days before full bloom provided effective control, whereas sprays applied after full bloom did not. Effectiveness of benomyl was attributed to its systemic translocation into developing bean buds and blossoms, and to retention of its fungicidal activity in senescent and dead blossoms. Phytopathology 61:669-674.

Additional key words: Phaseolus vulgaris, Botran, Thiabendazole, *Botrytis cinerea,* gray mold.

White mold of beans (*Phaseolus vulgaris* L.) caused by *Sclerotinia sclerotiorum* (Lib.) d By. has caused significant economic losses in snapbean production areas in New York in recent years. Bean production has been discontinued in some highly desirable locations because of repeated, heavy losses from white mold. Current production practices such as high plant populations and heavy fertilization to obtain maximum yields, together with repeated bean culture in the same fields, have undoubtedly contributed to the present white mold problem.

Foliage fungicides have provided various degrees of control (1, 4, 5, 11, 12, 13, 18, 19). Attempts by growers to control the disease by one or two sprays applied during the period of pod development have not been successful. Three or more spray applications of Botran (2,6-dichloro-4-nitroaniline) at weekly intervals beginning at full bloom have provided moderate-to-effective control in experimental plots (1, 11, 12, 13). However, this control method is considered economically unfeasible by processing snapbean growers. Thus, rather than resorting to fungicides for control, snapbean growers frequently harvest their crops about 4 days earlier than the scheduled harvest date in order to eliminate the risk of total crop loss. This early harvest may result in a 30% reduction in yield.

Because of the need for an effective and economically feasible method of control of bean white mold, research on the epidemiology and control of this disease was initiated at the New York State Agricultural Experiment Station, Geneva, in 1966. Results of epidemiological observations and tests on control by selected fungicides are presented in this report.

Epidemiology.—Environmental conditions.—Commercial snapbean plantings in 12 fields with recent histories of bean white mold were observed during three seasons for the first occurrence and subsequent development and spread of the disease. These plantings consisted of the cultivars Earliwax, Slimgreen, and Tendercrop. Planting dates ranged from 3 June to 20 July. Plantings on different dates in the same field or in nearby fields permitted observations on disease development in plants at various stages of growth under more or less similar environmental conditions. Plantings at scattered locations and in different years provided for observations on disease development under diverse environmental conditions.

White mold became epidemic 10 to 14 days after full bloom in each of the plantings, irrespective of planting date, cultivar, and environmental conditions. In general, outbreaks that occurred after mid-August were more destructive than those that occurred earlier.

Data on temperature and relative humidity were recorded from 15 June to 15 September during three successive seasons in bean plantings made in mid-June in the same field. These data were obtained by means of a recording hygrothermograph set 3 inches above the soil level in a vented weather shelter placed between bean rows.

White mold was first observed about 12 days after full bloom each year, even though environmental conditions before and after the blossom period differed each season. Meteorological data revealed no pattern of environmental conditions that predisposed bean plants to white mold. The microenvironment between bean rows after the plants had made sufficient growth to form a dense leaf canopy was less subject to wide variations in temperature and humidity than was the gross environment. The relative humidity in the plant environment reached 100% for at least several hours each night, even during periods of drought.

The influence of rains during each of three successive seasons was also observed in the same field. Rains did not alter the pattern of first occurrence of white mold after full bloom, but did appear to influence the incidence of the disease.

In 1966 and 1967, July rainfall totaled 3.6 inches (1 inch = 2.54 cm) each year and August rainfall totaled 1.7 and 5.4 inches, respectively. Despite these wide differences in August precipitation, about 90% of the plants were infected each year by the end of August. In 1968, July rainfall totaled 1.8 inches and August

rainfall, 3.9 inches. Under these conditions, only 53% of the plants were infected by late August. Although no meteorological data were taken in 1969, rainfall was deficient in July and white mold incidence in August was low. These observations suggest that July precipitation is of importance in development of primary inoculum.

Primary causal agent.—The soil surface between and within bean rows in fields with recent histories of white mold was examined at about 10-day intervals during the growing season for apothecia of *S. sclerotiorum*. These observations were made during three seasons in eight bean plantings.

No apothecia were found. At one location, 12 apothecia were found in three separate clusters in sod at the edge of a field. Seven of the eight fields examined suffered severe outbreaks of white mold during the course of these observations, indicating the presence of an abundance of inoculum even though no apothecia were found.

Attempts were made in one field to trap ascospores of *S. sclerotiorum*. Open petri dishes containing potato-dextrose agar (PDA) were maintained overnight in an inverted position 8 to 16 cm above the soil level between bean rows. The dishes were collected in the morning, covered, and held in cold storage until examined for spores. These dishes were then transferred to an incubator maintained at 22 C and examined over a period of 12 days for characteristic mycelial growth and sclerotia of the white mold fungus.

No spores of the white mold fungus were observed. However, growth of *S. sclerotiorum* frequently developed from minute particles of organic matter adhering to the surface of the medium. These observations suggested that mycelium in airborne particles of dried infected tissues and in particles of soil organic matter may serve as primary causal agents.

To determine if current season sclerotia served as inoculum sources during the same season, sclerotia collected on 15 August from infected bean pods in an earlier planting were buried to a depth of 1.5 cm in the soil between bean plants in another field where plants were approaching the blossom stage of growth. None of these sclerotia germinated or underwent any other noticeable change in 30 days. Plants in the vicinity of the buried sclerotia remained healthy.

Similar sclerotia were tested for germination in the laboratory. Untreated sclerotia and sclerotia that had been soaked for 5 min in a one-to-nine dilution of Clorox (5.25% sodium hypochlorite) were placed on moistened sand in covered petri dishes. Six plantings of three sclerotia/dish were maintained at 10, 16, and 22 C in unlighted chambers, and observed at various intervals for germination. When sclerotia germinated, they were transferred to the surface of moistened sand in petri dishes in a lighted chamber maintained at 22 C.

Sclerotia that were not surface-sterilized failed to germinate at any temperature in 60 days, at which time the tests were discontinued. Most of these sclerotia were covered by mycelium of various fungi. Surface-sterilized sclerotia remained free from surface fungal growth. Surface-sterilized sclerotia held at 10 and 22 C failed to germinate in 60 days. However, most of the surface-sterilized sclerotia held at 16 C germinated in 21 to 28 days.

Only stipes developed from sclerotia that germinated in darkness. Apothecial development was evident at the tips of these stipes 5 days after germinated sclerotia were transferred from dark to lighted chambers at 22 C. Ascospores were detected 8 days after the first evidence of apothecial development.

Because of the time and specific temperature requirements for their germination, it is unlikely that current season sclerotia serve as important sources of inoculum under New York conditions during the same season they are produced.

Role of bean blossoms.—The role of bean blossoms in the epidemiology of white mold was observed in replicated interplantings of Tendercrop bean made on 18 June, 3 July, 19 July, and 1 August in a field with a recent history of the disease. Seedlings and plants in each planting were observed for symptoms of white mold at 3- to 4-day intervals, beginning at emergence and continuing until pods were mature. Incipient infections were examined to identify the primary causal agent and to obtain information on the relationship of plant development to the first appearance of the disease. Secondary infections were also examined to obtain information on the spread and progress of the disease.

White mold was first observed 8 to 14 days after full bloom in each planting. The date of each planting with its corresponding dates of full bloom and first appearance of white mold were as follows: 18 June-2 August and 14 August; 3 July-14 August and 22 August; 19 July-26 August and 5 September; and 1 August-12 September and 26 September, respectively.

Bean seedlings in later plantings remained free from white mold even though exposed to the same inoculum potential and environmental conditions during the period in which plants of earlier plantings at a postbloom stage of development in adjacent rows became infected. The occurrence of the disease only after full bloom indicated that the blossoms were involved in the epidemiology of white mold.

The first symptoms consisted of brown, water-soaked spots in the axillary tissues of the main stem and lower branches of plants at a postbloom stage of development. Incipient infections were caused by invasion of plant tissues by mycelium of the causal fungus that emerged from cast blossoms lodged in the branch axils. The infection usually progressed slowly in the main stem, but advanced rapidly into the branches. Leaves of infected branches turned yellow, wilted, and were eventually shed, leaving dry, light-brown spurs of naked branches and petioles. Frequently, sclerotia and small white tufts of mycelium were scattered along the length of the dead branches.

Secondary infections were characterized by abundant mycelial growth from infected tissues, rapid disintegration of infected tissues, and rapid spread of the disease. Spread occurred by in situ contact of healthy with infected tissues, and by distribution of disintegrating tissues by gravity, wind, water, and by other

agents capable of transportation of infected plant parts. Stems, branches, leaves, and pods appeared to be equally susceptible. In a number of commercial bean plantings, spread of the disease was so rapid that the crop was rendered unmarketable about 7 days after first appearance of the disease.

The localization of initial infections in axillary tissues at the site of lodgment of cast blossoms indicated that these blossoms were essential intermediaries between the primary causal agent and the infection of the plants. The primary causal agent was unable to invade growing bean tissues, but readily invaded blossoms after they became senescent or died. The mycelium of the causal fungus established in cast blossoms, however, was capable of inciting infections in healthy growing tissues of bean plants. Senescent and dead blossoms invaded by the fungus thus play a key role in the development of epidemics of white mold.

Blossoms at different stages of maturation collected about 5 days after full bloom in four successive plantings of Tendercrop bean in a field with a history of white mold were tested for the presence of the white mold fungus. The blossoms were placed on moistened filter paper in petri dishes, four blossoms/dish and six dishes/blossom category from each planting. The dishes were covered, then placed in an incubator maintained at 22 C. Seven days later, the number of blossoms from which mycelium of the white mold fungus emerged was determined. The results of tests on blossoms from the different plantings were combined.

The blossom categories tested and the average incidence of blossoms in each category that produced growth of the fungus were as follows: freshly opened blossoms, 0%; uncast mature to senescent blossoms, 8%; cast blossoms lodged on lower branches, 58%; and cast blossoms on the soil surface, 87%; respectively.

These results confirmed previous observations that the primary causal agent was unable to invade healthy growing bean tissues, but readily invaded senescent and dead blossoms. The causal agent was not identified, but the presence of the fungus in cast blossoms lodged on bean foliage offered presumptive evidence that the infectious unit was airborne. Many cast blossoms on the soil surface were invaded by mycelium that appeared to have emerged from the soil.

Control.—Foliage sprays with Benlate 50W (benomyl) [methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate], Botran 75W (2,6-dichloro-4-nitroaniline), and Thiabendazole (TBZ 60%) [2-(4-thiazolyl) benzimidazole] were evaluated for bean white mold control in laboratory and field tests. The objectives were to determine the stage of plant development when fungicide sprays would provide maximum protection of blossoms from invasion by *S. sclerotiorum,* and to determine if control of blossom invasion would provide the basis for the development of an effective and economically feasible fungicide control program.

Laboratory tests.—Gallatin 50 snapbeans were planted in 8-inch pots on successive dates. Each planting was thinned to four uniform plants/pot 5 days after seedling emergence. When plants in the first planting attained full bloom, groups of four pots/treatment from each planting were sprayed for 2 min at 10 psi with Benlate 50W at 2.4 g/liter, Botran 75W at 3.6 g/liter, and TBZ (60%) at 3.6 g/liter. Plant development in the various plantings at time of spray application ranged from full bloom in the first plantings to seedlings with only primary leaves in the last planting.

Blossoms harvested at full bloom from each planting were used in evaluating the treatments for control. The interval from spray application to blossom harvest from the different plantings ranged from 0 to 23 days. The harvested blossoms were air-dried, then stored in paper boxes. Thirty to 60 days after blossom harvest from the last planting, four dried blossoms were placed on moistened filter paper in each of six petri dishes/treatment in each of two tests. One drop of diluted homogenized 10-day PDA cultures of *S. sclerotiorum* was placed on each blossom. The dishes were covered, then maintained in an incubator at 22 C. Seven days later, the number of blossoms from each treatment that supported growth of the fungus was determined.

Benlate, Botran, and TBZ effectively protected blossoms harvested from plants that were sprayed at full bloom (Table 1). These blossoms were directly exposed to fungicide sprays. In tests on blossoms that were harvested 3 days after the plants were sprayed, Benlate provided complete protection, whereas Botran and TBZ gave only fair control. Benlate still gave effective control in tests on blossoms collected 5 days after the plants were sprayed, while Botran and TBZ were not effective. These plants were at a green-bud stage of growth when sprays were applied. Benlate failed to provide effective protection to blossoms collected 10 and more days after the plants were sprayed.

In another test, Gallatin 50 beans planted on the same date were sprayed at full bloom with Benlate 50W, Botran 75W, and TBZ (60%) as previously described. Freshly opened blossoms were harvested 1 hr

TABLE 1. Control of white mold (*Sclerotinia sclerotiorum*) in blossoms harvested from successive greenhouse bean plantings sprayed with Benlate, Botran, and TBZ

Days from spray[a] to full bloom	Incidence of white mold[b]			
	Benlate[c]	Botran[d]	TBZ[e]	Unsprayed
	%			
0	8	8	12	100
3	0	48	33	100
5	6	92	75	100
10	38	100	100	100
13	87	100	100	100
23	100	100	100	100

[a] Gallatin 50 snapbeans planted in greenhouse on 1, 5, 13, 16, 19, and 24 February. All plantings sprayed with fungicides on 13 March when plants in first planting attained full bloom.

[b] Percentages based on no. blossoms that supported growth of *S. sclerotiorum* in 48 blossoms/treatment.

[c] Benlate 50W (benomyl).

[d] Botran 75W (2,6-dichloro-4-nitroaniline).

[e] TBZ (60%) (Thiabendazole) [2-(4-thiazolyl) benzimidazole].

after the sprays were applied, and then other crops of freshly opened blossoms were harvested from the same plants 3, 6, and 9 days later. The harvested blossoms were dried, stored, then inoculated as in the previous test.

Benlate, Botran, and TBZ effectively protected blossoms harvested from plants that were sprayed at full bloom (Table 2). These blossoms were directly exposed to fungicide sprays. Benlate still provided effective protection to blossoms harvested 9 days after the plants were sprayed. Botran and TBZ failed to protect blossoms that opened 3 and more days after the plants were sprayed.

These tests indicated that control provided by Botran and TBZ was principally of surface-protective nature; the fungicide deposited on the blossom surface prevented invasion of the blossom by the pathogen. Benlate provided control not only by surface protective activity but also by systemic activity of the fungicide absorbed by the plant and translocated into developing buds and blossoms. To obtain maximum protection of bean blossoms, the plants should be sprayed with Benlate about 3 days before full bloom.

Field tests.—Foliage sprays with Benlate 50W, Botran 75, and TBZ (60%) were evaluated for control of white mold in test plots located in commercial snapbean plantings in central and western New York. Planting dates ranged from 3 June to 20 July. Bean cultivars consisted of Earliwax in early plantings and Tendercrop and Slimgreen in main and late season plantings. All fields had recent histories of white mold.

Test plots were sprayed with water suspensions of the fungicides by means of a portable sprayer that delivered 60 gal/acre (561 liters/hectare) at 40 psi. Compressed CO_2 was used as a propellant. Nozzle placement on the spray boom provided for uniform fungicide spray coverage. Timing of spray applications was centered about the date of full bloom. Prebloom sprays were applied 3 to 7 days before full bloom; full bloom sprays, when the first flush of blossoms opened; and postbloom sprays, about 7 days after full bloom. Sprays were also applied at various combinations of these arbitrary dates.

Test plots were scored for white mold incidence 2 to 3 weeks after full bloom depending upon disease development. Randomly collected samples of 200 plants/treatment were pulled and examined for white mold at each scoring. Data were taken on incidence of infected pods in similar samples in some tests.

Fungicide evaluations.—Test plots located in a portion of a commercial planting of Earliwax bean made on 12 June were sprayed with Benlate 50W at 0.75 and 1.5 lb. active ingredient/acre (1 lb./acre = 1.12 kg/hectare), Botran 75W at 1.0 and 2.0 lb. active/acre and Thiabendazole (TBZ 60%) at 0.5, 1.0, and 1.5 lb. active/acre. Some plots were sprayed only at full bloom (28 July); some plots only after full bloom (7 August); and other plots, at each date.

No white mold was observed during the period of spray applications, but was generally present in untreated portions of the field 12 days after full bloom. The commercial planting in which the spray plots were located was harvested 5 days before the scheduled harvest date in order to avoid the risk of total loss of the crop.

In plots sprayed at full bloom and again 7 days later, Benlate at each concentration provided outstanding control (Table 3). Botran and TBZ provided moderate control at the 2.0-lb. rate, and only fair control at the lower dosages.

In plots sprayed only at full bloom, Benlate at each concentration provided effective control, whereas Botran and TBZ at each concentration gave fair to poor control. In plots sprayed only after full bloom, none of the fungicides gave effective control. However, the

TABLE 2. Control of white mold (*Sclerotinia sclerotiorum*) in blossoms harvested on successive dates from plants sprayed with Benlate, Botran, and TBZ at full bloom in the greenhouse

Days from spray[a] to blossom harvest	Incidence of white mold[b]			
	Benlate[c]	Botran[d]	TBZ[e]	Unsprayed
	%			
0	8	8	4	100
3	0	83	63	100
6	4	100	100	100
9	25	100	100	100

[a] Gallatin 50 snapbean plants sprayed with fungicides at full bloom and blossoms harvested 1 hr later; freshly opened blossoms then harvested at 3-day intervals from same plants.

[b] Percentages based on no. of blossoms that supported growth of *S. sclerotiorum* in 48 blossoms/treatment.

[c] Benlate 50W (benomyl).

[d] Botran 75W (2,6-dichloro-4-nitroaniline).

[e] TBZ (60%) (Thiabendazole) [2-(4-thiazolyl) benzimidazole].

TABLE 3. Control of white mold (*Sclerotinia sclerotiorum*) in field plantings of beans sprayed with Benlate, Botran, and TBZ at full bloom and 7 days after full bloom

Fungicides	Lb./acre (active)	White mold incidence at following spray schedules:[a]		
		Full-bloom + postbloom	Full bloom only	Postbloom only
			%[b]	
Benlate 50W[c]	1.50	0 a	2 a	48 a
	0.75	5 a	9 b	63 b
Botran 75[d]	2.00	37 b	60 cd	87 cd
	1.00	77 d	81 e	95 d
TBZ (60%)[e]	2.00	44 b	51 c	63 b
	1.00	53 c	69 d	80 c
	0.50	79 d	80 e	95 d
Unsprayed		90 e	91 f	95 d

[a] Earliwax beans planted on 12 June. Full bloom spray applied 28 July; postbloom spray, 3 August. Spray plots scored for white mold incidence on 15 August.

[b] Percentages followed by the same letter within columns do not differ significantly at the 5% level of confidence.

[c] Benlate 50W (benomyl).

[d] Botran 75W (2,6-dichloro-4-nitroaniline).

[e] TBZ (60%) (Thiabendazole) [2-(4-thiazolyl) benzimidazole].

incidence of infected plants in plots sprayed with Benlate at the 1.5 lb. rate was less than that in plots sprayed with Botran and TBZ at a rate of 2.0 lb./acre.

In one test, in addition to effective control of white mold, one spray of Benlate applied 3 days before full bloom provided effective control of gray mold, a pod rot caused by *Botrytis cinerea*. In plots sprayed with Benlate at 1-lb., Botran at 3-lb., and TBZ at 3-lb. active ingredient/acre, the incidence of pods with gray mold in a 200-pod sample was 3%, 15%, and 11%, respectively. In unsprayed plots, 20% of the pods were infected.

Timing of Benlate sprays.—Benlate 50W sprays at a concentration of 1.0-lb. active ingredient/acre were evaluated for control of white mold in seven tests conducted during 3 seasons. Sprays were applied 3 to 7 days before full bloom (prebloom), at full bloom, and about 7 days after full bloom (postbloom). Sprays were also applied at various combinations of these arbitrary dates. The results of these tests were combined.

Prebloom spray applications provided more effective control than did sprays applied at full bloom (Table 4). Postbloom spray applications were generally ineffective. A spray program consisting of a prebloom spray followed by a full bloom spray provided outstanding control in all tests. A prebloom spray plus a postbloom spray was no more effective than the prebloom spray alone. Also, a full bloom spray alone was as effective as the combination of a full bloom plus a postbloom spray.

These results indicated that foliage sprays with Benlate provided maximum control when applied during the period in which buds and blossoms were at an active stage of development. Benlate sprays applied after full bloom when blossoms were mature and senescent did not give effective control.

DISCUSSION.—Ascospores are considered to be the primary causal agents of bean white mold. Wind-borne ascospores could account for the sudden and widespread occurrence of the disease observed in commercial bean plantings. However, our failure to trap ascospores and to find apothecia in bean fields being destroyed by white mold suggested that other forms of primary causal agents than ascospores may be involved in the initiation of the disease. Our studies indicated that air-borne particles of organic matter carrying mycelium of the causal fungus could serve as primary infectious units, but their importance was not determined. Perennial mycelium and mycelium from sclerotia in the soil have been reported to incite bean white mold (2, 10, 18). In these studies, invasion of cast blossoms lodged on bean plants by mycelium extending from the soil to the bean foliage was not observed. The distribution of cast blossoms invaded by the causal fungus from the soil surface to aerial parts of the bean plants may also be involved in the initiation of plant infections. However, field observations indicated that the distribution of such blossoms was doubtful because they were held more or less in place on the soil surface by mycelial strands, and because air movement under the foliage canopy generally was not sufficient to transport blossoms from one location to another. Of the various possibilities, ascospores appear to be the most logical primary infectious agents because of their abundance and ease of dissemination. Although direct penetration of healthy, growing bean tissues by ascospores has been observed (4, 7, 10, 14), other research workers have reported that ascospores generally are unable to invade healthy growing bean tissues, and that most plant infections take place through the intermediary of dead blossoms (3, 10, 12, 17). These studies indicated that senescent and dead blossoms served as essential intermediaries between the primary causal agents and plant infections in the development of bean white mold. The interposition of dead blossoms and other flower parts between ascospores of *S. sclerotiorum* and plant infections has also been reported essential for the development of sclerotiniose diseases of crops other than beans (6, 8, 9, 15, 16).

Laboratory and field tests indicated that the outstanding control of white mold provided by Benlate was due to its absorption by bean foliage and its translocation into developing buds and blossoms. This absorption and translocation appeared to be a dynamic process requiring energy from actively growing tissues. Benlate, translocated into developing buds and blossoms, retained its effectiveness after the blossoms became senescent and died. This stability of Benlate in senescent and dead blossoms is of key importance in the control of bean white mold because such blossoms are the essential intermediaries between the primary causal agents and the initiation of plant infections.

The unique coincidence of the systemic activity of Benlate and its stability in senescent and dead bean blossoms with the essential role of such blossoms in the epidemiology of white mold provides the basis for the development of an effective and an economically feasible method of control. One spray of Benlate at a rate of 1.0-lb. active/acre, applied about 3 days before full bloom, will provide acceptable control at the least expense. A prebloom plus a full bloom spray at 1.0-lb. active/acre will provide maximum control. Sprays applied only after bloom will not provide effec-

TABLE 4. White mold (*Sclerotinia sclerotiorum*) control with Benlate sprays applied to bean plants at different stages of blossom development in field plantings

Blossom development at time of spray applications[a]	Incidence of white mold[b] %
Prebloom only	9 b
Full bloom only	18 c
Postbloom only	49 d
Prebloom + full bloom	2 a
Prebloom + postbloom	11 b
Full bloom + postbloom	16 bc
Unsprayed	79 e

[a] Benlate 50W (benomyl) [methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate] sprays at the rate of 1.0 lb. active ingredient/acre. Prebloom sprays applied 3 to 7 days before full bloom; full bloom sprays applied when first flush of blossoms opened; postbloom sprays applied about 7 days after full bloom.

[b] Average incidences of white mold in seven field tests. Percentages followed by the same letter do not differ significantly at the 5% level of confidence.

tive control because Benlate is more effective as a systemic than as a surface protectant.

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